TEXT-BOOK

OF

OPHTHALMOLOGY

BY

HOFRAT ERNST FUCHS

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AUTHORIZED TRANSLATION FROM THE TWELFTH GERMAN EDITION; COMPLETELY REVISED AND RESET, WITH NUMEROUS ADDITIONS SPECIALLY SUPPLIED BY THE AUTHOR AND OTHERWISE MUCH ENLARGED

BY

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WITH FOUR HUNDRED AND SIXTY-TWO ILLUSTRATIONS

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PREFACE TO THE FIFTH EDITION

In preparing the present edition, which is in several senses a new work, the translator has been placed in a position of peculiar responsibility and difficulty. No new German edition has been issued and none is at present contemplated. When, however, Hofrat Fuchs learned that another American edition was projected, he not only gave his permission for the insertion of such additions as in the translator's judgment might seem desirable, but also with characteristic kindness himself supplied notes of many additions and changes. These, therefore, as well as the many alterations made by the translator, are not contained in any German edition.

The translator with some diffidence has made a number of radical alterations in the arrangement of the text, which, he believes, will make the book more serviceable as a work of reference. The many pages of remarks, in fine print, which were massed as an appendix at the end of chapters or major divisions, and whose considerable value and interest were somewhat obscured by this arrangement, he has split into shorter sections, each placed in direct juxtaposition to the portion of the text with which it was related. Some of the more important items in the fine print he has transferred bodily to the text. He has also transferred to Part V, on operations, the descriptions of various operative methods previously scattered through other portions of the book.

These changes had the approval of the distinguished author. With regard to other changes, however, owing to the difficulties in correspondence entailed by the war, consultation with him was impossible, and these changes the translator has made quite on his own responsibility. In the chapter on motility he has entirely rewritten the section on the diagnosis of ocular paralyses, and in the articles on heterophoria, squint, and nystagmus has consolidated with the original text the matter which in the former edition he had himself added as an appendix. While, as always, the fact that matter is thus added or radically altered is indicated by the use of brackets and the initial D, so that there should be no misapprehension as to the authorship, the translator wishes in justice to the original author to make clear the fact that the latter is not responsible for these changes nor for any errors that may unwittingly have been made.

The progress of ophthalmology has necessitated numerous and important changes in all parts of the book. Probably of most importance to the American reader are the additions in the chapters on glaucoma, diseases of the retina, and disturbances of motility, and in the sections on refraction, accommodation, and operations. The latter section has not only been rearranged, but considerably added to. Among the many additions

scattered through all parts of the book, may be mentioned the remarks on tuberculin and vaccine therapy, the visual field and color testing, the mapping of scotomata and the blind spot, squirrel plague and eel's blood conjunctivitis, Samoan conjunctivitis, peculiarities of conjunctivitis in the Near East, extragenital gonococcus infection, inclusion blennorrhœa, the etiology of trachoma, blastomycetic dermatitis, superficial linear keratitis, sclerosis of the cornea, the etiology of iritis, sclerosis of the chorioid, suppurative chorioiditis, Elliot's summary of glaucoma theories, retinitis stellata, retinitis exudativa, and angiomatosis retinæ, the different forms of retinal degeneration, the varieties of accommodative troubles other than paralysis, and the newer operations.

The translator trusts that these changes, so largely made on his own responsibility, will not in any way serve to detract from the many excellencies of a book which, because of its author's unrivalled experience, knowledge, and judgment, has for twenty-seven years remained a model of its kind.

ALEXANDER DUANE.

139 EAST THIRTY-SEVENTH STREET, NEW YORK,
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PREFACE TO THE FOURTH EDITION

The translation now presented to the reader has been made from the twelfth edition of Professor Fuchs's celebrated book. Besides the numerous additions and corrections which Dr. Fuchs has inserted in all parts of the book and a number of new illustrations, there has been added a whole new part constituting a general introduction to the work. This valuable addition, comprising some sixty pages of matter, which is nearly all entirely new, considers in an eminently lucid and conservative way the general physiology of the eye, and the pathology, etiology, symptomatology, and treatment of eye diseases as a whole. The helpfulness of this feature of the book in introducing and elucidating the portions which follow and which relate to the diagnosis, pathology, and treatment of the special conditions in turn, will, we believe, be much appreciated by both the student of ophthalmology and the general practitioner.

The present edition, like the previous ones, contains both in text and illustrations numerous additions by the translator. These will be found mainly in the sections on functional examination, motor anomalies, refraction, and operations. For these insertions (distinguished by being inclosed in brackets and also, when of any length, by being signed with the initial D), the translator is wholly responsible.

In view of the favorable reception accorded to the previous American additions of Professor Fuchs's work in this country, the translator has little hesitation in offering still another, particularly as the German edition upon which it is based represents an essentially improved form of a work which has already proved to be of value to so many—and of a work, moreover, which in matter, scope, and treatment contains so much that appeals both to the professed ophthalmologist and to the general practitioner.

Alexander Duane.

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June 1, 1911.

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PART 1

INTRODUCTION

TEXT-BOOK

OF

OPHTHALMOLOGY

INTRODUCTION

GENERAL PHYSIOLOGY OF THE EYE

1. In many unicellular organisms—animals and plants, bacteria and protozoa—sensitiveness to light is a property of the body as a whole. In the more highly organized, multicellular animals, sensitiveness to light is no longer a function of the whole body, but is taken over by certain cells, which, in contradistinction to the rest, have retained the property of reacting to light. In its simplest form a visual organ of this sort consists of an epithelial cell connected with a nerve fiber. The epithelial cell has the power of transforming light into another form of energy of such a character that it can be conducted along the nerve fiber to a central nerve organ. Even the eye of man is essentially reducible to the same type of apparatus, which, however, here is developed to the highest perfection. For here, instead of a single sensory epithelial cell, there are an enormous number of such cells, which are united to form the retina and optic nerve. This is the essential part of the eye, and with its formation, too, the development of the eye begins (outgrowth from the rudimentary brain of the primary optic vesicle, which gives rise to the retina). All the other parts of the eye develop later and are designed for protection, for nutrition, or for optical purposes. The exterior fibrous envelope of the eyeball—cornea and sclera—serves to protect the delicate retina; the uvea subserves nutrition; and all of these, together with the lens, help to make a better optical apparatus—the cornea and the lens by the way in which they refract light, and the uvea because of its light-regulating diaphragm, the iris, and because of its dark pigment.

I. Exterior or Fibrous Coat (Corneo-Sclera)

2. The chief function of the corneo-sclera is to protect the inner coats of the eye, and its most important property, therefore, is its *rigidity*.

This is produced in the following way: the cornea and sclera are composed of densely laminated and interlaced, inflexible connective-tissue fibers, which are traversed by a moderate number of elastic fibers, so that with great rigidity there is afforded a slight, but only a slight, degree of elasticity.

The rigidity of the corneo-sclera is lessened at two points—first and most at the spot where the optic nerve enters the eye and where the sclera is reduced to a set of delicate fibrous bands called the lamina cribrosa; next at the corneo-scleral margin where the scleral gutter is tunneled into to form Schlemm's canal. Hence the spot where the optic nerve enters the eye is the first place to yield to intra-ocular pressure (pressure-excavation); and the corneo-scleral margin is the typical location for a rupture of the fibrous coats of the eye due to external violence. The slight degree of elasticity of the corneo-sclera is the reason why a uniform expansion of the latter does not take place when the intra-ocular pressure is increased, except in youth, when the elasticity is still fairly great (hydrophthalmus). As age increases, the corneo-sclera becomes less and less elastic, and admits of only partial protrusions, and in these, moreover, it is no longer a question of simple stretching but of rupture also (in the cornea, ruptures of Descemet's membrane in the case of hydrophthalmus and keratoconus; in the sclera, ruptures of its innermost lamellæ in the case of scleral staphylomata). The slightness of the elasticity makes itself apparent also when there is reduction of the intra-ocular tension. In a more elastic eyeball such reduction would lead to a uniform contraction of the organ, whilst in the human eye in this case folds are formed both in the cornea and sclera (e.g., in atrophic and phthisical eyes).

Rigidity alone would not afford the eye sufficient protection. "Pain is man's guardian." The sclera, which in large part lies imbedded in the orbital tissues, does not need to have any great sensitiveness, and hence has but few nerves; but the cornea, which is directed toward the outer world, has the greatest sensitiveness of any part of the surface of our body and has consequently the greatest profusion of nerves. A dense plexus of nerve fibers, in fact, lies in the epithelium itself, a thing which is not the case elsewhere on the surface of the body. Hence it is that in most men even the slightest contact of anything with the cornea starts up a sensation not of pressure but of actual pain; and thus it is guaranteed that the most inconsiderable lesions, the smallest foreign bodies, excite our attention and are not neglected.

3. Besides its function as a protective organ, the cornea has an optical function to fulfil. It must be transparent in order to admit the light rays, and it takes part in their refraction.

The *transparency* is dependent on two factors. The positive factor is the composition of the cornea—the fact that it is built up of lamellæ which are very homogeneous, refract the light equally, and have comparatively few cells between them. The negative factor is the absence of opaque tissue, above all, the absence of blood-vessels.

That, in order to secure complete transparency, it is necessary for all parts of a tissue to have like refractivity is proved by the following experiment: If we grasp an eye—for instance, a pig's eye which is being used for practising operations on—between our fingers and squeeze it hard, the cornea at once becomes cloudy, and when the pressure is released becomes clear again just as quickly. The original assumption that here we are dealing with the same sort of thing as occurs in the pressure opacity in cases of glaucoma must be discarded, for in this experiment when the pressure of the fingers is relaxed the transparency returns at once, while in glaucoma after the increase in tension disappears it still takes some time for the cloudiness to pass away. The explanation of the phenomenon is, rather, as follows: The cornea consists of fibers which run in different directions in the different layers. When we press the eyeball between the fingers, the fibers undergo a stress which varies in intensity according to the direction in which the fibers run. One-sided traction or pressure makes a single-refracting substance double-refracting, and this holds good for the fibers of the cornea, as we can convince

ourselves by examination with the polarization microscope. Owing now to the fact that the fibers of the cornea have become unequally double-refracting, a ray of light passing through the layers of the cornea is diminished in brightness just as in passing through a crossed Nicol prism (Fleischl). Such unequal refractivity of the separate layers of the cornea is the cause of many opacities which we see clinically. We sometimes observe in the cornea striæ or reticulate opacities which are called keratitis striata, but erroneously, since inflammatory changes are absent. What we find is a wrinkling of the corneal lamellæ, and, as a result of this, opaque spots develop, although the individual lamellæ are perfectly transparent.

Usually, however, *opaqueness* of the cornea is due to tissue changes. Even mere soaking of the corneal lamellæ with liquid makes them swollen and cloudy. On this fact depends the cloudiness of the cornea occurring with lesions of the endothelium, in consequence of which the aqueous enters the cornea from behind. So, too, soaking with lachrymal fluid from in front makes the borders of corneal ulcers, and particularly the borders of flap-edged wounds of the cornea, cloudy and swollen. In glaucoma the cloudiness of the cornea is caused by fluid, which is thrown out in minute droplets, particularly between the epithelial cells. The marked cloudiness present in recent inflammation of the cornea is caused by the deposition of cells and of unorganized exudate between the corneal lamellæ. After the subsidence of the inflammation new-formed connective tissue and vessels may remain between the corneal lamellæ, and thus produce a permanent opacity, while in true scars of the cornea the corneal tissue is actually replaced by connective tissue.

4. *Non-vascularity* is necessary if the demands of perfect transparency are to be fulfilled, yet there is no doubt that under certain circumstances this lack of vessels is disastrous to the cornea.

Under normal conditions the nutrition of the cornea is effected satisfactorily even without vessels, but under pathological conditions greater demands are made upon tissue metamorphosis. When, after a lesion of the surface epithelium bacteria invade the tissues of our body, the latter defends itself against the invaders by means of bactericidal and antitoxic substances, which to a certain extent are already present in the blood, and by means of leucocytes which, under the guise of phagocytes, take up the bacteria and digest them. When the invasion of bacteria occurs in vascularized tissue (e.g., the cutis), capillary loops lie everywhere very close at hand, from which the blood can act upon the germs at once in the manner above described. Certainly it is in this way that the vast majority of infections in our body are nipped in the bud. It is otherwise in the non-vascular cornea. This normally contains no protective substances in its tissue fluid. Invading germs at first multiply unhindered until they have formed so much toxin that the latter gets to the marginal vessels of the cornea and causes their dilatation. As a result of this, the protective substances of the blood and also the leucocytes pass over into the cornea, but even then they have to get back to the place where the germs are accumulated. Hence the distance from the site of the lesion to the corneal margin has to be traversed twice before any onslaught is made upon the bacteria, and in the meantime a considerable multiplication of the latter may have taken place. The distance, of course, is the longest in the case of lesions which occupy the center of the cornea, and this accounts for the fact that the most dangerous lesions—those which lead to ulcus serpens—lie almost always in the center of the cornea. The superficial lesions of the cornea, such as produce an ulcus serpens, certainly affect the marginal portions of the cornea more often than its center, since the area of the former is greater; but in this case the marginal loops of vessels are so near the lesion that the germs entering the latter are speedily rendered harmless.

To the non-vascularity of the cornea is probably also attributable the fact that the latter in spite of its more solid structure is so much more often the site of ulcers than is the conjunctiva [and that wounds of it heal slowly and are readily infected.—D.].

The inflammatory irritation emanating from a diseased spot on the cornea produces first a dilatation of the vessels at the corneo-scleral margin, but if the irritation lasts longer it produces *vascularization* of the cornea, offshoots growing out from the marginal vessels toward the inflamed area. Thus now the blood, which combats the noxious agency, is brought to the battle-ground directly and can therefore develop its action without loss of time. When a corneal ulcer is spreading steadily, and at length we see the new-formed vessels extend at one spot to the border of the ulcer, we know that here no further progress of the ulcer is to be apprehended, the vessels opposing an effective barrier against the disintegration of the corneal tissue. But often the vessels arrive too late to prevent the destruction of the tissue, the loss of substance having occurred before the vessels reached the diseased spot. Then the function of the vessels consists in bringing to its destination the material for repair—i.e., the material for filling in the loss of substance by means of cicatricial tissue. Since it takes a certain time for vessels to develop, then the more slowly the inflammation runs its course, the more likely are we to see the vessels appearing in the progressive stage of the inflammation and limiting the latter. In cases with rapid course the vessels do not make their appearance until the end of the process, and then simply initiate repair. Hence the impelling force which conveys both the out-pouring blood corpuscles and the out-growing blood-vessels to the site of an irritation—a force which has a general applicability for our body—produces in the cornea the characteristic clinical signs of keratitis and at the same time represents a process suitable for combating the things that have come in and caused the mischief.

The non-vascularity of the cornea is in part the cause of its low *temperature*. Owing to the evaporation of the lachrymal fluid on the surface of the cornea, the latter gives off more heat exteriorly than does, for example, the dry skin. To the latter, moreover, more heat is continually supplied by the blood circulating in the capillaries, while for the non-vascular cornea this direct supply of heat is wanting. Hence when the eye is open the temperature of the cornea is scarcely 30° [C.], and because of the thinness of the cornea (less than 1 mm. in the center), the low temperature continues to the deeper parts as well, amounting in the anterior chamber to but 32°, and finally in the vitreous to 36°. Closure of the lids, which stop the evaporation at the surface of the cornea and supply warm blood by means of their vessels, obviously causes the temperature to rise at once, so that when the lids are shut it is between 35° and 36° in the conjunctival sac. The low temperature of the cornea makes it possible for mold fungi to grow in it, while they cannot flourish elsewhere in the living human body because the temperature is too high. There is a mold-fungus keratitis, although, to be sure, it is rare (see § 218).

5. With regard to *nutrition* the fibrous coat of the eye makes few demands. Since its function is merely rigidity—and for the cornea also transparency—no consumption of material worth mentioning takes place in it, and its tissue metamorphosis is, therefore, very slight in amount. The sclera is nourished by the few vessels which it itself possesses. The cornea gets its nutritive material from the vessels of the corneo-scleral margin—i.e., mainly from the pretty dense marginal network of the limbus and from the scanty twigs which the anterior ciliary arteries that pass through the sclera give off deep down. From these two vascular districts superficial and

deep vessels grow out into the cornea when the latter is inflamed. Because the tissue metamorphosis of the cornea is so meagre, the deep vessels, few as they are, suffice to maintain it, for we can remove or cauterize the marginal network very extensively or even all the way round the limbus without the cornea's becoming necrotic.

The nutritive fluid passing from the blood-vessels into the cornea is distributed in the latter by diffusion. The former supposition that the lymph circulated in a system of cavities, the juice-canals of Recklinghausen, is no longer held.

Does the aqueous also take part in the nutrition of the cornea? To answer this question it must first be determined whether the aqueous can enter the cornea at all. With regard to the behavior of a tissue to a fluid that bathes it, two different physical processes are possible, filtration and diosmosis. In filtration the fluid goes through the membrane without change of composition, and only the undissolved solid constituents are kept back. Such a process, for example, takes place when the aqueous passes through the ligamentum pectinatum. In diosmosis (diffusion through a membrane), on the other hand, the fluid as such does not pass through the animal membrane; but between the two fluids which are present on the two sides of the membrane—thus between the aqueous and the lachrymal fluid in the case of the cornea—there takes place an exchange of the dissolved constituents until the osmotic tension on the two sides of the membrane has become the same. If, as the simplest case, we suppose that on either side of the membrane there is the same salt solution though of unlike concentration, salt will pass from the side of greater concentration to the other side, and from the latter, in turn, will pass in the opposite direction, until the concentration on the two sides is the same.

Which of these two processes, filtration or diosmosis, occurs between aqueous and cornea? It was formerly assumed that there was *filtration* of the aqueous forward through the cornea, and it was believed that a continuous percolation of aqueous was necessary not only for the nutrition of the cornea but also in order to maintain its transparency. This view is now known to be erroneous, the passage of aqueous into the cornea being prevented by the endothelium of the posterior corneal surface (Leber). If this is experimentally removed in the living eye at any spot, the cornea at the same spot becomes clouded because of the aqueous that gets in there. The same thing occurs under pathological conditions when the endothelium is injured, e.g., by inflammation in the cornea itself or by the deposition of inflammatory products upon the posterior corneal surface.

A similar part is played by the anterior epithelium of the cornea with regard to the lachrymal fluid which bathes the anterior corneal surface. The test with fluorescein proves this. If a one-per-cent aqueous solution of fluorescein is instilled into the conjunctival sac, none of it penetrates the cornea so long as the epithelium of the latter is intact. But as soon as a defect in the epithelium occurs at any spot, the cornea at that point is stained an intense green by the fluorescein that enters it.[1] In a way not as yet explained a like greenish coloration of the cornea by the instillation of fluorescein into the conjunctival sac occurs when the endothelium of the cornea is defective, e.g., in keratitis parenchymatosa.

Hence filtration of fluid through the normal cornea does not exist. How is it then with *diosmosis?* A priori, it must be assumed that as it exists in every animal membrane, so also it does in the cornea. In practice use has been made of this fact for a long time, solutions being put into the conjunctival sac when, as is the case more particularly with

[1] The entrance of fluorescein into the cornea, it is true, occurs by a process not of filtration but of diffusion; but even for the latter the epithelium and the endothelium of the cornea act as an obstacle if not as an absolute barrier.

the mydriatics and miotics, it is desired that they shall act upon the iris. If we drop atropine into the conjunctival sac and then, as soon as the pupil has dilated, draw off the aqueous and drop this into another eye, dilatation of the pupil takes place in the latter also. This proves that the aqueous of the first eye contains atropine, and that, therefore, the atropine salt has passed from the lachrymal fluid into the aqueous by means of diosmosis. In the same way, when chemically active foreign bodies or an aggregation of bacteria are present in the anterior layers of the cornea, irritating substances pass from the cornea into the aqueous and act upon the iris, so that first hyperæmia of the latter and afterwards inflammation with exudation are set up. Both, therefore, are constant accompaniments of every violent inflammation of the cornea.

Even under normal conditions salts and, to a slight extent, albuminous substances get by means of diosmosis from the aqueous through the endothelium and Descemet's membrane into the parenchyma of the cornea, which absorbs them; but, owing to the fact that the aqueous is poor in dissolved matter, this process is certainly of very subordinate significance for the nutrition of the cornea.

II. Middle Coat (Uvea)

6. The uvea, formed of the iris, ciliary body, and chorioid, has for its main function the nourishment of the nervous portion of the eye. Hence it lies in direct contact with the latter, between it and the fibrous envelope, and is, therefore, called the middle coat of the eye. It originates together with the fibrous coat from the mesodermal tissue that envelopes the ocular vesicle. This tissue, which was originally homogeneous in structure, little by little becomes differentiated into an outer and inner layer, corresponding to the outer and the middle coats of the eye. Finally an actual spatial separation is effected. In front, a large fissure develops, which becomes the anterior chamber, and, behind, the connection between sclera and chorioid becomes almost completely disunited, so that here is formed the perichorioidal space. Thus the membranes which originally formed one single tissue are finally connected at two places only, i.e., at the spot where the ciliary body is attached to the sclera and at the margin of the optic-nerve entrance.

Quite the reverse are the spatial relations of the middle coat of the eye to the inner coat, namely, the retina and its anterior prolongation. The inner coat of the eye originates from the secondary ocular vesicle. With this the uvea has nothing at all in common as a tissue, but simply lies in contact with the outer layer of the vesicle. From this layer develops later the retinal pigment. This latter simply lies on the inner surface of the chorioid and ciliary body without being connected with them, yet in the living eye it very rarely separates from the chorioid and never from the ciliary body. Still more intimate is the connection of the retinal pigment with the iris, in whose tissue the muscle fibers of the sphincter and dilatator pupillæ, which are derived from the retinal pigment, lie imbedded. That is, the genetically related membranes, the outer and the middle coats of the eye, lose their anatomical connection almost completely, because functionally they have little to do with each other, while, on the other hand, a functional

relationship leads to an intimate connection between the middle and inner coats, which genetically are perfectly distinct.

Besides the large cleavage space between the outer and middle coats of the eye, which subserves the circulation of lymph in the eye, there is a second space in the interior of the inner coat itself. What was originally the actual cavity of the primary optic vesicle is converted by the invagination of the latter, which serves to form the ocular cup, into a virtual cavity only, since the outer and inner layers of the cup are everywhere in contact though never in connection. This remains so through life, and hence arise consequences that are pathologically important. The retina separates from the pigment epithelium even more readily than the chorioid separates from the sclera (as it does, for example, after operation); and the dreaded detachment of the retina is nothing but the re-transformation of the virtual cavity of the ocular vesicle into a real one. Similarly in the iris the posterior epithelial layer (inner stratum of the ocular vesicle) separates readily from the anterior layer (outer stratum); this, for example, taking place in the rupture of posterior synechiæ, when the posterior layer remains on the lens, the anterior on the iris. Indeed, a spontaneous separation of the posterior from the anterior epithelial layer occurs not infrequently as a senile change.

The character of the uvea with regard to *rigidity* varies, the iris being extremely elastic and the ciliary body and chorioid, on the other hand, inelastic. The elasticity of the iris tissue is necessary if the play of the sphincter and the dilatator of the pupil is to take place readily. The elastic character of the iris is shown clinically in the frequent cases of peripheral incarceration of the iris, e.g., after eczematous ulcers, when we often see the iris of the opposite side drawn over to the very margin of the cornea (Fig. 92). If we wish to detach an anterior synechia by introducing a narrow knife into the anterior chamber and trying with this to divide the tag of iris that runs forward to the cornea, the attempt is usually unsuccessful, because the iris is so elastic that it is more readily stretched than cut even by the sharp knife. On account of the great elasticity of the iris, we practically never find transverse rupture of the iris after injuries, but simply separation of it from the ciliary body, or radial fissures, the latter because, owing to the radial course of its blood-vessels, the iris is very readily indeed split in a radial direction.

In distinction from the iris, the ciliary body and chorioid are inelastic. They rupture easily upon contusion of the eyeball, splits in the chorioid being then often observed. (Splits in the ciliary body, although also not rare, cannot be seen clinically.) Stress of a more gradual kind may also lead to rupture of the chorioid. Corresponding to the atrophic spots which occur in very myopic eyes in the region of the macula lutea, we often find ruptures, especially of the lamina vitrea, with gaping of the torn edges, caused by stretching of the chorioid (Salzmann).

7. In correspondence with its function, the uvea is very richly supplied with *vessels*, and is hence also called the tunica vasculosa. The vascular system of the uvea, which is known as the system of ciliary vessels, has but few anastomoses with the conjunctival vessels at the margin of the cornea, and still fewer with the vascular system of the optic nerve and the retina at the border of the foramen scleræ. But within the uvea itself the anastomoses are very abundant. The arteries are connected by means of two arterial arches, one at the root of the iris, the other close to the pupillary margin. The veins in the chorioid are arranged in whorls or vortices, and the veins belonging to any two adjoining whorls are connected by a series of arched anastomoses.

Hence, disturbances of circulation in the chorioid can be compensated for much more readily than in the retina, whose vessels possess no anastomoses connecting them together. The evidences of vascular occlusion—embolism and thrombosis—which in the retina afford such characteristic clinical pictures, in the uvea can at most be demonstrated anatomically, and produce no symptoms that can be clinically recognized. The only unfavorable factor in the circulatory conditions of the uvea is caused by the arrangement of the veins as they leave the eye. The vortex veins have to carry off out of the eye almost all the blood of the uvea. Owing, therefore, to the impossibility of speedy compensation, ligation of these veins leads to serious disturbances of circulation and to increase of tension. The vortex veins are not numerous in comparison with the abundant network of veins in the uvea, and in the region of the equator are unfavorably placed, in that the blood from the posterior half of the uvea must enter them by a retrograde course (i.e., from behind forward). Furthermore, the very oblique course of the vortex veins through the sclera in comparatively narrow and indistensible canals may lead to interference with the discharge of blood from the eye.

In the anterior segment of the eye the richness of the uvea in vessels is of service in the secretion of aqueous, in the posterior segment is of service in the nutrition of the retina and the secretion of the constantly consumed visual substances—of which, to be sure, we know as yet merely the visual purple. Both objects are accomplished by the fact that the chorioid has a special arrangement of vessels. The large vessels, which do not allow nutrient matter to pass from them directly, are placed in the portion that is furthest from the retina, and, on the other hand, all the capillaries are united to form a single layer, which lies as close to the retina as possible.

Like the cornea in the fibrous envelope, the uvea, too, has an *optical function*, apart from the above-mentioned production of visual substances. The muscular apparatus in the iris and the ciliary body help in securing distinctness of the retinal images, and the pigment content of the uvea together with the retinal pigment prevents diffuse light from getting through the iris, sclera, and chorioid and reaching the retina along with the light that passes through the pupil.

III. Inner Coat (Retina)

8. The retina originates from an eversion of the anterior brain vesicle, and is, therefore, to be regarded as a protruded portion of the brain. It is the primordial part of the embryonic eye, about which the two other coats of the eye become differentiated later on. The structure of the retina is calculated to secure the best possible optical function. For this reason the retina must be transparent, since the light-perceiving layer of rods and cones lies on its posterior side. Hence connective tissue is wanting in the retina in every place except the walls of the blood-vessels, and even these are transparent. Moreover, since the blood-column in the vessels is opaque, it is reduced to a minimum. In many vertebrates the retina is very poor in vessels or is absolutely non-vascular, and in man the fovea centralis, at least, which is the spot that is most important for vision, is destitute of vessels.

The optical advantage which arises from this scantiness of vessels entails, it is true, a disadvantage in that the retina, owing to its great consumption of material, does not find sufficient maintenance in its own vessels, but both *for nutrition and for function is also dependent on the vessels of the chorioid.* The vessels of the retina proper

lie in the inner layers, the vessels of the chorioid (chorio-capillaris) adjoin the outer layers of the retina, and consequently the inner layers are nourished by the retinal, the outer by the chorioidal, vessels. Neither one of the two vascular systems suffices by itself to maintain the function of the retina, and the latter, therefore, is dependent on both. Diseases of either one system or the other cause injury to the retina. And there is the additional fact that the relations of the circulation are very unfavorable for the compensation of circulatory disturbances. The retinal vessels have no anastomoses with each other. The retinal arteries are end-arteries, and closure of any of them at once puts the section of retina that is supplied by it out of the circulation. Furthermore, there are no anastomoses worth mentioning between the retinal and ciliary systems of vessels, so that the latter, whose circulatory relations are far more favorable, cannot substitute for the former. These circulatory relations together with the extremely delicate and complicated structure of the retina cause it to be readily diseased in spite of its protected situation.

Diseases of the retina which are set up through the medium of its *own vascular system* occur either as the result of circulatory disturbances (rupture, contraction, or occlusion of the vessels), or because toxic substances are carried to the retina through the blood (nephritic, diabetic, naphthalinic retinitis, etc.), or finally because the bacteria that are in the blood are arrested in the capillaries of the retina (this occurring probably in many forms of retinitis besides true metastatic retinitis in the narrower sense of the word). The influence of circulatory disturbances in the *ciliary system* of vessels upon the retina has been studied experimentally in animals, in whom ligation of the posterior ciliary arteries produces, first, necrosis with cloudiness of the retina, and afterward atrophy and pigmentation. In man the results of such a circulatory disturbance are not precisely known, but it is certain that in places where the chorioid has lost its vessels the retina in its outer layers becomes atrophic, too—this happening, for example, in high myopia, in which, corresponding to the atrophic spots produced in the chorioid by stretching, there are blind spots in the retina. Moreover, when the ciliary vessels bring phlogogenic substances or bacteria into the chorioid and set up inflammation there, the retina, too, must suffer at once on account of the intimate relationship between it and the chorioid. In an anatomical sense there is no unmixed chorioiditis, but simply a retino-chorioiditis, the outer layers of the retina over the chorioiditic spot being implicated as well.

IV. Lens

9. The lens subserves optical purposes exclusively, and in many regards behaves like the cornea. Its optical function is not associated with consumption of matter, and hence nutrient materials are required by the lens only in extremely small quantities in order to keep the capsular epithelium and the lens fibers from dying. This nutrient material the lens receives from the surrounding liquids—the vitreous and mainly the aqueous—by diffusion through the lens capsule. Definite preformed channels for the circulation of liquid within the lens do not exist. That metabolism in the lens goes on with extreme slowness is proved by the fact that pathological processes in the lens (opacities) often remain stationary for an uncommonly long time, or spread but very slowly.

In another regard, too, the lens is like the cornea. The lens fibers, like the fibers of the cornea, have the property of absorbing liquid in considerable amount and consequently of swelling up and becoming opaque. If after opening the capsule we place

a lens in water, or if in the living eye by opening the lens capsule we give the aqueous access to the lens fibers, the lens becomes cloudy and swollen. As in the case of the cornea, we must ask by what means under normal conditions the lens is protected from the entrance into it of the aqueous. Just as in the cornea it is not Descemet's membrane but its endothelium that keeps the aqueous back, so in the case of the lens it is not, or at least only in slight degree, the capsule, but mainly the epithelium of the latter that effects the same object. Hence every lesion of the capsular epithelium leads sooner or later to cloudiness of the lens. Thus are explained not only traumatic cataracts with rupture of the capsule, but also many other cases of cataract in which the epithelium is injured without rupture of the capsule. This behavior on the part of the lens finds practical application in the procedure of making a partially clouded lens completely opaque (artificial ripening of cataract). This procedure consists in the massage of the anterior surface of the lens, so that the capsular epithelium is injured by compression. Another cause of clouding of the lens is afforded if the composition of the aqueous or vitreous is essentially altered, or poisonous substances are present in these liquids, so that now as a result of diosmosis through the intact lens capsule the liquid that permeates the lens undergoes a change of composition. In this way either the capsular epithelium may be injured and thus the lens fibers be rendered cloudy in an indirect way, or the lens fibers may be affected directly. Thus are explained the opacities of the lens dependent on poisoning (e.g., naphthalinic cataract) and also most cases of complicated cataract, in which by disease of the inner coats of the eye an essential change is set up in the aqueous or vitreous. [Cf. also §§ 488, 490.]

V. Circulation of the Lymph

10. With regard to the circulation of lymph in the eye, there must be considered the passage of nutritive material into the eye (secretion) and its discharge from the eye (excretion).

The *secretion* of the fluids of the eye takes place almost exclusively through the uvea. Besides the fluid that permeates the tissues there is found a pretty large accumulation of fluid in the anterior and posterior chambers of the eye, which are hence known as lymph spaces. To call the aqueous lymph, however, is incorrect, inasmuch as the aqueous in distinction from lymph contains only traces of albumin and but few salts, and furthermore does not clot when drawn from the eye by puncture of the cornea. Clotting occurs only under pathological conditions, when the aqueous at once acquires a large content of fibrin and often forms a clot even when still within the living eye.

For the secretion of the *aqueous* the iris and ciliary body are of moment. That the iris alone does not secrete the aqueous is evident, since even in cases of absence of the iris—congenital or traumatic—the anterior chamber has its usual depth; and in such eyes, too, the aqueous when drawn off is rapidly replaced. But it does not follow from this that the iris has nothing at all to do with the secretion of the aqueous; a small quantity of the latter probably is derived from the anterior surface of the iris, even though this cannot be proved with certainty.

[While it is generally agreed that the intra-ocular fluids are exuded from the uvea, opinions differ as to the way in which this takes place, some saying that there is a true

secretion by the ciliary epithelium, others that there is a simple filtration, and others still that a combination of the two processes occurs (Elliot). According to Priestley Smith, it is likely that the aqueous proceeds mainly from the ciliary processes, the vitreous mainly from the orbiculus ciliaris.—D.]

The aqueous secreted by the iris can leave the eye again by way of the ligamentum pectinatum. But the aqueous that is produced by the ciliary body has no direct exit. It must first pass through the pupil in order to get out through the angle of the aqueous chamber. Hence when in seclusion of the pupil the communication between the two chambers is interrupted, the liquid accumulates in the posterior chamber and pushes the iris forward at the expense of the depth of the anterior chamber, whose contents, however, can still flow off so long as the root of the iris is not jammed against the periphery of the cornea so that the ligamentum pectinatum is blocked.

In any event, since more aqueous is secreted by the ciliary processes than by the anterior surface of the iris, a continuous flow of aqueous must take place from the posterior into the anterior chamber (and from here through the ligamentum pectinatum out of the eye). This flow is conceivable as taking place in two ways: either (1) the aqueous, being secreted in a uniform manner by the ciliary processes, flows quite uniformly, too, and in an imperceptible current through the pupil; or (2) the aqueous does not flow forward through the pupil continuously, since the margin of the pupil is held against the lens by the pressure of the liquid in the anterior chamber or perhaps by the tone of the sphincter pupillæ, and is lifted away from the lens only from time to time when the pressure in the posterior chamber has slowly increased until it is somewhat higher than in the anterior chamber, the result being that the aqueous discharges itself at intervals through the pupil into the anterior chamber. In this latter case the pressure in the two chambers would be subject to differences with periodic equalization, although one must conceive of these differences in pressure as being extremely slight. There are one or two observations which argue the possibility of such differences in pressure even when the pupil is normally open.

A continuous flow of liquid in the aqueous chamber results from the fact that the aqueous that is in contact with the vascular iris grows warmer and hence ascends, while that in contact with the posterior surface of the cooler cornea becomes cooler and hence descends.

The poverty of the aqueous in dissolved substances arises from the fact that the blood plasma does not pass as such through the vessel walls, but that in this passage certain substances are kept back, first by the vessel wall itself, and also, so far as the ciliary body is concerned, by the epithelial wall of the latter. It is a matter of importance that, in common with the other substances, the protective bodies which are already naturally present in the body and which are increased in cases of disease, fail to pass from the blood into the aqueous. The want of such substances in the aqueous favors (in the same way as has been shown to hold good for the cornea) the outbreak of bacterial diseases. Thereafter, however, owing to the inflammatory irritation and the dilatation of the vessels that are associated with it, there results the passage not only of an increased amount of albumin and fibrin, but also of the above-mentioned protective bodies, into the aqueous, and thus the inflammation produces a coincident change in the conditions of secretion which is calculated to combat the harmful agencies.

With respect to the *quantity* of aqueous that is secreted in a given time, formerly very exaggerated ideas were entertained. Observers allowed themselves to be led astray by the fact that after the aqueous is drawn off the anterior chamber fills full again in about six minutes. But it has been shown that the newly accumulated aqueous is essentially different from the normal; that, in fact, it is very rich in albumin and fibrin. By the paracentesis the ocular tension is suddenly very much reduced; the blood flows

in greater quantity than usual into the vessels, since they have been relieved of the pressure, and distends them, so that a liquid rich in albumin can pass through their walls. The stormy course of this process is proved by the changes found by Greef in the ciliary body, whose epithelium soon after the paracentesis is in places detached in the form of vesicles by the liquid which flows abundantly from the vessels. After paracentesis, moreover, just as in the case of inflammation, the protective bodies present in the blood pass over into the aqueous. We have, therefore, in the withdrawal of the aqueous a means of transferring these bodies into the aqueous, a fact by which is explained the therapeutic effect of paracentesis in many cases.

The rapid accumulation of aqueous after it has been drawn off is, therefore, favored by the artificial change of the pressure relations in the interior of the eye; but it would be a mistake to draw from this a conclusion as to the physiological secretion of aqueous. This certainly takes place with comparative slowness. According to Leber's observations, it takes three-quarters of an hour or perhaps longer for the aqueous to be completely renewed.

According to others, the aqueous is renewed even more slowly and perhaps, indeed, is scarcely renewed at all (Hamburger).

11. The outflow of liquids from the eye—*excretion*—must, obviously, so far as its quantity is concerned, be precisely equal to the inflow, as long as the intra-ocular pressure remains the same. As regards the path of the outflow, it must be premised that, both in the eyeball itself and in the orbit, lymph vessels are wanting, the conjunctiva of the eyeball alone possessing them. Hence the outflow, so far as it occurs in preformed channels at all, can take place only by means of lymph spaces.

The lymph spaces of the eye are in part recognizable anatomically, when for example they are lined by endothelium, which, however, is not the case for all tissue spaces in which lymph can flow. In that case experiment serves to demonstrate the lymph passages. Either there are brought into the blood channel chemical substances, for instance potassium iodide, which can be readily recognized and which are first poured out into the eye, and then leave it again, in doing which they can be followed on their way by means of the characteristic chemical reaction (Leplat); or colored solutions (carmine, Prussian blue), or fine suspensions (india ink), are injected into the interior of the eye (anterior chamber, vitreous), when they indicate by the coloration the path by which they leave the eye. Thus there has been found by an injection of a solution of Prussian blue into the anterior chamber, that after a short time a blue coloration can be traced through the ligamentum pectinatum into Schlemm's canal, and from there into the ciliary veins that anastomose with it (Schwalbe, Leber).

12. The lymph passages of the eye (Fig. 1) are distinguished into anterior and posterior.

The *anterior lymph passages* carry out the aqueous. This leaves the anterior chamber through the ligamentum pectinatum. The latter by the superposition of numerous fenestrated lamellæ forms a fine filter (Figs. 147 and 148) which does not transmit larger corpuscular elements, such as, for example, the red blood corpuscles, but only liquids and minute particles. According to the prevalent view these latter pass from here into Schlemm's canal, which is a venous circular sinus. Through the latter, therefore, and

the ciliary veins that anastomose with it, by far the greatest part of the lymph leaves the eye.[2]

Since the *ligamentum pectinatum* is the main channel of outflow for the eye fluids, very serious troubles are produced by its occlusion. The fluid is retained in the eye, and increase of tension develops which leads to blindness. Injury to the filter of the ligamentum pectinatum may occur in general through the following different processes: (1)

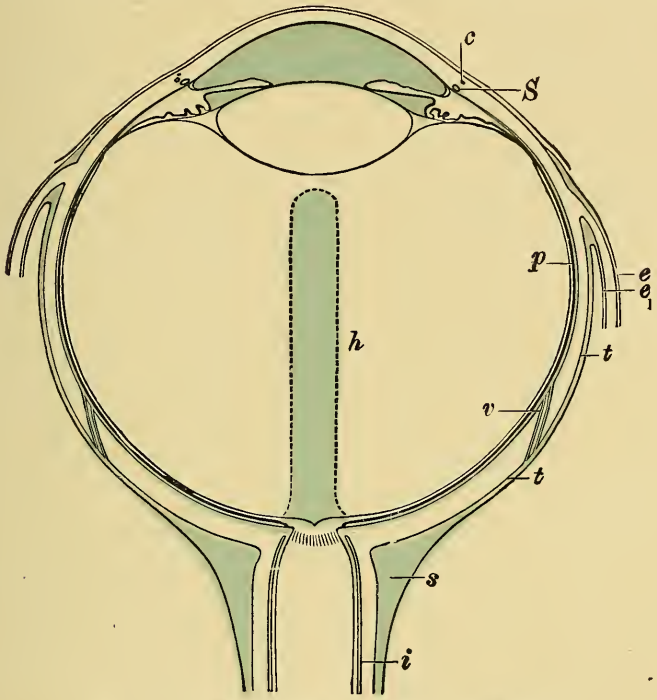

FIG. 1.—LYMPH PASSAGES OF THE EYE. (SCHEMATIC.)

S, Schlemm's canal; *c*, anterior ciliary veins; *h*, hyaloid canal; *p*, perichorioidal space, which communicates by means of the venæ vorticosæ, *v*, with Tenon's space, *t*, *t*; *s*, supravaginal space; *i*, intervaginal space; *ee*1, continuation of Tenon's capsule upon the tendons of the ocular muscles (lateral invagination).

occlusion of the minute openings by corpuscular elements which stick fast in them or by clots; (2) transformation of the delicate network into a tough and impermeable tissue by the remains of embryonic tissue or by inflammation; and finally (3) as a gross process, the apposition of tissue, usually the iris, which is applied to the inner surface of the ligamentum pectinatum and thus cuts it off from the cavity of the anterior chamber (see §§ 453 and 461). These changes, which form the basis of increase of tension in the human body, can also in part be produced experimentally, and thus an artificial glaucoma can be set up.

The *iris* must be considered as a subsidiary channel of outflow from the anterior chamber. The injection of india ink in a state of minutest subdivision into the anterior chamber shows that the particles of ink penetrate into the anterior layers of the iris,

[2] [According to A. Thomson, the ciliary and iris muscles by their traction on the scleral spur (see Fig. 147) open Schlemm's canal and the spaces of the ligamentum pectinatum, and so pump the aqueous out of the anterior chamber.—D.]

where they are then taken up by the blood-vessels and carried off. Penetration into the iris takes place chiefly through the openings on its anterior surface, namely, the crypts, through which the tissue of the iris is in free communication with the cavity of the anterior chamber. Several clinical observations are explained by this fact. When, for example, after an iridectomy the anterior chamber is filled with blood, this usually disappears rapidly wherever it lies upon the iris, which in fact takes up the blood, while it stays for a long time upon the capsule of the lens in the area occupied by the pupil and the coloboma.

The part played by the iris in carrying off the aqueous is usually regarded as insignificant, yet according to some the iris is just the part by which most of the aqueous is taken up, and the latter, moreover, is not discharged into Schlemm's canal, but by way of the lymph sheaths of the anterior ciliary veins.

13. The *posterior lymph passages* are: (1) The perichorioidal space (Fig. 1, *p*), i.e., the space between the chorioid and sclera in which lie the lax lamellæ of the lamina suprachorioidea. This space is continued outward along the vessels that pass through the sclera, especially along the venæ vorticosæ, and thus communicates with (2) Tenon's space (*t*), which lies between the sclera and Tenon's capsule. From this the lymph reaches the spaces which run along the optic nerve, namely, (3) the supravaginal space on the outer side of the dural sheath (*s*), and (4) the intervaginal space (*i*) between the sheaths of the optic nerve. Besides these there is assumed to be still another lymph passage in the posterior portion of the eye, which corresponds to the canalis hyaloideus or central canal of the vitreous (*h*) and which in the embryo lodges the hyaloid artery. Pathological processes argue the existence of such a lymph passage which runs in the vitreous straight back to the head of the optic nerve, for even in slight inflammations of the anterior portion of the eye the papilla of the optic nerve is found to be implicated, even when the posterior portion of the eye otherwise is still normal, so that we must assume that irritant substances from the focus of inflammation can get to the optic nerve first by a direct channel through the vitreous.

In any case, the posterior lymph passages carry only a very small fraction of the lymph out of the eye. Whether there are diseases which are referable to interference with this discharge of lymph posteriorly is uncertain.

VI. Intra-Ocular Pressure

14. The *intra-ocular pressure* is intimately connected with the lymph circulation. This pressure is practically the same in the three cavities of the eye, namely, the anterior and posterior chambers and the vitreous. No differences in pressure worth mentioning[3] can exist between the anterior and posterior chambers, since they would be at once equalized by way of the pupil. Again, any great differences in pressure between the cavity of the

[3] [This statement seems open to criticism. As Elliot points out, the very fact that there is a continuous inflow and outflow of liquid proves that the pressure at the point of entry is higher than at the point of exit. The difference in pressure, to be sure, is slight, but is important since, if it did not exist, there would be no flow at all.—D.]

two chambers and the vitreous are impossible, even though a diaphragm—the lens and the zonula—is interposed between the two, for this diaphragm can be displaced forward or backward too easily for such differences to occur, and, moreover, it is not perfectly impermeable to liquid. Considerable differences of pressure between the separate cavities of the eye can occur only under pathological conditions. For example, it may occur in the region of the anterior and posterior chambers in seclusion of the pupil, when the pressure in the posterior chamber rises high above that in the anterior. Quite considerable differences in pressure between the cavity of the two chambers and the vitreous may occur when the diaphragm that separates the two is rendered unyielding and impermeable by cyclitic membranes.

So far as normal conditions are concerned, we may for the purpose of simplifying the study of the conditions of pressure leave the lens out of consideration, and think of the eyeball as a capsule which comprises a single cavity filled with fluid. The fluid contained in the capsule exerts upon the inner surface of the latter a pressure which, in accordance with the laws of hydrostatics,[4] is transmitted with the same intensity in every direction, and hence presses with the same weight upon every unit of surface of the wall. A square millimetre of the posterior surface of the cornea has, therefore, the same pressure to bear as a square millimetre of any portion of the sclera.

15. The *intensity* of the intra-ocular pressure depends upon the relation between the capacity of the capsule and the amount of its contents. If the former becomes smaller, or the latter greater, the pressure rises, and vice versa. The capacity of the capsule of the eye is dependent on the area and shape of the cornea and sclera and on the elasticity of these membranes. Under physiological conditions, it undergoes such inconsiderable variations that the latter may be neglected and the capacity regarded as constant. The variations in intra-ocular pressure are hence referable to changes in the amount of *matter contained in the eyeball*, which may be increased or diminished. For example, the pressure at once sinks considerably when the aqueous has been discharged by puncture of the cornea.

Those portions of the contents of the eyeball whose amount is readily variable are the aqueous, the vitreous, and most of all the blood that circulates in the vessels of the inner tunics of the eye. Every increase or decrease of blood pressure in these vessels must result in a corresponding change in the ocular tension. It may be stated as a general proposition that the blood pressure in the vessels of the interior of the eye must always be a little higher than the intra-ocular pressure, because otherwise the arterial blood could not flow into the eye. When the blood pressure ceases, for example, at death, the tension of the eye drops at once. Owing to the small size of

[4] [Again, as Elliot shows, the question is one of hydrodynamics rather than hydrostatics. In other words, we are dealing with a liquid mass in motion and not at rest. Nor is the eyeball a closed capsule, but one within which the pressure must vary, though under normal conditions very slightly, at different points. This fact does not invalidate the essential accuracy of the statements in the text. —D.]

the vessels of the eye, the pulsatory variations of the blood pressure are so inconsiderable that the variations of the intra-ocular pressure which correspond to them are in large part compensated for by the elasticity of the fibrous coats of the eye. But even great and lasting changes in the blood pressure can affect the pressure in the eye for a moment only, and not permanently, because of the self-regulating action to be described later.

Under physiological conditions, variations in pressure can also be produced by the action of *external stress*. Every pressure exerted upon the eye from without increases the intra-ocular pressure by its own amount.

Hence, if with the finger we make gradually increasing pressure on the eyeball, and at the same time examine the eye with the ophthalmoscope, we see the retinal arteries become empty as soon as the pressure has reached a certain amount. For, owing to the external stress, the intra-ocular pressure has risen till it has become higher than the blood pressure in the central artery of the optic nerve, so that now both the latter and its branches are compressed. Under normal conditions the eye often undergoes pressure due to contraction of the eye muscles, and especially of the orbicularis palpebrarum when the lids are squeezed together. This, we may say, receives ocular demonstration in cases in which there is a thin place in the coats of the eye, for example, the thin floor of an ulcer or, better yet, a prolapse of the iris. Such a place, like a Marey's tambour, indicates by its protrusion and recession the variations of the intra-ocular pressure. In such cases we can see very clearly how in crying or coughing (owing to increase of the blood pressure), or how when the lids are squeezed together, the thin spot distends, and under certain conditions actually ruptures. The more prominent the eyeball is, the more powerfully can the recti muscles, and especially the orbicularis, press on it, and hence cataract operations, for example, are particularly dangerous in very protruding eyes, because in them vitreous can be expelled from the wound very readily by pressure on the part of the patient.

16. Although persistent variations of the blood pressure and contractions of the muscles surrounding the eye influence the ocular pressure, the latter, nevertheless, except for minimal variations, remains constant. That is, a process of *self regulation* of the pressure occurs, inasmuch as the outflow of fluid through the lymph passages (excretion) changes with the changing pressure. The rapidity of filtration of the aqueous of the eye depends, as in the case of any filter, upon the difference of pressure on the two sides of the filter. When, therefore, [either from external stress or internal accumulation] the pressure in the interior of the eye rises, more aqueous runs out, and vice versa. Moreover, the ocular pressure is also regulated, although, to be sure, more gradually, by the fact that it itself affects the fulness of the vessels in the interior of the eye, and thus indirectly affects secretion. When the ocular pressure rises, less blood flows into the interior of the eye, and because the vessels are less full the discharge of fluid into the interior of the eye diminishes. The reverse takes place when there is diminution of pressure. Such diminution is particularly great after escape of the aqueous. The direct consequence of this is a marked dilatation of the vessels in the interior of the eye, be-

cause the blood pressure in the latter is no longer supported in part by the intra-ocular pressure, but is supported simply by the vessel wall, which is not strong enough to withstand it, and hence distends. Owing to the dilatation of the vessels the secretion of fluid in the eye increases so that the normal pressure is soon restored.

This self regulation of the ocular pressure explains why an eye in which the lens is absent is no softer, inasmuch as the absent lens is replaced by aqueous, and why, on the other hand, when the volume of the lens increases (cataracta intumescens), no increase of pressure occurs, inasmuch as a correspondingly less amount of aqueous is present in the eye.

Another factor besides the circulation of blood and of ocular fluids in the interior of the eye, that is of significance for the maintenance of the intra-ocular pressure, is found in the osmotic processes. If the salt content and, consequently, the osmotic pressure of the blood increase (e.g., by the injection of salt or by the injection of salt solution into a vein), water is withdrawn from the eye into the blood, and the eye, consequently, becomes softer (Hertel). The increase in tension which we get, if we inject dilute acids into Tenon's space (Fischer), is produced by swelling of the sclera.

A certain regulating influence upon the ocular pressure belongs also to the nerves, the trigeminus and the sympathetic. Irritation of these nerves increases the ocular pressure; section of them decreases it. Hence extirpation of the cervical ganglion of the sympathetic has been recommended as a remedy for increase of tension, but has not proved efficacious, because the diminution in tension following division of the sympathetic is but transient. Many cases of sudden diminution in tension which are not otherwise explainable (for example, those occurring after contusions, erosions, and inflammation of the cornea), are referable to the influence of the nerves.

[Since external pressure on the eye produces increased outflow, the eye when the pressure is released is less full and hence softer than before. Hence, external pressure produced, e.g., by massage or a pressure bandage, temporarily reduces the intra-ocular pressure. When outflow is impeded, as in eyes that are glaucomatous or predisposed to glaucoma, this effect is less marked.—D.]

The miotics, as is well known, are employed in order to diminish the tension in glaucomatous eyes, while the mydriatics, on the other hand, can set up a glaucomatous increase of tension in eyes predisposed to glaucoma. [Even in normal eyes eserine or pilocarpine in ½-per-cent solution reduces the intra-ocular pressure by 3 to 8 mm. of mercury, the effect lasting from one to two hours or longer. Atropine has no effect on normal eyes. Cocaine diminishes the pressure from 1 to 4 mm. in normal eyes and still more in eyes that are hypotonic, while in glaucomatous eyes it acts like other mydriatics and increases the pressure. The intra-ocular pressure in also increased by sub-conjunctival injections, the effect lasting some hours (Lübs).—D.]

The intra-ocular pressure can be measured directly only by plunging a fine cannula into the interior of the eye and putting it in communication with a manometer. It has been found in this way that in a healthy human eye the pressure amounts to between 20 and 30 mm. of mercury; under pathological conditions it may rise to 100 mm. In practice, this method of measurement is not applicable, since it is associated with injury to the eye. If the coats of the eye are uninjured, however, we cannot test the ocular pressure, but only the *tension* to which the fibrous coat of the eye is exposed by the pressure acting upon it. For the methods of testing the tension of the eye, see § 81.

GENERAL PATHOLOGY OF THE EYE

17. Since in the special part of the work the diseases of the eye will be discussed according to their site and the anatomical changes that they set up, here, in their general consideration, the *etiology* will be taken as a starting point. The injurious influences which lead to morbid changes are in a very general way either physical, mechanical, chemical, or parasitic. Their effects, to be sure, cannot by any means be sharply differentiated in any individual case, and in most cases several of these injurious influences act in combination. Finally, as additional causes of disease should be considered degenerative processes and a congenital faulty constitution, both of which conditions in the last analysis are referable to the injurious influences first mentioned without its being precisely known in what way these latter act.

I. Physical Sources of Injury

(Heat, Light, Electricity)

18. The spectrum of any source of light along with the visible rays contains also those which are invisible. Adjoining the red end of the spectrum with its long wave-lengths are rays of still greater wave-length, the ultra-red or heat rays. Beyond the violet end with its short waves lie the ultra-violet rays of still shorter wave-length, which are chemically active. But visible waves of the spectrum also have both a thermic and a chemical action. When, therefore, irritation or inflammation of a tissue develops as a result of intense radiant energy, the injurious effect may depend either on the action of light, in the narrower sense of the word, or on a thermic or a chemical action, or on all three together. This can be determined in many cases by experiment, by passing light through special filters which transmit only one variety of rays. Heat rays do, but the luminous or ultra-violet rays do not pass without hindrance through a blackened plate of rock salt. On the other hand, heat rays are absorbed by water, especially by a solution of alum, and hence are not transmitted. The ultra-violet rays pass through rock crystal, but are kept back even by ordinary glass and to a still greater degree by a glass that is colored red or yellow, and also by fluorescent substances (e.g., a solution of quinine) and by certain hydrocarbons. Thus we are able to study independently of each other the action of rays of long and short wave-lengths upon the skin or on the eye.

19. The injurious effect of great *heat* in causing burns has always been known. To higher degrees of radiant heat are exposed particularly those who work by a fire, for example, workmen at steel forges. The skin pro-

tects itself against radiant heat by abundant sweating. The evaporation associated with this withdraws heat from the skin, and the same is true of the constantly moist surface of the eyeball, so that an injury of the superficial portions of the eye does not occur. How is it with the deeper parts? Through the refractive media of the eye the heat rays are refracted just like light rays, and are united into a focus situated on the retina, and they may develop there a considerable heat effect, just as we can by means of a strong lens set fire to a match held at its focus. But, to oppose this, is the fact that the media of the eye which contain such a large amount of water absorb the heat rays to such an extent that they scarcely reach the retina at all. [A large proportion of them—25 per cent according to Vogt—do pass through the cornea and sclera and reach the aqueous and superficial portions of the vitreous. Even so, they seem to cause trouble only when their action is very intense and is prolonged over a considerable period of time.—D.] Indeed, the only injury to the eye which can with probability be referred to radiant heat is glass-blowers' cataract (see § 487).

20. The *visible rays*, when they act too strongly, cause mainly a troublesome sensation of dazzling which produces blepharospasm and when it is of high degree is associated with actual pain in the eye. This pain due to dazzling cannot emanate from the retina, because the latter has no sensory nerves. These are present only in the anterior portions of the middle coat of the eye, i.e., in the ciliary body and the iris, which are both rich in such nerves. The ciliary body itself is not impinged upon by the light nor does it undergo change of any sort when light acts upon it, and hence it is not in question. The iris, on the other hand, contracts very energetically whenever the light acts strongly, so that the pain due to dazzling, like the pain that follows the instillation of eserine, is accounted for by the traction upon the nerves of the iris. [It seems questionable whether spasmodic contraction of the ciliary muscle may not also occur; not from the direct action of light, but in a reflex way. Moreover, the discomfort produced by excessive and ill-directed light is due not only to painful contraction of the iris, but also to conjunctival irritation (causing "sandiness" of the lids) and possibly, too, to fatigue of the extra-ocular muscles (Ferree). Probably part of the discomfort, particularly the conjunctival irritation, is due to the ultra-violet rays (see page 23).—D.]

Differing from the pain of dazzling, is *dazzling* itself, which is appreciated as a disturbance of sight.

It occurs when either too great an amount of light or too much diffuse light falls upon the retina. Excessive diffusion occurs either because there is an opacity in the refracting media of the eye or because the light is too intense. In the former case the disturbance of vision that takes place is explained as follows: In the normal eye the images of the objects in the visual field lie upon the retina, side by side and sharply separated from each other, the bright and dark parts in contrast. Now, if by means of a spot of opacity upon the cornea light is diffused uniformly over the whole retina, the distinction between

the light and dark portions of the retinal images becomes less striking. The following comparison may illustrate these conditions: In a well-taken photograph all details are seen sharply and distinctly. But if it is rather highly glazed, and we look at it obliquely, the glazing shines so that the details of the photograph can no longer be distinguished. As the glazing is perfectly transparent, the rays emanating from the photograph still arrive at our retina and produce there sharp images of the details of the photograph. But in addition there come numerous rays reflected from the surface of the glazing which so flood the whole retina with light that sharp retinal images are, so to speak, drowned out.

Vision is reduced for the same reason, and a sensation of dazzling develops when the illumination is much too intense. How is the diffusion of light effected in this case? The normal cornea is not, as is ordinarily assumed, absolutely transparent. We can see this from the fact that a portion of the cornea, which has light concentrated upon it by focal illumination, looks gray, insomuch that the tyro might suppose that there was a pathological opacity of the cornea. The cornea, therefore, always reflects a certain quantity of light. The like is true of the lens, and, generally, of all the refracting media of the eye. Because of this imperfect transparency of the refracting media, light diffusion takes place even in the normal eye, although, to be sure, under ordinary circumstances, it is too inconsiderable to excite notice. But when the illumination is very intense diffusion is disturbing because then an unusual amount of light diffuses over the retina. Moreover, if the illumination is very strong light also enters the interior of the eye through the non-transparent membranes (sclera and uvea) outside of the pupil, and adds to the dazzling.

Light that is too intense causes disturbance of sight not only by producing diffusion but also because the portions of the retina upon which it impinges become temporarily undersensitive, owing to the great consumption of visual substances. Everybody knows the troublesome after images which one gets so readily in looking at a sunset, and which cause disturbance in vision for some time. From prolonged looking at the sun, or other powerful sources of light (for example, electric light), a permanent disturbance of sight may result, there remaining for all time, at a point corresponding to the spot where the image of the sun was cast upon the retina (that is, as a rule, at the macula lutea), an area of indistinct vision, i.e., a central scotoma, so that minute vision, such for example as is required in reading, is permanently impaired. In serious cases of this kind, moreover, we find with the ophthalmoscope pigment changes in the region of the macula lutea, proving that actual pathological alterations of tissue were set up. After every eclipse of the sun, numerous cases of this sort are observed among persons who watched the eclipse with glasses which were not sufficiently smoked. Since the refractive media of the eye unite the sun's rays upon the retina to form an image of the sun (just as a glass lens does at its focus), it was formerly supposed that the retina was, so to speak, burned at the point at which the image of the sun was formed. But, we have learned above, the heat rays, on account of their absorption by the watery media, practically never reach the retina. Even for the ultra-violet rays the fact holds good, that they are kept back in good part by the media of the eye, so that the injury to the retina must, therefore, be attributed to the visible rays of the spectrum. Since under normal conditions these rays excite movement of the retinal cones and the retinal pigment, it can be understood how an increase of this process above a certain point may lead to pathological changes.

Another consequence of dazzling is *erythropsia* (red vision, from *ἐρυθρός*, red). When after being exposed to a strong light for quite a long time we go into a poorly-lighted room, we at once see the dark surfaces appear green, the bright surfaces (for instance a window in the room), vividly purple, as if there were a fire outside. Usually this phenomenon lasts only a few minutes, but in patients who have been operated on for cataract it may happen that the erythropsia extends over the whole day, or returns on the

following day in the morning or evening, so that the patients become very much concerned. Under ordinary conditions this phenomenon is observed only in persons with slightly pigmented and very sensitive eyes, and in them only to a very slight degree. It occurs more frequently when people are exposed to a prolonged dazzling from snow upon which the sun is shining, and if under these conditions the pupil is artificially dilated, erythropsia can be produced in anybody. But it is found most frequently in persons in whom the pupil has been enlarged by an operation (iridectomy) and particularly so, if at the same time the lens has been removed, i.e., in persons who have been operated on for cataract. It was through the latter, indeed, that we first learned to recognize erythropsia. When those who have been operated on for cataract lay aside their dark glasses some time after the operation and are then, for the first time, exposed to strong sunlight, they are frequently alarmed by the red vision. Erythropsia can be set up by the visible rays alone, but a simultaneous action of the ultra-violet rays is very probable, since the dazzling produced by light reflected from snow, which light is very rich in such rays, is particularly apt to produce this symptom. To the same effect is the circumstance that aphakic eyes are affected, since in these, as will be shown later, ultra-violet rays reach the retina in greater amount than they do in other eyes.

[Ocular discomfort, fatigue and drowsiness result from using the eyes by *insufficient illumination*.—D.]

21. The *ultra-violet rays* in sunlight are to a considerable extent absorbed by the atmosphere. Hence we find them in sunlight in greater amount the higher we are above the sea. The richness in ultra-violet rays of artificial sources of light varies. Poorest in this regard are the candle and the oil lamp, then follow in order the different kinds of Welsbach light, acetylene gas, the electric incandescent lights (the more so the more modern they are, i.e., the more light efficiency they have with the same strength of current), the arc lights, and lastly, the mercury-vapor lamp. The ultra-violet rays, if they enter the eye, are in large part absorbed by the refractive media of the latter. The lens, indeed, not only absorbs a portion of the rays, but also transforms another portion into rays of greater wave-length—visible rays—so that the lens itself emits a grayish-green light (fluorescence of the lens). Hence, aphakic eyes receive more ultra-violet rays upon the retina than do those which contain a lens; consequently they see the spectrum prolonged at its violet end, and they are easily affected with erythropsia. Since the lens absorbs or transforms so much ultra-violet light, it would be quite conceivable that it should be injured by rays of this sort, and Hess has actually been able to produce slight opacities of the lens by great and long-continued exposure of the eye to the rays of ultra-violet light. [Such opacities, it is said, are more readily produced in lenses containing small amounts of sugar or certain salts, and it is argued that diabetic and glass-blowers' cataract (see §§ 487, 488) and even ordinary senile cataract are set up by the effect of ultra-violet rays acting on lenses of altered composition (Burge). This view still requires confirmation.—D.] It is true of the retina, as of the lens, that it can be injured experimentally by ultra-violet light. But in man this occurs only under unusual circumstances and, even then, only in a transient way.

The case is otherwise with the action of ultra-violet light upon the exterior parts, the skin of the lids and the conjunctiva. It has been proved by experiment (Widmark) that what has been so long known as sunburn, i.e., the erythema of the skin which one gets from the prolonged action of the sunlight, is produced not by the heat rays nor the visible rays, but by the ultra-violet rays. In travelling over the glaciers one is especially apt to get sunburn (called glacier-burn) because, at great elevations above the sea, the sunlight is richer in ultra-violet rays, and this, moreover, is particularly the case with light that is reflected from the snow. When glacier-burn affects the eyes it is called *snow blindness*, because those who are affected by it are scarcely able to open their swollen and inflamed eyes. The same symptoms may be excited by intense electric light (in working with the flaming arc light, or with the arc produced by short circuit, etc.), when it bears the name of *ophthalmia electrica*. [A less intense degree of acute conjunctivitis is produced by a few minutes' exposure to the intense light of the arc used in electric welding. (Collis).—D.] Snow blindness and electric ophthalmia have this feature in common, that the patient feels nothing at first, and the inflammatory symptoms begin only after a latent period of from half a day to a day. These symptoms consist of swelling and redness of the lids and conjunctiva, and sometimes also of erosions of the cornea and contraction of the pupil, caused by the irritation. Dangerous as the condition appears to the laity, on account of the violent pain and the photophobia, it nevertheless passes off in a few days. Cold compresses, and the instillation of a weak solution of cocaine, to relieve the pain, suffice for its treatment.

Such violent inflammations apart, the ultra-violet rays when they act less strongly seem also able to produce slighter changes of a chronic nature. Many persons who previously could work well by artificial light get into trouble after the introduction of the electric light, especially one rich in ultra-violet rays. They have a sensation of heat and burning in the eyes, and the edges of the lids and the conjunctiva are hyperæmic. Some have thought that spring catarrh, a chronic inflammation of the conjunctiva, is attributable to the ultra-violet rays of sunlight, but weighty objections argue against such an assumption.

The injurious action of light is obviated by protective glasses (see § 45).

22. Of the *Röntgen* and *radium* rays when there has been too prolonged action, there has been observed, not only in experiments upon animals but also upon man, injury to the eye, namely, inflammation of the exterior parts and degeneration of the retina. The latent period in these cases is much longer than it is in the case of the action of the rays we have just been considering, being from one to three weeks. In the therapeutic application of these rays to the adnexa of the eye, the eye itself should be protected by a shield of lead.

Lightning when it strikes the eye produces in it external injuries, chiefly burns and the inflammations that result from them. Moreover, in the interior of the eye there may occur opacities of the lens and atrophy of the optic nerve, but these conditions commonly do not become manifest until some time after the accident. They are produced not like the external injuries by the action of heat, but by the power possessed by the electric discharge to kill living cells even without the development of heat. Hess has proved this for the lens, showing that by electric shocks the epithelium of the lens capsule is made to die, the result being a clouding of the lens.

II. Mechanical Injuries

23. Mechanical injuries of acute onset are wounds; those that act slowly are pressure, traction, etc. Into this matter we will not enter here, but only into one property of the eye, which belongs to it alone among all the organs of our body, and which leads to the most serious mechanical disturbances. The eye, in fact, is in large part a hollow structure filled with liquid which has no free communication externally, as the outflow of liquids takes place simply by filtration. The slight resilience of the envelopes of the eye affords a close comparison with the capsule of the skull. Increase of tension, due to a disproportion between capacity and contents, is a frequent occurrence both in the capsule of the eyeball and in that of the skull, and the consequences are analogous in both. When in youth the capsule is still capable of expansion, it gives way and enlarges (the eye in the case of hydrophthalmus, the skull in the case of hydrocephalus), so that the contents have less to suffer from the pressure. On the other hand, such injury from pressure is the more marked the more rigid the capsule has become with advancing age (in the eye by greater rigidity of the sclera, in the skull by ossification of the sutures). Then as a result of the pressure the delicate nerve elements are the first to suffer (in the eye the optic nerve in the case of glaucoma; in the skull, the brain in the case of intracranial increase of pressure).

The lymph of the brain can flow off not only to the spinal cord but also forward in the intervaginal space of the optic nerve. Owing to the fact that in intracranial increase of tension lymph accumulates in this space under high pressure, there is afforded a mechanical source of injury, which frequently causes the blindness occurring in diseases of the brain (choked disk).

III. Chemical Sources of Injury

24. Chemical injuries are by far the most frequent cause of pathological processes. The vital phenomena going on in cells are complicated chemical processes which are affected by every alteration in the chemical composition of the liquid surrounding them. In a more extended sense of the term, parasitic diseases also act to produce chemical injury. In itself the bacterium would add as little harm to the tissues and would be tolerated with the same absence of reaction as a granule of india ink, pro-

vided that like the latter it were chemically indifferent. But by the production of toxic substances micro-organisms produce an injurious effect locally; by the diffusion of these substances they act on the neighboring parts; and by transmission of these substances through the lymph and blood-vessels they act on remote organs. By its toxic products the diphtheria bacillus sets up the local inflammation in the throat, and by the toxins that pass over into the circulation it causes the fever and the paralysis. Hence, diseases of chemical and parasitic origin cannot be sharply differentiated, and it is only from practical considerations that we will here regard as diseases of chemical origin those only in which the coincident action of microbes is, according to our present knowledge, excluded.

Chemically active substances working upon the surface of the eye produce in it corrosion or inflammation. In the same way, chemically active bodies (for example, chips of metal which penetrate into the interior of the eye) commonly produce severe inflammation. Again, such inflammation is sometimes also set up by substances which in other places do not act to cause chemical irritation. For instance, atropine dropped into the conjunctival sac produces in some persons follicular catarrh or erythematous swelling of the lid. We must then suppose that there is a special individual susceptibility, which, in fact, makes itself so very evident in other parts also in the action of drugs (an example is the drug exanthems). This individual susceptibility plays a particularly great part in the case of organic poisons. It may be that some persons have in the fluids of their body substances which act to protect them against certain organic poisons and which are absent in other people. [It also happens that some are unusually susceptible to a poison, because their tissues having once been affected by it are sensitized, or put into a state in which they react more readily to the poison than before. This sensitization, called *anaphylaxis*, is illustrated in the susceptibility of tuberculous subjects to doses of tuberculin, too small to affect the healthy (see page 69).—D.]

A good example of this sort of susceptibility is the conjunctivitis which accompanies hay fever. This is produced by the pollen granules of the gramineæ which, passing through the air, get upon the conjunctiva and the nasal mucous membrane, and there cause inflammation by means of an organic toxic substance contained in them. The proof of the toxic nature of this inflammation is afforded by the fact that it is possible to prepare an antitoxin against it. But there are only a few people who are subject to hay fever, that is, who are susceptible to the corresponding toxin, and whose susceptibility is actually increased as a result of having one attack of the disease. One who previously free from hay fever comes into a region where it is frequent and acquires it there, in the following years gets it again readily, even when at home. [Other ocular diseases plausibly attributed to anaphylaxis are phlyctænular conjunctivitis (Verhoeff), parenchymatous keratitis (Derby), and sympathetic ophthalmia (Elschnig).—D.]

Conversely, against other organic poisons a certain *immunity* develops in the course of time. This, for example, is true of the jequirity bean, the infusion of which when placed upon the conjunctiva produces, as a result of the abrin contained in it, a

violent conjunctivitis, which, in consonance with its toxic origin, does not set in until after a certain period of latency. Not only can we prepare an antitoxin combating the action of abrin (Roemer) but after a jequirity conjunctivitis has once occurred it is not possible to produce a conjunctivitis by a second application of the agent except after a rather long interval. The like is true of dionin.

A combined mechanical and chemical action probably lies at the bottom of those cases in which conjunctivitis is set up by small foreign bodies of a special sort (the hairs of the caterpillar, the hairs from certain species of primula, the dust from hyacinth bulbs).

In other cases, the chemical noxa do not make their attack upon the surface, but from the blood, from which they are thrown out into the tissues that become affected. Two agents that are employed in skin disease, chrysarobin and naphthalin, are good examples of this. Chrysarobin often causes conjunctivitis, not only when it comes into contact with the conjunctiva locally but also when, starting from remote parts of the body, it undergoes absorption. Naphthalin, when ingested into the body, causes retinitis and opacity of the lens.

Chronic inflammations or degenerative processes are produced by chemical poisons more frequently than are acute inflammations (so in the case of alcohol, tobacco and many other poisons).

25. Autotoxis.—In addition to the poisons that are brought into it, the body is also harmed by those which it itself produces. A transition form between the introduced and the self-produced poisons are those which are developed in the digestive canal by abnormal processes of decomposition from nutrient materials which have been introduced there and are not in themselves toxic. By some, the cause of many diseases has been regarded as consisting in such *intestinal auto-intoxications.*

Hitherto, however, but very few facts have been surely demonstrated, and intestinal intoxication is much too often forced to serve as a convenient etiology for the most various diseases. [It seems to be, at least occasionally, the cause of iritis and other diseases of the uvea, including glaucoma. It is often a question, however, whether the uveal disease in this case is due to the absorption of toxic food products, produced by faulty metabolism in the intestines, or to absorption of the toxins of the bacteria which cause the faulty metabolism.—D.]

Likewise unproven as yet is the contention that eye diseases, and in particular chronic iritis, are caused by disease of the oral mucous membrane due to bad teeth, or by disease of the tonsils or the accessory cavities of the nose. [In the opinion of the translator, however, the proof of this contention is now incontestably established by a series of cases, several of which he has seen himself.—D.] Better established and longest known is the etiologic part played by the metabolic diseases proper, like gout, diabetes, etc., even though the precise way in which the general disorder of metabolism causes the local disease (e.g., the iritis or retinitis in the case of the eye) is still obscure.

Many *diseases of individual organs* lead to diseases of the eye, which not infrequently are the first thing that directs the attention of the physician to the causal affection. It is an acquisition of most recent times to have recognized that in many cases these inter-relations depend upon altered chemical action. We owe this knowledge in part to the discovery of the function of the glands which possess an interior secretion; disease being caused both by the increase of this secretion above the normal and by its cessation. Again, examination with sera has shown how, by utilizing the roundabout passage through another animal, we can study the interchange of action in the organs

of the same body. In this way it has been demonstrated that diseased organs can deposit in the blood the products of their metabolism or their decomposition which cause, almost like a regular chemical reaction, definite changes in other organs or tissues. Here is still a wide field for investigation lying before us. Only a few of the best known facts can be adduced as examples here.

Upon hypersecretion of the thyroid gland depends Basedow's disease, which in fact produces important symptoms in the eye. Deficiency of secretion of the thyroid gland on account of congenital or acquired absence of the latter leads to myxœdema. [According to Dunn, acquired hypothyroidism causes certain forms of cyclitis, especially that occurring in interstitial keratitis due to hereditary syphilis.—D.] Cessation of function of the parathyroid glands is the cause of tetany, among the consequences of which are opacity of the lens (particularly lamellar cataract) and inflammation of the optic nerve. Grave disturbances of nutrition, above all acromegaly, are produced by degeneration of the hypophysis, in which case the eye suffers, although, to be sure, indirectly, owing to pressure of the enlarged gland upon the optic nerve.

While it is only as a rare occurrence that we have to do with diseases of the eye that result from secretory disturbances in the glands having an interior secretion, cases are very frequent in which the eye, owing to chemical influence, is implicated in disease of other organs. The most frequent case of this sort (equally important for the ophthalmologist and the internist) is the inflammation of the retina occurring in nephritis. Up to a short time ago, the connection between the two has been sought for in vascular changes or in the increase of blood pressure, conditions which both usually accompany chronic nephritis; or it has been supposed to consist in the retention in the body of sodium chloride, due to its insufficient excretion by the kidneys. Although these factors cannot perhaps be altogether disregarded, probably the main injury to the retina is done by toxic matter which is formed by the decomposition of kidney substance and which possesses a particular affinity for the tissues of the retina. As is known, we can by injecting the blood of an animal of genus A into an animal of genus B obtain in the latter a serum which, if injected into A, dissolves its blood corpuscles. ("We obtain hæmolysin for A by immunizing B with A's blood.") If, now, we inject into B a kidney extract obtained from A (B being thus immunized), the serum of B if injected into A causes in the latter not only nephritis, but also, if it is injected into the carotid, retinitis (Zur Nedden). The nephrotoxin formed in B acts chiefly upon the kidneys of A because in the animal B, which has been rendered immune with the kidney extract, there are formed antitoxins against all of the albuminous substances present in the kidney. That the retina, too, is diseased is due to the fact that the retina has one or two kinds of albumin, which it possesses in common with the kidneys, and which, therefore, are attacked by the nephrotoxin. That the metabolic products of the normal kidney do not injure the retina of the same animal, presupposes that the organs of one and the same body (or of another individual of the same species) are immune to its own metabolic products. (Unless this is presupposed, the life of the organism would be impossible.) But when there is disease of an organ with disintegration of tissue, this immunity may not suffice. [And an excessive susceptibility to disease may be set up by decomposition products originating in the patient's own organism (*auto-anaphylaxis*).—D.] These conclusions still move very much in the region of hypotheses, but in any case the experiments which form their basis throw a new light upon the inter-connection of the organs and their dependence upon one another through the agency of chemical processes.

Diseases of the eye sometimes develop not only as a result of diseases of individual organs, but also in consequence of *physiological processes* taking place in them. As an example may be adduced the inflammation of the optic nerve, which sometimes occurs in the course of pregnancy or lactation, without there being any other demonstrable

disease of the body. It is conjectured that in this case also the fault lies with the action of toxins, which either have been formed as an exceptional thing in the course of the physiological processes occurring in the organs concerned, or toward which the eye does not possess its normal immunity.

IV. Parasitic Causes of Injury

26. Varieties of Infection.—Inflammation of the eye develops most frequently as a result of infections. In almost every person with a high fever we find the conjunctiva reddened and the eye glued up; i.e., we find a slight conjunctivitis. To a particularly great extent is this the case in measles. Many acute infectious diseases, however, entail for the eye consequences that are considerably more serious, namely, metastatic inflammation of the uvea and retina and inflammation of the optic nerve. The chronic infectious diseases implicate the eye even more frequently than do the acute. It is well known what a prominent part as regards eye diseases falls to the share of tuberculosis (including scrofula) and syphilis. Sometimes the eye complication follows the primary disease after a number of years, as, for example, in the case of the optic-nerve atrophy in tabes, whose cause (syphilis) has apparently been cured long before. The participation of the individual organs in an infectious disease takes place in two ways—by direct invasion of the organs by the microbes, and by the action of the toxins produced by the disease.

The micro-organisms may either attack the eye itself primarily or make an entry at some other portion of the body, and the eye is implicated secondarily. The former is denoted as ectogenous, the latter as endogenous infection. In both cases a distinction must be made, as has been emphasized more at length above, between the direct action of micro-organisms in situ and their remote effects. In the direct infections of the eye we are pretty well informed regarding these relations, because there are a sufficient number of dissections at hand. In ulcus serpens, for example, the suppuration in the cornea is excited by the bacteria which are there present, but the associated iritis and the accumulation of pus in the anterior chamber are produced by remote action, the bacterial toxins, which are diffused through the cornea into the anterior chamber, causing the iris to become inflamed. Much less known are the relations in cases of the second kind, in which the eye simply participates in a general infection. In the two most important chronic infectious diseases, tuberculosis and syphilis, the eye is very frequently affected. In some cases of this sort the tubercle bacilli or the spirochætæ are found in the tissues of the eye, but in cases still more numerous their demonstration is not as yet feasible, and it will be the task of some future time to make the matter clear. Until then, the clinician will often have to content himself simply with bringing a given affection of the eyes into etiological connection with a general infection.

27. (*a*) **Ectogenous Infection.**—The exterior portions of the eye, the margins of the lids, the conjunctiva, and the cornea, can be attacked by micro-organisms directly, and these may either get a foothold upon the intact surface or may require a lesion of the epithelium in order to penetrate into the tissues and injure the latter. The interior of the eye is not accessible to ectogenous infection until traumatism or ulcerative perforation enables the micro-organisms to enter.

The *border of the lids* presents favorable conditions for the lodgement of microbes. Between the cilia there collect the desquamated epithelium and also the secretion of the Meibomian glands, both of which in pathological cases form scales and crusts, and as an additional factor there is the moistening by tears. The border of the lid, like the skin in general, quite regularly harbors the staphylococcus albus and also the xerosis bacillus. The latter is a bacillus of the group of diphtheria bacilli, and can be distinguished from the latter with certainty only by animal experimentation, which shows whether the injected cultures have a pathological action or not. Both the staphylococcus albus and the xerosis bacillus are under ordinary circumstances non-pathogenic for the eye. In the case of inflammatory disease, such as blepharitis ulcerosa and acne pustule of the border of the lid (hordeolum), the staphylococcus aureus is also present. Moreover, being a part of the external skin, the border of the lid may participate in all diseases of the skin and may then harbor the morbific germs which characterize the latter.

The *conjunctiva* of the eyeball and the cornea form a moist surface which is exposed to the air and upon which the particles of dust and the germs which are conveyed through the air remain and adhere. Hence the conjunctival sac is almost never free from germs and, indeed, at times the most varied kinds of germs, saprophytic and pathogenic, occur there. But they do not multiply in the conjunctiva, because by the winking movement of the lids the surface of the eye is swept at regular intervals, and the tears together with the germs contained in them are carried into the nose. This can be shown if cultures of readily demonstrable non-pathogenic bacteria (e.g., the bacterium prodigiosum) are placed in the conjunctival sac. Soon these can be demonstrated in greater and greater quantity in the nasal mucus, while they disappear at the same rate from the conjunctival sac. Furthermore, the constant gradual current of tears passing down through the lachrymo-nasal duct prevents germs from ascending against the current from the nose into the conjunctival sac. When, owing to deficient winking, the drainage of tears is arrested, the amount of germs contained in the conjunctival sac at once increases. This amount is, therefore, greater in the morning than in the evening, and is particularly apt to be increased by a bandage because the latter checks the movement of the lids. By increasing the germs prolonged bandaging of the eye suffices of itself to produce a catarrh of the conjunctiva. We see this often in the clinic when

after a cataract operation the other eye, which has not been operated upon, is bandaged for one or two days, whereupon this eye often shows a catarrh of the conjunctiva which was not present before. Hence, many operators before every major eye operation apply a test bandage for a day to see whether the eye will react to this with a catarrhal irritation.

Besides the mechanical part which the tears play with regard to the bacteria, a bactericidal action has also been attributed to them, but this if present at all is extremely slight.

Although the most various kinds of germs fall from the air into the conjunctival sac, yet, as is the case on the border of the lids, the non-pathogenic white staphylococci and the xerosis bacilli are the only germs found as regular inhabitants of the conjunctival sac, and they actually occur in smaller quantities in the conjunctival sac just because they are continually removed by the tears. Of pathogenic bacteria only the pneumococcus and the streptococcus occur with any degree of frequency on the conjunctiva (in at most 5 per cent of the cases). When, as is usually the case, the conjunctiva is not affected by these germs, they do not as a rule lead to infections of wounds on the eyeball. A healthy organism, owing to its normal protective forces is, in fact, a match for some few germs even if they are pathogenic. Hence, even in the pre-aseptic time it was comparatively seldom that one had to complain of infection in operations on the eyeball. If such an infection did set in, this usually took place, as we know from experiment, not from the germs in the conjunctival sac—it being pre-supposed that the latter was normal—but from dirty instruments. [Even when the conjunctiva is not normal, infection of wounds of the eyeball is comparatively rare. This is shown by the results of cataract operations in Palestine, where, although there is scarcely an eye that is free from trachoma or conjunctivitis, post-operative infection is rare (Butler). —D.]

The *inflammations of the conjunctiva* are in very great part caused by special micro-organisms, some of which have the peculiarity of not being pathogenic either for other mucous membranes in man or for animals at all (some monkeys, perhaps, excepted). In this category belong the ordinary exciting agents of acute conjunctival catarrh, namely, the bacillus of Koch-Weeks and the diplobacillus of Morax-Axenfeld. Others are dangerous for a few other mucous membranes besides the conjunctiva and mainly for the mucous membrane of the uro-genital tract, as is the case with the gonococcus. In contrast with these most frequent morbific agents, others which are generally pathogenic, like the pneumococci, streptococci, diphtheria bacilli, etc., take a subordinate place. [The pneumococcus, however, is a fairly frequent cause of conjunctivitis. Two very frequent infections, specific for the eye, namely, trachoma and inclusion blennorrhœa, differ from all the others in that they are transmitted by a filtrable virus, i.e., one that contains no demonstrable micro-organisms (Axenfeld).—D.]

Besides ectogenous, endogenous infection occurs as a cause of conjunctivitis (the conjunctivitis of measles has been adduced above as an example of this, and another example is the metastatic gonorrhœal conjunctivitis), but these cases are rare in comparison with those of ectogenous infection.

Without there being any special infection from without, the amount of germs contained in the conjunctiva is greatly increased by the stagnation of tears, which accompanies diseases of the lachrymal sac. In the healthy *lachrymal sac*, inasmuch as the tears do not stay in it, disease of the mucous membrane is not produced by the germs which constantly arrive with the tears from the conjunctival sac into the lachrymal sac. In fact, even in inflammations of the conjunctiva, the lachrymal sac is usually not infected by the germs coming from the conjunctiva. This condition changes at once when, owing to stenosis of the lachrymo-nasal duct, the tears stagnate in the lachrymal passages. Then the germs multiply, and cause a catarrhal disease of the mucous membrane of the lachrymal tract (dacryocystitis chronica). Then in the contents of the lachrymal sac there are found germs, the pneumococci being the most numerous, and after them the staphylococci with other pyogenic bacteria. Owing to the fact that the tears now stagnate in the conjunctival sac, too, the latter contains the same bacterial flora, so that wounds accidentally made become infected readily (ulcus serpens) and so, too, operations on the eye are readily followed by suppuration.

28. The *cornea*, being a tissue placed upon the surface of the body, is, just like the conjunctiva, more in danger of ectogenous than endogenous infection. From the former, develop in the first place many of the corneal ulcers which are so frequent. With regard to ectogenous infection there is an apparent contrast between the conjunctiva and the cornea, in that the bacteria which get into the conjunctival sac are able to infect the conjunctiva, even when intact, while it is assumed that for the cornea a defect of the epithelium is necessary for infection to take place. It would follow from this that the epithelium of the cornea is more resistant to germs than is the epithelium of the conjunctiva. But perhaps the difference is only apparent. For while in the conjunctival sac the germs can linger for a time, they are at once swept by the lids off the smooth surface of the cornea, and it is only when depressions are made in the latter, as a result of small defects in the surface, that the germs can remain lying in these. These epithelial defects may be caused by a slight traumatism. The cornea, just like the rest of the surface of the body, is exposed to external injuries, but it has a most delicate epithelium, which is quite soft and besides is seated on the smooth Bowman's membrane, so that it can be readily stripped off en masse. Epithelial lesions of the cornea, therefore, are among the most frequent of occurrences.

The body that causes the injury may itself inoculate the cornea directly with bacteria, as is probably to be assumed for many cases of deep or actually perforating

wounds of the cornea, to which suppuration of the edges of the wound succeeds. In mere epithelial lesions, a simultaneous inoculation with the micro-organism probably occurs but rarely (e.g., in the infection of the cornea by aspergillus fumigatus). Ordinarily we have to deal with a secondary invasion of bacteria into the small wound, for we find the infection generally caused by bacteria which scarcely ever occur on the foreign bodies that cause the injury, but occur very commonly in the conjunctival sac. The most frequent of these bacteria requiring mention is the pneumococcus, which, especially in disease of the lachrymal sac, is to be found regularly in the conjunctival sac, and is the usual exciting cause of ulcus serpens. Of other bacteria which occur in the diseased conjunctival sac and which then also can excite inflammation of the cornea, should be particularly mentioned the diplobacilli of Morax-Axenfeld [also in Palestine and Egypt the Koch-Weeks bacillus and the extra-genital gonococcus.—D.].

We call the cases in which an inflammation of the cornea sets in as a result of a conjunctival lesion secondary keratitis, in contrast with primary keratitis, in which the cornea is the first point of attack of the injurious agent. In the cases of secondary keratitis traumatism is not required in order to produce a defect of the epithelium, but this defect may also develop because, owing to the abundant secretion in the conjunctival sac, the corneal epithelium is macerated and in places exfoliated, so that access to the corneal tissues is opened to the germs present in the tissue. Hence we particularly often find secondary corneal ulcers near the margin of the cornea. For in a severe case of conjunctivitis the swollen limbus rises steeply from the cornea, so that here a dead angle is produced, in which the secretion remains lying because it cannot be brushed away by the movement of the lids. Consequently the corneal epithelium is macerated in this spot most of all. Infection of the cornea may be produced by the exciting cause of the simultaneously present conjunctivitis, but may also be produced by any other pyogenic agent. Thus in conjunctivitis gonorrhoica and diphtherica, we commonly find in the corneal ulcers not the specific bacteria but the ordinary pyogenic germs.

In suppurative processes in the cornea, besides the bacteria above mentioned, there have been found as isolated instances others of very different kinds. It is interesting that among them occur those which elsewhere are not pathogenic at all (e.g., the bacillus subtilis) or which so far have been found only in the cornea (the bacillus of Zur Nedden).

29. The ectogenous infection of the *interior of the eye* as a rule takes place only when the eyeball is laid open, either by perforating injury or by a perforating ulcer of the cornea. In exceptional cases, a very thin place in the envelopes of the eye may enable the bacteria to enter, this, for example, happening in subconjunctival rupture of the sclera, in which defects of the epithelium and minute splits in the conjunctiva might form the portal of entry. Particularly dangerous are old scars of the cornea, with inclusion of the iris, when the scar is protruding and thinned. By the movement of the lids the epithelium may be injured at the most prominent portion of the scar, and the germs which enter there get not into the cornea but at once into the included iris and thus into the interior of the eye ("late infection of the eye").

In recent injuries either the body that causes the injury carries the germs into the eye (primary infection), or these germs during the succeeding days, as long as the wound remains unclosed, enter from the conjunctiva (secondary infection). In perforating injuries primary infection

is the more frequent process, in contradistinction to the slight traumatisms (erosions) of the cornea, in which, as above mentioned, secondary infection is the rule. Whether a primary or a secondary infection lies at the basis of an inflammation can in part be determined by the period at which the inflammation sets in. In a primary infection of the eye by pyogenic agents, a severe inflammation begins in twenty-four or at most forty-eight hours. A later outbreak of the inflammation argues a later onset of infection, i.e., a secondary infection.

30. The eye is not a single cavity, but is divided by a diaphragm consisting of the lens and the zonula into an anterior and a posterior segment. As regards infection the two behave in different ways.

Perforating wounds in the *anterior division of the eye* often heal without inflammation, even when they are produced by foreign bodies that are not sterile, because the aqueous which gushes out at once effects a natural irrigation and cleansing of the wound. (Thus it is explained why minute perforating punctured wounds of the cornea, in which the aqueous does not escape, are more dangerous than large incised wounds. In the pre-aseptic time discission of a secondary cataract was in the hands of some operators a more dangerous procedure than the cataract operation itself.) Even the very frequently occurring perforation of the cornea by ulcers but rarely leads to purulent infection of the interior of the eye, because, from the moment of perforation up to the closure of the opening, aqueous continually oozes out and impedes the progress of germs in the opposite direction.

Quite different is the behavior of the *posterior division of the eye* with regard to a perforating injury. The vitreous does not flow out in any great quantity, but lies in the wound. The latter, therefore, is not irrigated—in fact, the protruding vitreous affords subsequently a convenient path for the entrance of germs. The bacteria arriving in the vitreous find here an excellent nutrient material, the more so since the natural protective substances are absent from the vitreous. Hence many bacteria which otherwise are not pathogenic for the body, i.e., which are saprophytes, excite severe inflammation when introduced into the vitreous. The bacillus subtilis (hay bacillus) is the saprophyte that oftenest causes a severe inflammation (usually a panophthalmitis).

Not only does infection develop more readily in the posterior division of the eye than in the anterior, but it has much more serious results in the former. Even a severe inflammation in the anterior segment does not necessarily lead to blindness. Its consequences, posterior synechiæ and pupillary membranes and even a secondary opacity of the lens, can in many cases be removed by operation. An infection in the vitreous cavity leads in slight cases to a plastic, in severe cases to a purulent, inflammation. In the latter case the pus may either become encapsulated (abscess of the vitreous) or

break through to the outside (panophthalmitis). Even in the slightest cases, those of plastic inflammation, the eye usually becomes blind, because the exudates subsequently shrink and detach the retina from its bed.

From what has been said it can be understood that operations in the anterior segment of the eye could be made with good results even in the pre-aseptic time, while any manipulation in the vitreous cavity was scouted, because, as experience showed, it was always followed by suppuration of the eye. The aseptic method has made a transformation in this regard, but for traumatic lesions the old law still holds good.

While infection of the interior of the eye commonly affects only one of the two segments of the eyeball, this is not true of the subsequent inflammation. With regard to this, three cases occur: 1. The inflammation remains absolutely confined to one segment. We may see in the depth of the vitreous an abscess develop about a foreign body while the anterior segment remains quite normal. To be sure, cases of this kind in ectogenous infection are very rare; in endogenous infection they occur more often. Particularly in the metastatic inflammation, which is produced by the meningococcus and which accompanies cerebrospinal meningitis, we not so very rarely see the yellow reflex of pus in the depth of the eye while the anterior segment is normal. Of course, too, the converse may occur (i.e., suppuration in the confines of the anterior chamber while the posterior division is intact), only this cannot commonly be proved, since a view into the deeper parts is prevented by the exudate. 2. In infection of one segment there develops in the other a somewhat less severe inflammation due to the action of toxins which diffuse from the primary focus of inflammation through the diaphragm into the other segment. This is the most frequent case. With infections of the vitreous cavity there is present in the anterior segment an iritis. Similarly with infection of the anterior segment there develops a non-purulent exudate in the anterior portion of the vitreous, or, in the lightest cases, there develops at least hyperæmia of the retina and optic nerve. This latter is found at times as a remote effect even in non-perforating ulcers of the cornea. 3. The agents which excite infection actually pass from one segment to the other and lead to a severe inflammation there.

Which of the above-mentioned cases occurs depends mainly on two circumstances, the virulence of the infection and the density of the diaphragm. The latter is diminished to the greatest extent in case of absence of the lens (e.g., in an eye that has been operated on for cataract) and to a less extent in the event of an injury to the lens. In cases of the latter sort, there is the additional factor that the swelling lens masses give a much better nutrient material for bacteria than does the aqueous. It has been shown by experiment that the same infection situated in the region of the anterior chamber has much more serious consequences if the capsule of the lens has previously been ruptured. It is an old experience that perforating injuries with opening of the lens capsule are more often followed by severe inflammation than are precisely similar injuries with an intact lens. For the same reason in the pre-aseptic time the operation for cataract was followed in a certain number of cases by purulent infection, while this almost never took place after an iridectomy.

The bacteria that enter the anterior chamber multiply in the aqueous and often also grow out into the iris. The bacteria which get into the vitreous grow abundantly in the latter, but do not ordinarily pass into the inner coats of the eye. The purulent inflammation of the latter depends, therefore, on the remote action of the bacteria present in the vitreous. The inflammation consequently affects mainly the inner surface of these membranes, i.e., the retinal coating of the ciliary body and the retina itself,

on which account the name endophthalmitis is a proper one for this sort of inflammation. When the bacteria are particularly abundant or virulent, necrosis of the coats of the eye develops, starting from their inner surface. Necrosis of the cornea leads to the entrance into it of leucocytes and to a demarcating inflammation with the clinical picture of a ring abscess. Necrosis of the inner coats of the eye in the posterior segment affects first the retina, then, when of great intensity, the chorioid also, and last of all the sclera, and thus paves the way for perforation of the coats of the eye in panophthalmitis.

Apart from the question of its situation (anterior chamber or vitreous) and of individual susceptibility, the severity of the inflammation depends mainly on the quantity and virulence of the invading bacteria. In the traumatic inflammations the most frequent exciting cause of purulent inflammation is the streptococcus and secondly the bacillus subtilis; in operative injuries the pneumococcus comes first and the streptococcus second.

Slighter inflammations also occur after injuries and operations. Such inflammations set in only after a rather long period of incubation, sometimes indeed not till after the lapse of weeks, and run a milder course. In place of the purulent exudate there appears a delicate plastic exudate or precipitate. However, even these cases of insidious inflammation may lead ultimately to the destruction of the eye. In exceptional instances such mild cases may be produced by the ordinary pyogenic germs, while in other cases are found bacteria which are less virulent or which in other situations are not pathogenic at all. In other cases, again, it is not possible to demonstrate the presence of any bacteria whatever.

31. (*b*) **Endogenous Infection.**—Endogenous infection consists in this, that pathogenic germs get into the blood and are carried with it into the vessels of the eye, where they settle. The nature of the eye inflammation depends mainly upon the nature of the germ. The pyogenic germs cause a purulent inflammation of the eye, which generally passes over into panophthalmitis. It is these inflammations that are known as metastatic inflammations in the narrower sense of the term.

Metastatic ophthalmia is produced either by ordinary pyogenic germs, among which the streptococci rank first and the pneumococci second, or by bacteria which are specific for certain definite diseases (e.g., the meningococcus, the pneumobacillus, the influenza bacillus, the typhoid bacillus, etc.). Moreover, a mixed infection may also occur. The diseases caused by these bacteria, and above all puerperal fever and ordinary pyæmia, are the main cause of metastatic ophthalmia. But sometimes—as, for example, in the case of an inconsiderable attack of influenza or a very small purulent focus somewhere in the body—the original disease may develop in so mild a guise that it is overlooked, and the panophthalmitis apparently develops spontaneously (cryptogenous metastatic ophthalmia).

Although it is the rule that endogenous infection with pyogenic germs produces a purulent inflammation of the eye, yet, just as has been stated to hold good for ectogenous infection, exceptions occur in the sense that sometimes the inflammation shows a light character, and in fact may be limited to individual foci in the chorioid or retina which go on to healing. [This seems to be even more the case in endogenous infection; germs that would cause suppuration, if injected into the eye, producing only chronic plastic inflammation when reaching the latter by endogenous paths (Selenkowsky). Examples of

this are plastic iritis and similar affections caused by endogenous infection with germs, especially streptococci of tonsillar and dental origin and with influenza bacilli.—D.] A mild course is comparatively frequent in cerebrospinal meningitis, and it is the rule in relapsing fever, whose exciting cause, however, the spirillum, is not one of the pyogenic germs. In relapsing fever the eye is implicated usually under the form of a benign irido-cyclitis. These cases form a transition to the second group of cases of endogenous infection, which are caused by the germs that excite chronic infectious diseases.

32. Of *chronic infectious diseases* must be mentioned in the first place tuberculosis (lepra resembles it), syphilis, and gonorrhœa, when by entrance into the blood channels it has led to general infection; [also the so-called rheumatic affections, which are probably in most instances due to infection either with the gonococcus or the less virulent varieties of streptococcus. —D.]. These diseases are complicated by eye inflammations which, in conformity with the pathogenic properties of their exciting germs, are not acute and purulent but chronic and non-purulent. The different membranes of the eye are attacked by these inflammations with different degrees of violence, and this does not depend simply on the vascularity of the membrane, for the non-vascular cornea is often affected. It is assumed that the microbes in question or their toxins possess a differing affinity for the individual membranes of the eye. By far the oftenest to suffer is the uvea, and in this the iris and ciliary body suffer the most, the chorioid less. Tuberculous, syphilitic, and gonorrhœal irido-cyclitis are frequent diseases.

The way in which the uvea participates in the general infection, whether by the presence of the bacteria themselves in the tissue or by the mere action of toxins, has been but little investigated as yet and is probably not always the same. In tuberculosis at times iritis occurs with evident tubercle nodules which without doubt are caused by the bacilli themselves; but still more often we find a chronic iritis which neither clinically nor microscopically shows tubercle nodules nor allows us to demonstrate bacilli by the microscope or by inoculation, so that we should be inclined to attribute the iritis to a simple toxin action. But the possibility is by no means excluded that in such cases the tubercle bacilli themselves, although in scanty number and in a weakened state, may be the cause of the inflammation. Stock, by injecting tubercle bacilli into the blood of test animals, has produced small nodules in the iris and white patches in the chorioid, which disappeared again after a short time, while severe tuberculosis always develops when the tubercle bacilli are introduced into the eye. Stock explains the benignity of the affection in the first case by the assumption that the tubercle bacilli may have undergone weakening, due to their stay in the blood. The like explanation may hold good for the cases of benign tuberculosis of the uvea in man.

The cornea becomes diseased under the guise of keratitis parenchymatosa, keratitis eczematosa (scrofulosa), and sclerosing keratitis. The first disease is in most cases to be attributed to hereditary syphilis, the two latter affections to tuberculosis. Here, again, it is not determined whether the disease of the cornea depends upon the direct action of the microbes which may get into the cornea from its marginal vessels or upon the action of toxins. In animals, at all events, there can be produced by the inoculation of syphilitic material an inflammation which resembles parenchymatous keratitis and in which spirochætæ can be demonstrated in the cornea. A similar keratitis can be produced experimentally, by injecting the trypanosomes, which are akin to the spirochætæ. The retina, in which the metastases that occur in the course of acute infectious

diseases are most often localized, is rarely affected primarily, but very often secondarily in chronic infectious diseases.

By *endogenous infection of wounds* we understand the lodgement of microbes, which are present in the circulating blood, in those parts of the body that have been injured by traumatism. This process can be set up experimentally by injecting pyogenic germs into the blood channel and then injuring the eye. What can be done in experiments on animals must be regarded as probably also possible in man, and some cases which have been carefully examined belong with great probability in this category [of endogenous wound infection]. But we must be very careful how, without surer proof, we accept this process as a convenient explanation of all possible diseases whose causes we do not know.

V. Degenerative Processes

33. The line cannot be sharply drawn between simple degenerative processes and inflammatory processes that run a very chronic course. Simple degeneration has its purest embodiment in the changes which *old age* brings with it. Rather arbitrarily some of these changes are called physiological, others pathological, according as they are present in the majority or only in the minority of old people, or, even less rightly, according as they proceed without or with disturbance of the function of the eye. The pinguecula and gerontoxon are regarded as physiological changes, but senile cataract as pathological, although quite small opacities of the lens are but rarely missing in very old people.

A form of senile degeneration which is disseminated over the whole body is the hyaline degeneration of connective tissue and of the elastic fibers. In the eye this degeneration is the origin of the pinguecula and also of that thickening of the pupillary margin by which its motility is impaired. Hyaline exudations and thickenings are found in old persons in Descemet's membrane, and also in the lamina vitrea of the uvea, where they sometimes become visible with the ophthalmoscope, as so-called "druses," and may even give rise to disturbance of sight, if they actually occupy the region of the macula lutea. Another senile change in this region is the central senile retino-chorioiditis, which occupies a mid-place between degenerative and inflammatory processes, and which, since it leads to a central scotoma, is a not infrequent cause of the impaired sight of old people. The cystic degeneration of the retina occurring in old people affects only the region of the ora serrata and does not, therefore, impair the sight. Very often the eye becomes diseased indirectly, as a result of senile change in the vessels. Arteriosclerosis may lead, on the one hand, to rupture and, on the other hand, to narrowing or occlusion of the blood-vessels in the retina, the results of which are hæmorrhages, and degenerative processes in the retina. In sclerosis of the main trunk of the ophthalmic artery or the internal carotid, the optic nerve may be so compressed by the hard vessel wall that it atrophies.

Physical influences, without exceeding their customary limits, may lead to degenerative changes, if either they act in a cumulative way for a long period of time, or if a tissue that has too little resistance is opposed to them.

The former is the case with the pinguecula, which, although it is a senile phenomenon, yet develops only at that portion of the cornea which is exposed to the air; the latter is true of the zonular opacity of the cornea which develops in the region of the palpebral fissure in eyes which have suffered in nutrition, owing to some grave disease. In exceptional instances, zonular opacity of the cornea occurs as a simple senile change, in which case age constitutes the cause of the diminished nutrition of the cornea.

Poisons attack first of all the most sensitive portion of the eye, the retina, and in this again the ganglion cells which are particularly susceptible to poison. These cells are simply killed by the poison, and as a necessary consequence there is developed an ascending atrophy of the nerve-fiber layer of the retina and optic nerve. This process has been demonstrated experimentally for a series of poisons (quinine, filix mas, etc.) and by some is asserted to occur in the case of other poisons also (tobacco, alcohol, etc.).

For a series of degenerative processes we do not, up to the present time, know the cause. Only a few of these processes need be mentioned here: In the cornea, nodular and lattice-shaped opacity and keratoconus; in the lens, many forms of partial opacity; in the retina and optic nerve, retinitis pigmentosa and those changes which characterize Sachs's family amaurotic idiocy. These degenerations occur rather often with an hereditary or family distribution, so that probably they are founded on some *congenital defective structure* of the tissues, which later in the course of life becomes manifest as an actual disease.

VI. Congenital Defects, Heredity

34. We call many a thing a congenital defect of the eye without being able to offer sure proof that it actually was present at the very moment of birth. Gross changes in the eye of a new-born child, to be sure, excite the attention at once, but changes in the background of the eye are naturally not discovered until much later, on the occasion of some ophthalmoscopic examination, and even complete opacities of the lens are usually not noticed until some weeks or months after birth. So it happens that with regard to one of the most frequent forms of cataract, the cataracta perinuclearis, we do not yet know for certain whether it is congenital or is acquired in the first years of life. Of one anomaly which is called congenital, the medullated fibers of the retina, we know certainly now that it cannot be congenital, because at the time of birth the optic nerve itself possesses no medullary sheaths, these being formed first in extra-uterine life. The laity proceed in a still more inconsequent fashion, changes which develop soon after birth (e.g., those due to ophthalmia neonatorum) being often alleged to be congenital.

Congenital defects are of two kinds. One kind depends on an interference of development (malformations, in the narrower sense of the word),

the other on some disease of the fetus. A sharp line of distinction between the two cannot be drawn in all cases.

The *disturbances of development* are attributable either to a defective constitution of the germ-cell itself, having its origin in the father or the mother, or to the action of the neighboring parts upon the developing germ-cell. A good example of the first case is congenital ptosis, which sometimes is inherited through several generations. It is due to entire absence or defective development of the levator palpebræ. The same is the case with colobomata or complete absence of the iris, with congenital displacement of the lens, etc. Malformations based upon a defective germ-cell show typical forms which find their explanation in embryology. Injurious effects of the surrounding parts on the germ-cell are shown in the case of pressure of the amnion upon the fetus, or in the presence of amniotic bands, by which many anomalies in the lids and conjunctiva are accounted for.

Fetal diseases arise either from diseases of parents (especially syphilis) or from traumatic injuries which affect the fetus while still in utero. In new-born children, we may find either the results of diseases that have already run their course—such as opacities of the cornea or staphylomata following keratitis, adhesion of the pupil or atypical colobomata following iritis, etc.—or diseases (for instance, retinitis pigmentosa) which are still recent and which undergo further development in extra-uterine life. Congenital tumors also (for example, angiomata, nævi, gliomata) occur, which grow larger in after life. The congenital defects which are produced by external injuries or by diseases are not so typical as the malformations proper. In recent times, the production in an experimental way of disturbances of development by action from without forms a fertile field of study.

In this text-book, the malformations proper, being the really typical changes, are the conditions mainly pictured. Some of them, e.g., albinism, form but one of the symptoms of a general inhibition of development or they are at least accompanied by other congenital anomalies of the body; but generally the malformation of the eye is the only congenital defect present. Most malformations can be attributed to inhibition of development. The earlier this sets in the more marked are the consequences. Thus in inhibition occurring in the earliest stage of existence there is anophthalmus, in which case only a scarcely recognizable rudiment of the eyeball is present. It can be readily understood that the inhibition of development most frequently affects those parts in which the processes of development are most complicated. In the eye this is the region of the fetal ocular fissure. Incomplete or too late closure of this is one of the most frequent causes of congenital malformations. That is, if of extreme degree it is the cause of microphthalmus with appended cyst; when of slighter degree, is the cause of coloboma of the inner membranes of the eye, of ectopia of the lens, etc. In the human being after birth, then, we find either the actual condition set up by the inhibition of development or else its consequences. Thus, for example, the cysts, which sometimes hang upon the microphthalmic eyeball and are usually considerably larger than the latter, are in after-development due to ectasis of the insufficiently closed spot in the wall of the eye. In such a way as this an organ which was originally well developed may be secondarily altered.

Many malformations are caused by the persistence of the fetal structures, which otherwise undergo retrogression even before birth (e.g., a persistent hyaloid artery, or a persistent pupillary membrane). Hydrophthalmus is attributed to persistence of the fetal ligamentum pectinatum, so that the tissue in the angle of the anterior chamber is made more dense. This interferes with the filtration of the aqueous outward, so that there are developed increase of tension and, afterward, enlargement of the entire eye. Here, too, the conspicuous thing is not the malformation proper but its final result, the enlargement of the eyeball.

35. The most important cause of the malformations proper is a defective constitution of the germ-cell, which is acquired through *heredity*. In the wider sense of the term, heredity takes place in two ways, by transmission of a defective tendency which sooner or later develops into a malformation or a disease, or by the transmission of the disease itself. The most frequent example of the latter case (so-called *pseudo-heredity*) is the inheritance of syphilis, which takes place by the direct passage of spirochætæ from the mother to the fetus; furthermore, chemical substances (e.g., alcohol) circulating in the blood may cause the fetus to be diseased. But by *heredity in the true sense* we understand simply the transmission of a defective tendency to the descendant. [The defective tendency thus transmitted may be simply an exaggerated sensitiveness to special forms of injury (hereditary anaphylaxis).—D.]

Weissmann has formulated the following hypothesis with regard to heredity in general: The chromatin substance of the nucleus of the ovum which has just been fertilized, and which consists of the combined paternal and maternal chromatin, is not completely consumed in building up a new organism by a process of continuous subdivision, but a small portion of it is stored up and remains as the germ-plasm. This, in the developed organism, is deposited in the germ-cells themselves, and from it springs the next generation (continuity of germ-plasm). When, therefore, father and son have the same hereditary defects, this arises from the same defective germ-plasm. For the first in the line of ancestors who shows the defect at all, it must be assumed that for unknown reasons the germ-plasm from which he was derived was constituted differently from that of his predecessors (primary germ variation). But when this abnormal constitution of the germ-plasm has once developed, it can, in accordance with the continuity of germ-plasm, be transmitted to all succeeding generations, as the production by animal breeders of new varieties of animals from a single abnormally formed individual proves.

Heredity from one generation to the next is *direct heredity*. But one or more generations may be skipped, the defective tendency of the germ-cell remaining latent (*indirect* or *discontinuous heredity*, or atavism). This skipping of generations may actually go so far that the like structure is found only in our ancestors among the animals. For example, the presence of hyaline cartilage in the semilunar fold, which is extremely rare in Europeans and somewhat more frequent in the lower races of mankind, represents the cartilage in the membrana nictitans of the mammalia. Discontinuous heredity is found quite regularly in two eye diseases, color-blindness and hereditary neuritis with subsequent atrophy of the optic nerve (Leber's disease), and in this latter there is the additional peculiarity that the disease remains latent in the female members, so that they are exempt from the disease, while they are just the ones that transmit it to their successors. The diseased father has healthy sons and daughters. The children of the

sons are also healthy, but the children of the daughters are affected, so far as they are of the male sex. (Hæmophilia [and sometimes nystagmus] behaves in a similar way.)

A defective tendency in one progenitor, which has remained latent, may become manifest in the descendant, if, by chance, the other progenitor possesses the same latent tendency, so that the two tendencies are added together, and thus from two healthy parents diseased descendants are produced (potentized heredity). Of course, a faulty tendency having the same trend is most apt to be found in those persons who have the same descent, for which reason degenerated children are more frequently derived from the marriage of blood relations than from other marriages. As an example may be mentioned pigmentary degeneration of the retina and the degenerative hereditary deafness often associated with it, which conditions comparatively often occur in the offspring of consanguineous parents.

The way in which *acquired diseases*, in the narrower sense of the word, are inherited is still more obscure than is inheritance in general. The occurrence of such inheritance is established, but in any case it is much less common than in generally assumed.

The inherited anomalies or diseases may (1) already be present at birth, e.g., in the case of ptosis, coloboma or absence of the iris, ectopia of the lens, congenital cataract. (2) They are present at birth only as a tendency and do not develop until later on, instances of this being the form of optic neuritis above mentioned, retinitis pigmentosa, and many kinds of cataract. Finally (3) the faulty disposition dependent on heredity is not necessarily a disease at all, but may be only a variety of anatomical construction which predisposes to disease when other injurious influences are added—examples of this being the yielding character in the sclera that predisposes to myopia and the smallness of the eyeball that predisposes to glaucoma.

VII. Symptoms Associated With Eye Diseases

36. Altered Secretion.—The *secretion* in inflammation of the conjunctiva is either mucous or purulent, according to the intensity of the inflammation. By the drying of the secretion the lids stick together, especially over night. Only in conjunctivitis eczematosa the secretion of mucus is little marked in comparison with the profuse secretion of tears, because this disease in its pure form affects only the conjunctiva bulbi. The secretion of tears is also associated with diseases of the eyeball itself, including the inflammations of the cornea, iris, and ciliary body. However, both these diseases and conjunctivitis eczematosa, if they last a long time, readily lead to catarrhal inflammation of the conjunctiva and hence also to a secretion of mucus in addition to the secretion of tears.

Since many tears flow down into the nose when the secretion of tears is profuse, a patient often has to blow his nose, and hence thinks that he is suffering from a cold. At the beginning this is incorrect, but when profuse lachrymation has lasted for a long time the mucous membrane of the nose does finally get into a catarrhal state, owing to the irritation produced by the tears.

37. [**Photophobia**[1] is a term used to denote both the discomfort and the sense of dazzling and blinding produced by light (see § 20). The discomfort varies from a sense of burning, smarting, and sandiness of the lids or smarting of the eyeball to a severe cramping pain. It is associated with nictita-

[1] From φῶς, light, and φόβος, fear.

tion or when severe with blepharospasm.—D.] Photophobia originates both in the terminal fibers of the trigeminus and also in the retina. It expresses itself under the form of an unpleasant, even painful, sensation and blepharospasm. Most of the nerve endings of the cornea are in the epithelium, hence foreign bodies, small erosions, and superficial inflammations of the cornea are accompanied by marked photophobia, particularly if the nerve terminals are irritated by frequent winking. (Bandaging of the eye therefore, produces relief.) Deep ulcers of the cornea, in which the superficial nerves have been destroyed, or deep wounds of the cornea produce much less photophobia, so that we may almost say that the photophobia is often in inverse relation to the gravity of the corneal change. Inflammations of the iris, which is very rich in sensory nerves, are also associated with marked photophobia. In all these cases, the mere opening of the lids, even when the illumination is weak, suffices to evoke reflex blepharospasm.

The case is different with the photophobia that emanates from the retina. This photophobia occurs only when there is a great amount of light falling upon the eye (e.g., when one looks at the sun), but in that case affects healthy eyes also. In the case of the retina, it is not sensory fibers (since these are not present in it) but optical fibers that convey the reflex to the sphincter of the lids and of the pupil.

[The grittiness and burning of the lids seems to be due to the direct action of light, especially the ultra-violet rays, on the conjunctiva; and the sense of fatigue or strain produced by excessive or misdirected light (asthenopia photogenica) is probably attributable to tiring of the ciliary or extra-ocular muscles. Specially trying is light changing rapidly in intensity or the attempt to use the eyes in a very unequally illuminated room. Similar symptoms with a sense of drowsiness are caused by insufficient illumination.—D.]

A symptom that frequently accompanies photophobia, whether emanating from the trigeminus or the optic nerve, is reflex *sneezing*. Particularly in conjunctivitis eczematosa with marked photophobia sneezing sets in with great regularity whenever the attempt is made to open the eyes.

38. Glandular Swelling and Fever.—Normally, the *pre-auricular lymph gland* is not perceptible to the touch. When it can be felt through the skin this proves that there is some swelling of the gland. Such swelling is associated with eye diseases dependent upon infection of the eye. The conditions that most frequently give rise to it are, on the one hand, the phlegmonous inflammations (hordeolum, panophthalmitis, phlegmons of the orbit), and also inflammations that are particularly virulent (vaccine blepharitis, gonorrhœal and diphtheritic conjunctivitis). A marked glandular swelling is especially characteristic of Parinaud's conjunctivitis (see § 170). Among chronic infectious diseases, the tuberculous and syphilitic inflammations of the eye and its adnexa lead to swelling of the preauricular gland.

Fever is but rarely produced by an inflammation of the eyes—most frequently by the above named phlegmonous inflammations and by inflammations that are specially virulent.

39. Pain.—Pain is associated only with the inflammations of the anterior portions of the eye which possess sensory nerves; the chorioid, retina and optic nerve are incapable of exciting pain (the pain in acute retrobulbar neuritis comes not from the optic nerve itself but from its sheaths). The pain is in no definite relation to the severity of the inflammation, but in respect to its degree apparently keeps pace with the photophobia and the secretion of tears, as is obvious since all three are excited by irritation of the trigeminus.

The inflammations of the conjunctiva do not produce any violent pain, but only a sensation of a foreign body or of moderate burning and pressure. Sharp pain in the course of a conjunctivitis points to an involvement of the cornea. The inflammations of the cornea and sclera cause pain which is felt chiefly in the eye itself. On the other hand, in inflammation of the uvea and in increase of tension the pain usually radiates to the neighboring parts, and chiefly to the forehead, less often to the ears or the teeth of the upper jaw. This sort of pain is known as ciliary neuralgia, and in fact sometimes assumes a neuralgic character, since it occurs in attacks, and between the attacks there are intervals in which the pain is absent, even though the condition of the eye shows no recognizable variation. Indeed, not infrequently, just as in the case of true neuralgia, the attacks of pain recur every day at the same time (especially in the evening or at some hour of the night), and are then often favorably influenced by quinine. The radiation of the pain to the neighboring parts, especially to the forehead, often causes the patient himself to localize the starting point of the pain incorrectly, since he states that he feels no pain in the eye itself. If, then, there are no striking changes in the eye, the eye trouble itself may readily be overlooked and the morbid condition be regarded as a true neuralgia.

The kind of pain may give a valuable clew to the diagnosis. Beginners readily confound an iritis accompanied by marked injection, but without evident exudation, with a conjunctival catarrh. If the patient states that he has pain in the bones over the eye, we may be sure that it is not a simple conjunctival catarrh that is present, but an iritis.

The pain in severe inflammation of the eye often has the unpleasant character of becoming particularly violent just at night and depriving the patient of his night's rest. In irido-cyclitis, as in acute glaucoma, pains occur which are among the most severe that a man can possibly have, and which (especially in glaucoma) can actually excite symptoms of irritation of the brain, e.g., vomiting.

40. Headache.—Apart from pain which radiates from the eye to the head, actual headache may also emanate from the eyes even when the latter are not really diseased, this being the case, for instance, in the headache occurring in hypermetropes when the eyes are strained. If, therefore, the patient complains of frequent headaches, the physician treating him should always

think of the possibility of their originating from the eyes. The ophthalmologist sees many patients who have been sent to him by his colleagues, merely on account of headache and in order to determine whether the eye has anything to do with the headache. In a definitive way this can be done only by means of a precise examination of the eyes. But the experienced physician will frequently conclude what the cause of the headaches probably is from the description of the headaches and the accompanying circumstances which the patient gives. One should therefore, first of all, get from the patient a precise description of all his symptoms, as where the pain is situated, what are its intensity and quality (dull, compressing, boring, stabbing, hammering, etc.), whether it is constant or occurs in attacks, and in the latter case whether it is associated with any definite causes or any definite time of the day. A headache originating in the eye is localized in the anterior portion of the skull, that is in the vicinity of the orbit, in the forehead, and in the temples, while pain in the occiput does not usually occur from this cause. [To this rule, however, there are not a few exceptions.—D.]

Headache emanating from the eye can easily be recognized when we find the eye *inflamed*, weeping, photophobic, or sensitive to pressure. But often the objective changes in the eye are slight, in fact they may be actually absent at the moment of examination. This is particularly the case in prodromal glaucoma. Since the prodromal attacks usually occur in the evening, the physician often chances to see the patient in his office after the attack has elapsed and finds nothing abnormal in the eye. Then a precise history must afford assistance, a history which is particularly characteristic when the prodromal stage has lasted a long time. The headaches, the patient says, came on less frequently at first; afterward more and more often all the time; in summer he is almost free from them, in winter suffers much more often. The pain usually comes on in the afternoon or evening, and ceases with sleep; the patient is never waked by it from sleep. The headache is often produced by excitement, the visit of company, or by going to the theatre, etc., and the patient is hence regarded as nervous. In addition there is the associated disturbance of vision, consisting of some clouding of the sight and the appearance of colored rings about a light. But these disturbances are often slight and if they are limited to one eye they are readily overlooked by the patient. The examination of the eye then usually shows hypermetropia, a strikingly shallow anterior chamber, and perhaps arterial pulsation (in the retina) when even very slight pressure is made by the finger upon the eye. But as long as no glaucomatous excavation is present a certain diagnosis can be made only by having the patient visit us just when he is having an attack of headache. We shall then find in the eye the characteristic, even if slight, changes of a prodromal attack of glaucoma, and upon dropping in pilocarpine the symptoms pass off in about a quarter of an hour.

Headaches in which there is nothing to be seen in the eye externally originate in errors of *refraction* (hypermetropia and astigmatism), [in anomalies of accommodation—D.], and in disturbances of *muscular* equilibrium, especially latent divergence (or exophoria). [Headache may also be caused by esophoria and hyperphoria, particularly the latter.—D.] Such headaches are distinguished by the fact that they occur only after prolonged exertion of the eyes, and hence are never present upon waking in the morning. For the same reason they do not exist in small children, but occur first at an age when greater demands are made upon the eye. [To these statements some exceptions must be made. The pain due to muscular trouble and sometimes also to refractive errors may be present when the patient first wakes from sleep, particularly if the eyes have been subjected to strain the night before. Again, small children may suffer from headache due to eye strain.—D.] By hypermetropes the complaint is also made that the print swims after prolonged reading, by patients with muscular disorders the complaint that occasionally the lines become double and run into each other; and these latter patients also not infrequently allege that they experience a sensation of slight nausea when they strain their eyes.

Neurasthenics, too, when they strain their eyes have pain which they locate sometimes in the lids, sometimes in the eyes themselves, or behind the eyes, or in the head. The discomfort often sets in, within a few minutes, sometimes after reading a few lines, while in disorders of refraction or of the muscles, it does not set in until after rather prolonged exertion of the eyes.

Headaches of another kind which by the patient are often referred to the eyes are migraine, the headaches occurring in disease of the frontal sinus, and supra-orbital neuralgia.

In *migraine* the pain as a rule is unilateral. It is violent, boring, and situated over the eye, in the forehead and above it, but sometimes also in the eye itself. The attack is often introduced by a disturbance of vision (scintillating scotoma); during the attack the patient feels best if he can lie quietly in a darkened room; glaring light pains him; there is a sort of photophobia. All of these circumstances may cause the patient to believe that his eye is affected. In distinction from the headaches that really emanate from the eye, the migraine attack shows a quite typical course. Beginning with or without a scintillating scotoma, it rises pretty rapidly to a considerable pitch and usually lasts for from half a day to a whole day; then nausea ensues, and frequently vomiting, whereupon the patient feels better and the attack comes to an end. [Not all attacks of migraine have this typical course.—D.] Typical, also, is the way in which the attacks recur at certain intervals. They occur usually not oftener than once or twice a month (in women, who suffer from migraine more than men do, commonly in conjunction with menstruation), and with increasing age

grow less frequent and milder. In a person who suffers from migraine, a true migrainous attack can be set up by straining of the eyes, just as it can by many other injurious influences.

In empyema of the *frontal sinus*, there are also frontal headaches, but they are always on the same side, which is commonly not the case in migraine. When drainage into the nasal cavity is free, the pain is not particularly violent, but is more like a dull pressure. On the other hand, upon closure of the excretory duct and accumulation of the secretion, very violent, boring or hammering pains set in. The pain often comes in attacks, or sometimes shows a periodical increase, especially in the morning, when the secretion has accumulated during the night. Sometimes the pain increases when the head is bent forward. Local symptoms are tenderness of the upper margin of the orbit to pressure or percussion, tenderness to pressure of the supra-orbital nerve, and in acute cases a slight œdema of the skin over the frontal sinus. In addition there is a history of frequent colds in the head or blocking of the nasal cavity on the side of the pain. A certain diagnosis, however, can be made only on the basis of a rhinological examination.

Lastly, supra-orbital *neuralgia* has, in common with the two other kinds of headache, its localization in the forehead; and the tenderness of the supra-orbital nerve to touch occurs in the disorders above mentioned, too. Its character of recurring in attacks neuralgia shares with migraine, and the unilateral character of the pain it shares with disease of the frontal sinus. During the attack the eye of the affected side often is less wide open and weeps, and is even slightly injected, so that the suspicion of there being some eye trouble might be excited. It is to be stated as a characteristic sign of supra-orbital neuralgia that the pain is particularly violent and that the tenderness of the nerve, when touched during the attack, is so great that the patient draws his head back at once when this is done. The attacks set in suddenly after an interval of entire freedom from pain and sometimes are of but short duration, but recur either every day at certain hours or in irregular fashion several times a day.

Headaches of a persistent character, but with temporary exacerbations, accompany uræmia. *Nephritics*, therefore, suffer much from headaches, which are deep-seated and are boring in character. Sometimes there are vertigo and vomiting. In chronic nephritis frequent headaches are often the only thing of which the patient complains. In increase of *brain-pressure* (chronic hydrocephalus, meningitis serosa, brain tumor) the pain often has a similar character, but is still more often accompanied by vertigo and vomiting; often also there is tenderness of the skull to percussion at the point where the severest pain is situated. Hence in patients who complain of frequent headaches, the examination of the urine for albumin and of the eyes for changes at the optic-nerve entrance should never

be neglected. These latter changes may last a long time without there being any interference with sight, so that it would be a mistake to believe that an ophthalmoscopic examination is superfluous if the patient sees well.

In *syphilitics* deep boring headaches occur which undergo exacerbation chiefly at night. In *gout* and in rheumatism (rheumatic nodes on the branches of the trigeminus) there is present a quickly varying and boring or tearing pain in the orbit or about it or in the forehead. *Arteriosclerosis*, general *anæmia* and its opposite, *plethora*, give rise to headache. Young persons are often brought to the ophthalmologist on account of headaches which are thought to be connected with eye-strain caused by study. But in many of these cases no cause is to be found for the headache, which is then given the name of *habitual* headache and usually diminishes or disappears as the patient grows up. [It is to be noted that very slight errors of refraction or some obscure or apparently trivial muscular error may cause persistent headache. If these causes are overlooked, as sometimes happens, an erroneous diagnosis of habitual headache may be made.—D.]

41. Vertigo.—Vertigo is another very widespread symptom, on account of which the patients often visit the ophthalmologist with the idea that the vertigo starts from the eyes. It is advisable, therefore, to enumerate, just as was done with regard to headache, the different kinds and causes of vertigo, and to show how we can tell whether the vertigo is to be attributed to the eyes.

Vertigo, like headache, is a subjective symptom not to be recognized from any objective changes, so that the physician is dependent on the statements of the patient. These statements are much less precise than in the case of headache, partly because most men are not able to describe their symptoms intelligently anyhow, and partly because they are really dealing with sensations which are very difficult to define. We must, therefore, find out from the patient exactly what sensations he has during the attack of vertigo, by what conditions it is usually occasioned, and how long it lasts. By an examination of this sort it becomes apparent that disturbances of the most varied kind are called vertigo, there being included along with true vertigo various sensations of distress and disorders of vision. The disagreeable sensations caused by sudden noises and by heights (in looking down from a height) are quite generally called dizziness, but have nothing to do with real vertigo. Of visual disorders it is mainly the transient sort that are erroneously called vertigo by the patients, who complain that they are "dizzy in their eyes" when owing to hypermetropia the print swims after they have read a long time or when, owing to weakness of convergence, diplopia occurs, and the lines run into each other. Muscæ volitantes, a prodromal attack of glaucoma, scintillating scotoma, the momentary obscurations occurring in choked disk, are also often denoted as vertigo.

Vertigo in the narrower sense of the word either takes its origin from some visual derangement, or it develops independently of the latter,

although even then not infrequently it is accompanied by visual derangement. The sensations which the patient has in the different forms of vertigo may on precise analysis be differentiated into the following varieties:

1. Uncertainty in movement, as in grasping at objects and still more in walking, going up stairs, etc. This derangement arises from *false localization,* the object that is grasped at or the spot on which it is desired to plant the foot being seen in the wrong place. To a very slight extent, everyone has this sensation who for the first time in his life wears glasses, when he walks about. He sees the floor in front of him raised or, on the contrary, depressed, and is afraid of falling. This phenomenon very soon passes off as the patient becomes habituated to his glasses, and only in the case of strong glasses, such as, for example, patients wear who have been operated on for cataract, does it happen that some cannot get accustomed to them. For the glass, when the patient does not look through its center, acts like a prism, and the more so the stronger it is, and, owing to prismatic deflection, the object sighted appears in the wrong place. Hence, too, the trouble is the greatest when the patient looks quite obliquely through the glass, e.g., in looking down when he goes down stairs. We should, therefore, see to it that the glasses are well centered (see § 750) and should advise patients who have had a cataract operation not to look obliquely through their glasses, but rather to turn the head in the direction in which they wish to look.

To a much greater degree this same trouble is produced by false localization in paralyses of the eye muscles, so that even slight degrees of the latter may be very troublesome. An insignificant paresis of the trochlearis, which becomes manifest only in looking far down, may actually make the act of going down stairs dangerous, unless the patient himself remembers to close the paralyzed eye, whereupon the vertigo at once disappears. [So too a very marked and persistent asthenopia is caused sometimes by the very slightest amount of paresis in the inferior rectus, a paresis which ordinary tests fail to reveal.—D.]

In paralyses of the eye muscles there is an additional factor which is calculated to excite vertigo, namely,

2. The *false movement* of objects produced by false localization (see §654). The vertigo thus generated is often extremely great, but disappears at once on closure of the paralyzed eye. It is especially the peripheral paralyses that produce marked vertigo, while in paralyses of central origin the vertigo is often quite inconsiderable, and even the diplopia scarcely reaches the consciousness.

A false movement of objects always occurs when the images on the retina are displaced without the patient's being conscious of a movement of his eyes corresponding to the movement of the images. In paralyses of the eye muscles this happens because, without the patient's knowing it, voluntary movements of the eye are absent; but in other cases because,

conversely, movements of the eye occur which do not reach the patient's consciousness. These latter involuntary and unconscious movements occur in nystagmus and in vertigo produced by turning round. In the ordinary sort of nystagmus originating in childhood, no false movement occurs, because the patient is habituated to his abnormal eye movements and has learned to take them into account in judging of the displacement of the images on his retina. In nystagmus that develops later—miners' nystagmus, nystagmus originating in the ear, and nystagmus in disseminated sclerosis—false movement of objects and consequently vertigo do occur. Unconscious movements of the eyes also develop in the attempt to follow objects that are continually passing by, as, for instance, when one looks from a bridge at the water flowing beneath. When in that case the gaze falls upon objects which do not alter their position with regard to the eye (the flooring of the bridge, the bank), these appear to be moving in the opposite direction. The same thing occurs when the objects are still but the man is moving, e.g., when he is looking from a moving railway train or if he whirls round (vertigo in dancing). In the latter case there is a further factor which adds to the false movement and which is the irritation of the nerve terminals in the semicircular canals. This leads to another variety of vertigo. This variety is characterized by

3. The sensation of *disturbed equilibrium*, of falling or of being turned in a certain direction, so that by a movement of the body, which compensates for this imaginary disturbance, a fall in the opposite direction may easily result. To the highest degree such vertigo is present in Ménière's disease; to a less extent it is produced in patients with ear trouble by syringing the ear, the use of the air douche, etc. These cases are characterized by the fact that with them are associated disorders of hearing that correspond to the affection of the inner ear. But even in healthy ears the vestibular apparatus can be irritated and thus a vertigo be produced by whirling round, by swinging, by syringing the ear, or by galvanization. This irritation is commonly associated with nystagmic movements of the eyes which in themselves are calculated to set up vertigo. But the main cause of the latter lies in the irritation of the vestibular nerve, since the vertigo is evoked even when the eyes are closed.

4. Vertigo forms with headache and vomiting the triad which belongs with the symptoms that regularly accompany *intracranial increase of tension*. In addition, but not of quite such regular occurrence, is a fourth symptom, choked disk. Vertigo can be made available as a focal symptom only when it develops in a particularly intense way and is combined with ataxic symptoms, as is the case in involvement of certain portions of the brain, especially the cerebellum and the vermis.

5. A sensation in the head that cannot be precisely described, frequently combined with a transient feeling that everything is becoming

black before the eyes, is among the consequences of sudden changes of *circulation* in the brain, occurring thus in anæmic persons when they sit up suddenly, and occurring as a precursor of syncope and in arteriosclerosis of the arteries in the brain. It is known that disorders of the *stomach* are sometimes associated with vertigo, which can sometimes be excited voluntarily by pressure on the epigastrium. The way in which vertigo is excited by the stomach has not yet been made clear. Caution, moreover, must rule in making the diagnosis of stomach vertigo, since any vertigo, if intense, may lead to vomiting, which should not then without further consideration be regarded as a disease of the stomach.

42. [*Asthenopia*[2] is a sense of strain and weariness in the eyes and head, set up by the use of the eyes. The patient with asthenopia says that the eyes "give out." It may occur alone (asthenopia simplex), or be associated with pain in the eyes (asthenopia dolens), headache (asthenopia cephalalgica), or irritation, redness, and burning of the eyes or lids (asthenopia irritans). It may be accommodative, i.e., caused by refractive errors, especially hyperopia (see § 781) and astigmatism; muscular, i.e., caused by anomalies of the exterior muscles (see § 691); nervous, i.e., caused by hysteria or neurasthenia (see § 579); photogenic, i.e., caused by excessive or improper illumination (see § 37); or reflex, i.e., produced by morbid conditions of other organs, especially the nose and its accessory sinuses and the teeth. Asthenopia is the commonest of all eye symptoms and the one that is most readily relieved by proper treatment, which consists essentially in the removal of its cause.—D.]

43. [Other symptoms of which the eye patient sometimes complains are:

1. Double images (monocular or binocular *diplopia*; see §§ 646 and 650).
2. Multiple vision (*polyopia*, see §§ 471, 790).
3. *Metamorphopsia*, *micropsia*, and *macropsia* (see §§ 116 and 797).
4. Flashes of light before the eyes (*photopsiæ*). These are due to irritation of the retina from pressure, traction, etc. Not infrequently they indicate a beginning detachment of the retina.
5. *Chromatopsia*.[3] In this objects appear unduly colored; looking, for example, too red (erythropsia, see § 20), too blue (cyanopsia[4]) or too yellow (xanthopsia[5]).
6. Floating specks before the eyes (*muscæ volitantes*, see § 502).
7. *Reflex disturbances* (remote pains, nausea, tics of various kinds). These are not infrequently set up by refractive and muscular anomalies.
8. *Head-tilting* (sometimes amounting to actual torticollis). This occurs in astigmatism (being then usually slight) and in motor anomalies, particularly pareses of the ocular muscles (see § 655).—D.]

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[2] From ἀσθενής, weak, and ὤψ, sight.
[3] From χρῶμα, color, and ὤψ, sight.
[4] From κυάνεος, blue, and ὤψ, sight.
[5] From ξανθός, yellow, and ὤψ, sight.

GENERAL THERAPEUTICS OF THE EYE

Hygienic Regulations

44. General Hygienic Regulations.—These consist in the prescription of a light diet and in the case of inflammatory eye affections also in the withdrawal of alcohol. But from very old people who have been accustomed to take a little alcohol regularly, the latter should not be withdrawn, and as little ought we without further consideration to take alcohol away from actual drunkards. Such a rule applied after an operation, for example an operation for cataract, would be directly calculated to provoke an attack of delirium tremens. A good night's rest is important; if this is disturbed by severe pain, which very often sets in just at night, an anodyne must be administered in the evening. Of the ordinary remedies acetylicosalicylic acid (aspirin) has a particularly good effect in the pain caused by inflammation of the eye. With very violent pain, however, a morphine injection is the only thing that helps. As far as possible we should permit the patient to go out every day into the fresh air, but if he is very photophobic not till after sundown. He should stay in an atmosphere free from dust and smoke (avoiding restaurants filled with smoke, etc.). With any serious deep affection of the eye [inflammation, hæmorrhage, traumatism —including operations], abstention from work is required, including both heavy physical work and also any work that would strain the eyes. [It is important, therefore, in these cases to keep the patient in bed, so that as small demands as possible shall be made on the general and intraocular circulation, and eye and body shall have less chance to move about. Sometimes in inflammations and usually after operations, in order to rest and protect the eyes it is necessary to use protective glasses or a bandage (see §§ 45, 47). In general, such protection must be applied to both eyes, even though but one is affected.—D.]

The patients often think that if they bandage the affected eye they can with impunity read with the sound eye. But it is not possible to exert one eye alone. Accommodation, and convergence, and the contraction of the pupils that is associated with them always take place in both eyes at the same time and to an equal extent, and in experiments upon animals the illumination of one retina suffices to cause contraction of the cones and advancement of the pigment in the non-illuminated eye as well. If, therefore, a diseased eye is to be protected from the light, it is not sufficient to bandage it alone; both eyes must receive protection.

Absolute exclusion of light by bandaging both eyes or staying in a very dark room is scarcely ever required. We have abandoned treatment in the dark, which formerly was often employed and which depressed the patients physically and mentally. The like is true of the bilateral bandage which formerly was applied for a series of days, especially after cataract operations. A not infrequent consequence of complete exclusion of light in old persons who are predisposed to dementia is mental derangement. In a

person who has been operated on for caratact I bandage both eyes simply on the day of the operation, and from the first day on let the patient stay—and without harm to him, either—in quite a light room.

45. Protective Glasses.—As stated above, when protection from light is indicated, this should be applied to both eyes. For this purpose protective glasses are used. For the visible rays we use glasses of a neutral gray color. These are indicated for healthy eyes when an excess of light has to be kept out (in travelling over snow, in the tropics, and in certain industrial pursuits).[1] They are indicated for diseased eyes, to protect them against even ordinary daylight, when either there is great sensitiveness to light (photophobia) or disease of the retina or uvea. Since the organs are stimulated by light to the performance of their function, the shutting out of light is the first condition for putting them in a state of rest.

Since all kinds of glass keep out much of the ultra-violet light, the ordinary smoked glasses also give in most cases a sufficient protection against the ultra-violet rays. Hence such glasses are always indicated when with much ultra-violet light very dazzling visible rays are present (in dazzling from snow, in work with the arc light, etc.).[2] If we have to do with a light that is less intense, these glasses by cutting off the visible rays impair the ability to see minute objects. For such cases it is desirable to have a glass which keeps out the visible rays either little or not at all, but keeps out the ultra-violet rays as completely as possible. Glasses having this property are those which with a yellow or red color have also a certain chemical composition. Fieuzal devised the first glasses of this sort. Better still is the euphos glass recommended by Schanz and Stockhausen, which is not too deep a yellow. [Other glass mixtures of various names and compositions have been devised. An amber glass not too deeply tinged is fairly satisfactory. Having very little color tinge but quite effective in excluding the ultra-violet rays is the Crookes' glass.—D.]

46. Eye Guards.—For purposes of prophylaxis against eye injuries eye guards are used. These should protect the eye not only in front but also at the side. On the other hand, they should not exclude the air from the eye altogether, because by so doing an unpleasant feeling of heat is engendered, and the glasses themselves become covered on their inner surface with moisture. Hence the lateral protection must be effected by means of a fine wire mesh, which permits the circulation of air. [To further coolness and circulation of air, an open space can be left between the upper rim of the guard and the brow, since foreign bodies practically never reach the eye from above (Collis).] The transparent part lying right in front of the eye

[1] [Also in looking at the sun, especially through a telescope. For this purpose and also in certain forms of welding sometimes deeply colored glasses are used, e.g., a red glass backed with a green or a red glass backed with a blue.—D.]

[2] [When smoked or colored glasses are ground in the form of convex or concave lenses, the thickest part, whether edge or center, looks darker than the rest. This can be obviated by grinding a plano-convex or plano-concave lens of white glass and cementing to it a plane slab of the desired color.—D.]

is made of glass or, on account of their being less fragile, of celluloid, mica, or wire tissue. But glass is the only material transparent enough to afford really good sight. These spectacles are hot, and readily become covered with sweat, moisture, dust, or smoke, and unfortunately, therefore, are worn by the workmen with much reluctance. [The glass also soon becomes clouded from the constant impact of metallic and other particles. Hence the guard should be so arranged that the glass can readily be removed and replaced (Collis).—D.]

47. Eye Dressings.—A dressing for the eye may be either a protective or a pressure dressing. The object of the *protective dressing* is simply to keep the lids closed and still. By this means erosions, ulcers, or wounds on the surface of the eye are kept from being scoured by the movement of the lids, a process by which pain is set up and the regeneration of the epithelium and the precise apposition of the lips of the wound are interfered with. Hence in major operations we usually bandage both eyes on the first day in order to put the lids in a state of complete rest. When there is much secretion from the eye, a dressing is not indicated. [A bandage should also be used with circumspection for an eye that is predisposed to glaucoma, since the pupil dilates when the eye is covered and the mydriasis so caused may set up a glaucomatous attack.—D.] The protective dressing should be as light as possible. First, a bit of gauze is laid on the closed lids, then the hollow over the eye is packed flush with cotton, and the cotton held in place by strips of plaster or a bandage. In children, from whose heads an ordinary bandage always keeps slipping because of their continual restlessness, an immovable dressing made of a starch bandage is indicated. If the lids stick together beneath the dressing we smear the layer of gauze which lies upon the eye with an ointment containing boric acid or ichthyol. If it is a question of protecting the eye against a blow from the hand, which might force open a recently united wound (such a thing may readily occur in children at any time, and in adults during sleep), a protective wire cage or an aluminum shell (Snellen) is fastened over the eye. [See also Ring's mask, Fig. 410.—D.] A moist dressing in which the cotton is soaked in boric-acid solution and the like, and which has a piece of waterproof material placed over it, readily produces eczema of the skin of the lids.

The *pressure dressing* is made by applying more cotton to the eye and drawing the bandage tighter. When such a bandage is taken off after being on some time, the eye is somewhat softer than usual; in fact, some times the cornea is thrown into fine wrinkles, and the eye weeps and shows some ciliary injection. The pressure acting on the eye from without has increased the intra-ocular pressure by its own amount, so that now the aqueous flows out under a higher filtration pressure, and the eye thus becomes softer. The application of the pressure dressing is made with the expectation that pathological liquids, e.g., the subretinal liquid in detachment of the retina, will,

like the aqueous, more readily leave the eye. But very frequently the pressure dressing is badly borne; the eye becomes suddenly very soft, the anterior chamber unusually deep, and the ciliary injection may actually increase into an iritis. We must, therefore, proceed very circumspectly in applying a pressure dressing. The sensitiveness of many eyes with detachment of the retina towards a pressure dressing, arises from the fact that in such eyes the vitreous is not healthy and under the pressure dressing its volume rapidly decreases. Eyes which are sound in their deeper parts usually bear the pressure bandage well. We can, therefore, bring it into application without danger and even with the additional help of an elastic bandage, if we are dealing with ectasiæ of the external coats of the eye. An ectasia which has already developed probably cannot be relieved by this means, but probably also the formation of such an ectasia can be prevented. If the yielding spot (the thinned floor of an ulcer in the cornea, a prolapsing iris, a softening of the cornea due to pannus or severe parenchymatous keratitis, a softened portion of the sclera) is no longer able to resist the normal intra-ocular pressure, it is possible by diminishing this pressure with a pressure bandage for a protrusion at this spot to be averted until the place has become sufficiently firm. With this object, I apply an elastic bandage once or twice a day for as long a time as the patient can bear it without pain, which is usually for some hours.

In all cases where there is either the thinned floor of an ulcer, a recently united wound, or a still open perforation of the eyeball, sudden increase of pressure may lead to bursting open of the weak spot and to escape of the contents of the eye. Therefore, everything must be avoided which may produce a sudden *increase of pressure*, such as squeezing the lids, bodily labor like lifting a heavy weight, even stooping, straining at stool, chewing, and sneezing. Sneezing can be avoided if pressure is made at the right moment with the finger on the hard palate in the region of the foramen incisium. When perforation threatens, rest in bed is indicated.

Symptomatic Treatment

Apart from this so-called hygienic treatment of the eye, disease of the eye requires special treatment adapted to it, which may be both symptomatic and causal. For symptomatic treatment both physical and medicinal remedies are at our disposal.

48. The **physical** methods of treatment are:

(1) The *action of temperature*, usually under the form of cold or hot compresses. Because of the thinness of the lids, the effect of these compresses extends not only to the conjunctival sac but also to the eyeball itself, in fact even to the tissues of the orbit. The effect of iced compresses is more thorough than that of warm compresses. The former can depress the temperature by about 4° C., the latter can raise it 1° or more. The effect of *cold*

compresses is to contract the blood-vessels and thus combat the hyperæmia. They are chiefly used in recent injuries, after the action of corrosive agents and of severe cauterization of the conjunctiva, or after the expression of trachoma granulations, and finally in gonorrhœal conjunctivitis during the first stage. They diminish the feeling of heat and the pain. Furthermore, since cold retards physical and chemical processes, they are applied to diminish the swelling of the lens when it threatens to develop too violently, after wounding of the lens capsule. Much more in use are *hot* compresses. Heat dilates the blood-vessels, increases the hyperæmia, and accelerates metabolism, so that the natural protective substances of the blood are brought right to the spot. Phagocytosis by the leucocytes that have migrated from the blood-vessels is increased. Heat, therefore, reinforces the curative efforts made by nature in combating the lesion. Hence hot compresses find extensive application in inflammations of all sorts in the anterior portions of the eye, and often also act to reduce pain. But apart from theoretical considerations, it must be determined by observation how in individual cases the application of cold or heat is borne.

The compresses should be applied thus: The patient lies on his back, and the compress is kept on the closed lids by its own weight, and must not be pressed hard or tied tight upon the eyes. In order, also, that it may not press on the eye, it should not be too heavy. The best way of proceeding is to take a piece of linen or gauze, which is folded several times, so as just to cover the eye and its immediate vicinity; over this, a piece of water-proof material is applied in order to prevent too rapid alteration of the temperature. For cold compresses, the material is placed in cold water or upon a block of ice. Instead of plain water, lead water or a sublimate solution is employed in some cases. Hot compresses are made with hot water or hot chamomile tea. In any event the compresses must be changed often. To obviate this, some have advised an ice-bag instead of the cold compresses and thermophores or Leiter's tubes instead of the warm poultices; but all of these appliances have the disadvantage of being too heavy for the eyes. The electric thermophores alone, are light, but they are not to be had everywhere.

[Hot compresses are usually applied continuously for a certain period (say ten minutes), then intermitted for another period of twenty to fifty minutes. Cold compresses give the best results if applied without any intermission at all (De Schweinitz).—D.]

The direct application of moist heat to the cornea is effected by means of one of the ordinary kinds of atomizers (vaporization), and the application of dry heat by a current of heated air applied through a special apparatus designed for the purpose. Both forms of application are employed mainly in inflammations of the cornea and to clear up corneal opacities.

[The application of moist heat may be made by dropping very hot normal-salt solution directly upon the eyeball. An ordinary eye-dropper being filled with the

solution at a temperature ranging from 40° to 46° C., the lids are separated and the liquid is allowed to fall drop by drop, upon the sclera, or in certain cases upon the cornea. The temperature should be tested by dropping the liquid first upon the back of the hand, but it will be found that in many cases the eye will tolerate liquid too hot for either the skin, the lids, or the palpebral conjunctiva to bear. This form of application is of use in scleritis, keratitis, irido-cyclitis, and uveitis. Hot moist applications may be replaced by *diathermy*, or the passage through the parts of a high frequency electric current, which by the resistance of the tissues is converted into heat.—D.]

49. (2) [*Phototherapy*, or the application of light, especially light very rich in ultra-violet rays, has been used in corneal ulcers (Hertel, Schanz).—D.]

50. (3) *Radiotherapy* [better *actinotherapy*], by means of the Röntgen rays, radium [and mesothorium] is employed in the case of new growths upon the lids and cornea and in tuberculosis and trachoma of the conjunctiva. [Also in various forms of keratitis and in spring catarrh.

In applying either radium or X-rays it is important that the α-rays and β-rays should be filtered out, as they act harmfully on the superficial parts. The γ-rays do not do this, and at the same time they penetrate deeper, and act much more energetically on morbid growths than do the α-rays and β-rays (Leber).—D.]

51. (4) *Electricity* gives good service in paralysis or spasm of the orbicularis. It is also applied in paralyses of the eye muscles. We are not, however, able to make an eye muscle contract by means of the faradic current, unless the muscle is exposed, and it is, therefore, a question whether faradization is of any therapeutic value at all in paralyses of the eye muscles. The case is scarcely better for the galvanic current, which has but very slight current density when it flows through an eye muscle. So, too, the therapeutic results of galvanization in ordinary atrophies of the optic nerve are very doubtful. [According to Coleman, the sinusoidal (alternating) current produces often great and permanent improvement in these cases.—D.] Better authenticated is the action of electricity as an analgesic in eye inflammations. A saucer-shaped electrode is placed on the closed lids, the other electrode is put in the patient's hand, and a weak faradic current is passed (Reuss). Electricity achieves actual triumphs in all cases in which the action of suggestion is concerned, that is, in the numerous cases of neurasthenic and nervous asthenopia and amblyopia. The way in which we apply the electricity is of less consequence than our ability to impart to the patient the conviction that electricity is a very powerful curative agent which will surely relieve him. Electricity can also be applied for the purpose of making remedies (especially iodine and mercury) which are dissolved in water, penetrate into the tissues of the eye by means of the galvanic current (cataphoresis).

[Alypine, cocaine, and holocaine may be introduced into the tissues by cataphoresis, in order to anæsthetize them for operation (see § 803).

Differing from cataphoresis, in which the substance as such is conveyed into the tissues, is *electrolysis*, in which a chemical compound is dissociated, one component of

it (ion) collecting at the anode, the other at the cathode. Conveyance of ions by this means is called *iontophoresis* or *ionic medication*. A current of not over 2 milliamperes is used. Substances applied by iontophoresis are zinc sulphate in ⅓- to ½-per-cent solution (in ulcus serpens, dendritic keratitis, and herpes corneæ) and quinine sulphate in herpes zoster. In a more mechanical way electrolysis is used to remove cilia and shrivel up vascular tumors of the lids (see §§ 826, 828).—D.]

52. (5) *Hyperæmia* (*Bier*) can be induced in the eye in the form of suction hyperæmia. A glass of the shape of a cupping glass is placed with its mouth upon the lids, and the air in it is rarefied by suction made with a rubber bulb. There are produced œdema of the lids and conjunctiva, some exophthalmus, and evidently also an exit of fluid from the eye itself, for the latter directly after the procedure is softer. But this diminution in tension is not lasting, and the therapeutic value of the whole procedure in cases of increase of tension is still questionable. On the other hand, the method is employed with advantage in the case of purulent processes in the lids.

53. (6) *Blood-letting* is accomplished by means of leeches, or with Heurteloup's apparatus. Leeches to the number of from 6 to 10 are placed on the temple, not too close to the lids, as otherwise the latter become œdematous and swollen. Blood-letting is proved to have a particularly favorable action in severe irido-cyclitis.

In inflammation of the deep parts (chorioid, retina, optic nerve) use of it is but rarely made now. In such cases we withdraw the blood from a point over the mastoid process because an emissary vein of Santorini, which carries off the blood from the transverse sinus and thus indirectly from the cavernous sinus and the ophthalmic vein, empties here.

54. (7) *Massage* of the eye is used mainly in the case of scleritic nodules, and, combined with the application of yellow oxide of mercury ointment, for clearing up corneal opacities. [It is sometimes used in glaucoma.]

[Deep massage is used in retinal embolism to dislodge the embolus (see § 518). It is best done with the fingers, which are introduced as far back into the orbit as possible while the eye is turned far in different directions. Another, perhaps more effective, way is to make deep pressure with the fingers far back, or to squeeze the ball between two fingers placed on opposite sides of the globe, and then suddenly release the pressure—a manœuvre which causes the blood to flow suddenly into the vessels (see page 19).—D.]

55. (8) *Diaphoretic treatment* plays a great part in ophthalmology. It is employed for inflammations (scleritis, irido-cyclitis, chorioiditis, retinitis, retro-bulbar neuritis), and for the absorption of blood, exudate, opacities of the vitreous, and subretinal liquid. Diaphoresis by the subcutaneous injection of pilocarpine is so disagreeable for the patient that it has [largely] been given up. The most usual means of producing sweating are: (*a*) the taking of 1–2 grammes of aspirin with linden flower tea or hot lemonade, and then covering up warm in bed; (*b*) hot baths followed by wrapping in a dry woolen blanket; (*c*) a hot-air bath in bed (in this the air is heated by an alcohol lamp and is carried by a tube beneath the bed-clothes, which are raised

somewhat by means of a cradle); (*d*) vapor baths; (*e*) the electric-light bath. We select any one of these procedures, depending upon the patient's wishes and the local conditions, and we produce diaphoresis every day or at longer intervals according to the severity of the case and the patient's strength.

Sweating has a therapeutic value only when it is very profuse. If it remains only in the stage of congestion which precedes the sweating, or if the skin becomes but slightly moist, we get simply the disadvantages of the procedure without its advantages. If by sweating we wish to remove pathological products from the eye, the patient should take altogether very little liquid, since what we are trying to effect is the absorption of pathological effusions, by removing a large quantity of water. If, on the other hand, it is a question of removing toxic substances from the body, we make the patient drink water in abundance, since the excretion of sweat increases proportionally, and the injurious substances are the more abundantly eliminated.

Diaphoretic treatment is contraindicated in arteriosclerosis and heart disease, in patients who are in a very reduced state (especially tuberculous patients), and in pregnancy.

56. Medicinal remedies are applied in solution or as ointments or in the form of powder. *Watery solutions* are intended to be dropped into the conjunctival sac once a day or oftener. Solutions which irritate and hence are followed by rather profuse secretion, should not be instilled directly before the patient goes to sleep, because the resulting reaction interferes with his going to sleep, and the increased secretion is prevented by the closed lids from escaping freely. On the other hand, remedies are to be instilled in the evening when we wish them to act particularly by night (thus, for example in the case of the mydriatics in iritis, since the pupil is at its narrowest during sleep and hence synechiæ are most apt to develop at this time). If after being used a long time the solution shows cloudiness or flocculi it is to be renewed. In eyes with recent wounds due to injury or operation, only sterile solutions should be instilled. Slightly warm solutions pain less than cold solutions when they are instilled and are also rather more easily absorbed. It is useless to instil the solution in large quantities at any one time, for only a little remains in the conjunctival sac, and the rest at once flows off. One good drop is sufficient; if while we are dropping it in we make the patient look up and at the same time draw down the lower lid, the solution stays longer in the conjunctival sac and is absorbed in greater amount.[3] Drugs which alter the size of the pupil arrive by diffusion through the cornea into the aqueous, and thence act upon the iris (see page 8). A part of the solution is also absorbed by the conjunctiva and by the nasal mucous membrane, the solution reaching the latter through the tears. Ordinarily, this absorption is too slight to result in any general effect (symptoms of poisoning). This occurs only when the instillation is made very often, or when very concentrated solutions are used.

[3] [This is particularly the case if, after the patient looks up and while we are still holding the lower lid away from the eye, we direct him to look down so that the cornea is immersed in the fluid lying in the conjunctival sac.—D.]

By some the alkaloids are applied in an *oily solution.* Application in the form of an ointment which is introduced into the conjunctival sac is used particularly for atropine and cocaine and is advisable when there is a profuse flow of tears which would wash out a watery solution at once, while an ointment will stay longer in the conjunctival sac. The alkaloids are also obtainable in the form of small *tablets,* which are placed in the conjunctival sac and dissolve there.

As to *ointments,* those that are intended for the margin of the lid are rubbed in upon the closed lids in the evening before the patient goes to sleep, so as to act during the night. Crusts and scales are previously to be removed as far as possible by washing them with tepid water [and soap, § 602 —D.]. Since sometimes it is impossible absolutely to prevent some of the ointment from getting into the conjunctival sac and upon the cornea, very irritant ointments which are well borne by the skin cannot be applied to the eye. For lid ointments a fat should be chosen as a basis which is somewhat hydrous, for the water as it gradually evaporates cools the inflamed borders of the lid (cooling ointment). We accordingly select the unguentum emolliens or, as this soon becomes rancid, vaselin with which a small quantity of hydrous lanolin is mixed. Other ointments are designed to be introduced into the conjunctival sac, including besides the atropine and cocaine ointments already mentioned, mainly ointments made with iodoform (in the case of wounds and ulcers), with copper citrate (in trachoma), and with white or yellow oxide of mercury (in corneal processes).

Just as in the case of irritant collyria, the copper and mercury ointments, owing to their irritant action, should not be introduced into the eye at night before the patient goes to sleep. Ointments intended for the conjunctival sac should not contain any coarse particles which would act as a mechanical irritant. The medicinal substance must, therefore, be in the very finest powder, and must be rubbed up with the ointment bases with special care so as to form a uniform mixture. (This is particularly the case with the yellow oxide of mercury ointment. This should be kept in black jars, since it is gradually decomposed by the light.)

Powdered remedies must be in a very fine powder. Calomel, gallicin, and dionin are sprinkled with a brush upon the conjunctiva of the depressed lower lid, not upon the cornea; iodoform is sprinkled upon the raw spot in the eye.

57. The remedies most used in eye diseases are as follows (for physiological action, see §§ 322 et seq.):

(1) *Atropine.* This dilates the pupil and paralyzes the accommodation. It is therefore used, on the one hand, to relieve irritation and inflammation of the iris, and on the other hand, to produce paralysis of the accommodation, for the sake of getting a precise determination of the refraction. In practice, usually a 1-per-cent solution of atropine sulphate is used. When a particularly great effect is to be attained, we place a granule of the atropine salt in substance in the conjunctival sac, where it dissolves in the tears, and affords a concentrated solution. Still greater is the effect if we

combine atropine with cocaine so that contraction of the dilatator of the pupil is added to paralysis of the sphincter. We cocainize the eye just as for an operation, and after anæsthesia has set in, place a granule of atropine sulphate in the conjunctival sac. When it is employed in such an intensive way, symptoms of poisoning (see § 323) may set in, which do not usually occur with the instillation of a 1-per-cent solution. Specially susceptible to atropine are very small children who in about a quarter of an hour after the instillation of a 1-per-cent solution become red in the body, while, on the other hand, in them the dilating action on the pupil is slight. We should not be led astray by the latter fact nor instil atropine repeatedly, since even cases of death have originated in this way.

In applying atropine (especially atropine in substance) to the eye we preclude symptoms of poisoning if we prevent the tears from flowing down into the nose. With this object we draw the lower lid away from the eyeball for a little while, so that the tears pour out over the cheek, or we compress the lachrymal sac with the finger. In cases of severe poisoning a subcutaneous injection of morphine is indicated as an antidote.

Atropine is contraindicated in old persons with shallow anterior chamber, because in them glaucoma might be set up by the dilatation of the pupil, and obviously it is rigorously forbidden when glaucoma has already broken out. [The same is true of homatropine, euphthalmine, and cocaine.—D.] It must farther be discarded in persons who show an intolerance toward atropine (see § 323).

In such cases the atropine, according to circumstances, must either be simply given up or it must be replaced by another mydriatic. Of these the most suitable is *scopolamine hydrobromide,* and in ¼- to ½-per-cent solution, for it is more poisonous but also more active than atropine. [By some this in ⅕-per-cent solution is used regularly instead of either atropine or homatropine in determining the refraction (see § 792).—D.]

Since atropine in ordinary dosage causes disturbances of sight lasting a week or more, we should never instil it without substantial reasons, as unfortunately is so often done. For the same reason we do not use atropine when it is simply a question of dilating the pupil to facilitate examination with the ophthalmoscope, but employ homatropine [or euphthalmine].

Too much caution cannot be inculcated in regard to the senseless way in which *atropine* is often used, as it still is, unfortunately, by many general practitioners, who instil it in every kind of eye disease. In many cases—e.g., in conjunctival catarrh—atropine is not only superfluous, but also causes the patient annoyance through the disturbance of vision produced by its use; and in eyes which have a tendency to glaucoma, atropine may actually inflict great injury by determining an attack of acute glaucoma. Accordingly, atropine should be employed only upon quite specific indications, and should be applied no oftener than is requisite to obtain just the result desired.

58. (2) *Homatropine.* [This is used in 2-per-cent or 3-per-cent solution to paralyze the accommodation and thus facilitate the determi-

nation of the refraction (see § 792). The effect on the pupil and vision in this case lasts several days and is not promptly controllable by pilocarpine or even eserine. Hence when we do not wish to produce such a thoroughgoing action, but simply to dilate the pupil in order to make an ophthalmoscopic examination, we use a [½- or] 1-per-cent solution, the effect of which lasts less than twelve hours and can be abrogated by one or more instillations of 1-per-cent pilocarpine. It is particularly important not to use the stronger solution in case there is any suspicion of glaucoma.

A 2-per-cent solution of euphthalmine is used for the same purpose as a 1-per-cent solution of homatropine.

Homatropine in 2-per-cent solution may also be used as a tentative application instead of atropine in abrasions and slight infiltrations of the cornea (see §§ 208, 247) and as a means of diagnosis in suspected iritis (§ 336). In 1-per-cent solution it may be used to diagnosticate a suspected glaucoma (§ 442) and to ascertain the site of election for an optical iridectomy (§ 856).—D.]

59. (3) [*Eserine* (physostigmine) and *pilocarpine* narrow the pupil and, by contracting the ciliary muscle, stimulate the accommodation. They may, therefore, be used in paresis of the iris and ciliary muscle, but, as their action is transient, are effective only when the paresis is well on the wane (see § 797) or when the paresis itself is transient, e.g., when due to homatropine or euphthalmine. When used for this purpose they should be given in full doses, i.e., the eserine in ¼- to ½-per-cent (in certain cases even 1-per-cent) solutions, and the pilocarpine in 1- or 2-per-cent solutions.

Of much greater value are these agents in glaucoma, in which they are used in a strength and frequency varying according to the acuteness of the glaucomatous process and the results of the treatment (see § 458).—D.]

60. (4) *Cocaine.* The practically important properties of cocaine are its anæsthetic and mydriatic action. Owing to the latter, it is employed in order to produce a transient dilatation of the pupil for the purposes of examination, to reinforce the effect of atropine [and homatropine, and to promote the absorption of these agents and also of miotics, especially in inflamed eyes]. Still more important is the anæsthetic action of cocaine, the therapeutic application of which Koller was the first to show. A 2- to 5-per-cent solution of cocaine hydrochloride is used. If this is instilled several times at short intervals, we obtain complete insensibility of the conjunctiva and cornea, while the sensitiveness of the iris is but little reduced. The anæsthetic effect lasts about ten minutes. It is most frequently used as a preliminary step to any painful manipulation of the eye, such as the removal of foreign bodies and actual operations. [For the latter purpose it may be introduced not only by instillation, but also by subcutaneous or subconjunctival injection or by cataphoresis (see §§ 51 and 803).—D.] It also serves to diminish photophobia and blepharospasm, and hence facilitates examination. Finally, cocaine is frequently prescribed for

instillation in painful inflammations of the eye. But since it renders only the superficial portions of the eye insensible, it acts to relieve pain only in inflammations of the cornea, but not in irido-cyclitis, glaucoma, etc. But it is precisely in lesions of the cornea that cocaine can also do harm. We can convince ourselves of this fact when we instil cocaine a number of times in succession before an operation. The epithelium of the cornea then often becomes opaque and dull, or may even be exfoliated in places.

There are two reasons for this; first, the direct injurious action of the cocaine on the epithelial cells and, second, the drying of the surface of the cornea which occurs because, as a result of the insensitiveness of the cornea, the lids do not wink enough. We should not, therefore, apply cocaine for too long a time and, while we are applying it, should take care that the patient keeps his eyes shut after each instillation; then we shall rarely have to complain of "cocaine opacity." If we entrust a patient with a cocaine solution, to remedy some painful inflammation of the eye, he is easily induced to drop it in often because the analgesic action of the cocaine lasts only a short time. This frequency of instillation because of the injurious action of cocaine on the corneal epithelium may readily exert an unfavorable influence on the course of a keratitis. [Holocaine, especially in ointment or in oily solution, is a better drug than cocaine in such cases, and dionin or acoine is usually much better than either.—D.]

The mydriasis produced by cocainization remains for some hours and may annoy the patient by the disturbance of sight that is associated with it. Both the mydriasis and the disturbance of sight can be speedily relieved by the instillation of a drop of pilocarpine. [Part of the disturbance of sight is caused by dryness and consequent unevenness of the cornea. This can be relieved if the patient shuts his eyes at intervals so as to moisten the cornea.

For the diagnostic use of cocaine in paralysis of the sympathetic, see § 401.—D.]

61. After the introduction of cocaine other local anæsthetics were prepared by the aid of chemistry. In deciding as to their value, the difference in their toxic power has usually been the main point considered. So far as their application to the eye is concerned, however, this factor is of little account, because of the minute quantity in which these agents are applied. A more important thing is their influence on the blood-vessels of the eye and on the pupil. Only those anæsthetics will be spoken of here which have found application in ophthalmology.

Holocaine is more poisonous than cocaine. It has no influence on the blood-vessels and causes very little dilatation of the pupil. [It is used in 1-per-cent solution and is a very effective anæsthetic, used particularly when we wish to avoid acting on the pupil or tension (hence, especially in tonometry, in operating for glaucoma, and in the removal of foreign bodies).—D.]

All the other agents are less poisonous than cocaine. Of these—

Novocaine is the most similar in action to cocaine but is weaker. [Its effect is heightened by the addition of 1 part in 5 of a 2-per-cent solution of potassium sulphate. It is the best anæsthetic to add (in 4-per-cent solution) to subconjunctival injections, and with the addition of adrenaline it can be used to anæsthetize the eye for enucleation.—D.]

Tropococaine and *stovaine* dilate the pupil less than cocaine; with the former the conjunctival vessels are unchanged, the latter dilates them somewhat. [Stovaine is used in a 4-per-cent, tropococaine in a 1- to 3-per-cent solution.—D.]

Eucaine B and *alypine* leave the pupil quite unaltered but dilate the blood-vessels somewhat, and hence are advantageously combined with a little adrenaline. [Eucaine is used in a 1- to 3-per-cent, alypine in a 2- to 4-per-cent or, if injected subconjunctivally, in 1-per-cent solution. By some good authorities alypine is regarded as the best of local anæsthetics.—D.]

Acoine has no effect on the vessels and the pupil. It is used mainly as an addition to solutions which are to be injected under the conjunctiva; but unlike cocaine, it has the disadvantage of tending to produce inflammation in the tissues. [A 1-per-cent solution in castor oil may relieve for a number of hours the sharp pain produced by erosions or abrasions of the cornea (especially those remaining after the removal of a foreign body).—D.]

62. (5) *Adrenaline, suprarenin, paranephrin* are extracts of the suprarenal capsules, and *tonogen* [and *epinine*] are synthetic preparations which have the property of causing the smooth muscle fibers supplied by the sympathetic to contract, and hence act primarily as vaso-constrictors. If one of these drugs is dropped into a very much reddened eye, the latter after a few minutes (or even a few seconds) becomes pale as that of a corpse and remains so for an hour or longer. [The large conjunctival vessels and the deep (scleral) vessels are not usually affected. It may hence be used to differentiate between a deep and a superficial injection (in suspected iritis, etc.), and to show the character of tumefactions of the conjunctiva, since it reduces that part of the tumefaction due to vascular engorgement, but leaves unchanged that due to exudates.—D.] On account of its transient action the suprarenal extract is not adapted for the treatment of inflammation of the eye,[4] but only for the production of a temporary ischæmia. If we have an inflamed eye to operate on, it reinforces the action of the cocaine and diminishes the bleeding during the operation. It may also be instilled during an operation, in order to diminish a troublesome bleeding. For the same purpose we add it, in the proportion of two or three drops to the cubic centimeter, to solutions of cocaine or other anæsthetics intended for subcutaneous or subconjunctival injection.

63. (6) *Dionin* (ethyl-morphine hydrochloride), when introduced in powder or in a 5-per-cent solution into the conjunctival sac, produces, first, much burning and injection of the eye, after which, in a few minutes, a marked hot œdema of the conjunctiva develops which may go on to a tense chemosis [with puffing of the lids and with sneezing. Some recommend beginning with a 2½-per-cent solution and gradually increasing to

[4] [It is said, however, to increase the albumin content of the aqueous and hence favor development of antibodies. If so it would act favorably in inflammatory conditions.—D.]

10-per-cent.—D.]. The œdema disappears again in a few hours. The great hyperæmia of the conjunctiva causes the natural protective substances of the blood to pass into the tissue of the conjunctiva and into the interior of the eye. A further curative factor is the fact that the escape of blood-serum in quantities into the conjunctiva can act to modify the circulation in the eye. We, therefore, attribute to dionin an effect in promoting absorption, for which reason we employ it for clearing corneal opacities. In scleritis and irido-cyclitis, dionin ameliorates the violent pain and also little by little diminishes the injection of the eye. Furthermore, it acts very well to counteract marked photophobia in conjunctivitis eczematosa, parenchymatous keratitis, etc. [In detachment of the retina and other affections of the deeper parts it may be used by subconjunctival injection (see below).—D.] On repeated application less and less œdema is produced and the therapeutic effect of the drug ceases too. Then its further application would produce only a useless sensation of burning without any curative effect.

[Dionin is said occasionally to produce rise of tension without causing mydriasis. Hence in doubtful cases it may be well to combine with it a miotic (Elliot).—D.]

64. (7) *Subconjunctival injections.* The injection of active liquids beneath the conjunctiva has a twofold effect, namely, irritation of the tissue and alteration in the conditions of diffusion. The irritation of the tissue manifests itself at once by pain and by redness at the site of injection. A more remote consequence of the irritation is the passage of the natural protective substances from the blood into the tissues and spaces of the eye, and upon this probably depends part of the curative effect of the injection, especially in corneal ulcers. The alteration of the conditions of diffusion occurs because after the injection there is present beneath the conjunctiva a salt solution (e.g., a 5-per-cent solution of sodium chloride) having a concentration quite different from that which belongs to the liquid in the vitreous. The differing osmotic tension tends to become equalized through the animal membranes which lie between the two liquids (sclera, chorioid, retina). Thus there is given a stimulus to metabolism, which may promote the absorption of subretinal liquids [in detachment of the retina], of exudates in the inner membranes of the eye, and of hæmorrhages and vitreous opacities. The injections are made with a hypodermic syringe after the eye has been cocainized. In corneal diseases they are made beneath the anterior portion of the conjunctiva (but not too near the limbus). In diseases of the posterior segment of the eyeball they are made in Tenon's space [but not far back]. The redness and swelling that set in after the injection disappear after one or more days; and according to the way this occurs the injection can be made either every day or at longer intervals. After long-continued injections [especially if combined with

acoine] there is formed an adhesion between conjunctiva and sclera, which becomes more and more solid all the time. The substances which are oftenest used for injection are mercury oxycyanide (1 to 3 minims of a solution varying in strength from 1:1000 to 1:4000) and a 5- to 10-per-cent sterilized salt solution (one-half to one syringeful), [also dionin in from ½- to 5-per-cent solution.—D.]. In order to make the injection less painful cocaine [or better 0.2 gm. of a 4-per-cent solution of novocaine or of a 1-per-cent solution of alypine] is added. In corneal ulcers, in which it is a question of causing an irritant effect, I am in the habit of using the mercury solution, and the salt solution in diseases of the deep parts where the excitation of diffusion is the thing to be considered. In inflammatory irritation of the eye injections are not well borne.

65. (8) *Strychnine.* This exerts an excitant action upon the optic nerve, so that even in normal eyes it produces a slight although, to be sure, not permanent increase in the visual acuity and an enlargement of the field of vision. For therapeutic purposes, a ½-per-cent solution, of which a quantity equal to one-half or the whole of the contents of a Pravaz syringe —i.e., as much as 5 mg. of strychnine per dose—is injected once a day beneath the skin of the temple. [This dose may be increased to 10 or even 12 mg., especially in paresis of the iris or ciliary muscle, in which condition it may give good results.—D.] It acts best in disturbances of vision unattended by changes visible with the ophthalmoscope, especially in hysterical and neurasthenic forms, which, however, generally afford a good prognosis anyway. In serious lesions of the optic nerve, as in progressive atrophy, we often obtain with it an improvement in the sight and especially an enlargement of the field of vision; but these changes are not permanent.

Causal Treatment

66. Causal treatment is conducted in accordance with the etiology of the individual case. Hence it admits of a general consideration only so far as infectious diseases are concerned. The warfare upon the microbes and upon the effect they produce in the tissues can be accomplished by local and by general treatment. *Local* treatment is possible only when the bacteria are situated on or in the superficial portions of the eye, i.e., the conjunctiva and the cornea, and are hence accessible to direct action. Since many diseases of the conjunctiva and cornea are of bacterial origin, great hopes, which, however, have not been fulfilled, were put in the beginning on antiseptic measures. Only two remedies have proved to be actually serviceable, remedies which do not belong to the antiseptics proper at all, and both of which were already employed long before the discovery of bacteria, namely, zinc sulphate and silver nitrate. The former exerts a specifically injurious action on the bacillus of Morax-Axenfeld, the latter on the gonococcus. [To these may be added optochin, which is a specific

against the pneumococcus, and possibly against the gonococcus.—D.] On the other hand, the antiseptics proper in any such degree of concentration and with any such length of application as would be required for killing bacteria, produce extreme irritation of the eye, and injure its tissues so much that they do more harm than good. [This statement does not apply to the use of antiseptic solutions as a preliminary to operations (see § 802) nor to the occasional use of iodine as a direct antiseptic (not caustic) application to the eye.—D.] If the bacteria are situated not altogether on the surface, but in the tissue itself, they can be annihilated only if the tissue is destroyed at the same time. This is effected by means of caustics. These can be applied only when we have to do with a germ mass in the superficial membranes of the eye and above all in the cornea. Moreover, the germ mass must not have too great an extent, as, otherwise, too widespread destruction would have to be made. The caustic must be so fashioned that its action shall be precisely limited to the affected spot. This is true of the eye more than of other organs, because, for example, in the treatment of corneal ulcers every square millimetre of transparent cornea is of importance. Hence, liquid caustics or those which dissolve quickly are unsuitable, because they would flow over the neighboring parts and destroy them. [The action of nitric and pure carbolic acid can be sharply localized, and these two agents are often used to limit corneal suppurations.—D.] Answering best to the requirements propounded, is the actual cautery which is usually employed under the form of a fine galvano-cautery loop. It is used above all in ulcus serpens and other rapidly advancing corneal ulcers. Weaker caustics are a stick of copper sulphate, whittled to a sharp point, which is employed to advantage in keratitis dendritica, and tincture of iodine which is applied with a swab to the floor of the ulcer, after curetting away the necrotic portions.

Where caustics are not advisable, the attempt must be made to strengthen the natural resisting power of the tissues against bacteria. Serviceable in this way are the application of heat, subconjunctival injections, and paracentesis of the cornea, measures to which belongs the property of favoring the secretion of protective substances into the tissues of the eye, and most of all into the cornea.

[Under the head of local treatment may be included the removal of a focus of *infection remote from the eye*, but causing disease of the latter. Such foci are found in the teeth, tonsils, accessory nasal sinuses, and intestinal tract, and cause all sorts of eye disorders (prominently keratitis, iritis and uveitis, optic nerve disease, weakness of accommodation, and asthenopia). Not infrequently, after the infection from these foci has become generalized, even thoroughgoing removal of the latter does not suffice, but has to be combined with a vaccine treatment (see § 69).—D.]

67. *General treatment* has to reinforce local treatment and is the only form of therapy possible when the bacterial focus is not directly accessible. Besides the remedies, like diaphoresis, mercury, and iodine, which are applied generally in infectious diseases of various kinds, there are also those which possess a quite specific action for certain morbific agents. These methods of treatment are just now beginning to develop on the basis of experimental research, and it should not, therefore, excite surprise that their results as yet are somewhat uncertain. They are based on the fact that by passing through one infection one acquires an immunity, varying in degree, against a new infection by the same morbific agent, because the body forms specific protective substances against the latter. The action of the protective substances may consist either in their killing the bacteria themselves or in their neutralizing the poisons produced by the bacteria. Such an immunity may also be produced artificially, and that either in a direct or indirect way. The former is *active immunization* [or vaccine therapy], which consists in injecting into the patient dead or weakened cultures of bacteria of the same kind as those by which he has been made ill, so that he himself may form in his own body substances protecting against these bacteria. The type of such an active immunization is the injection of tuberculin. The indirect method is called *passive immunization* [or serum therapy], which consists in subjecting an animal to active immunization, so that its serum forms protective substances which then are employed for rendering the patient immune. The protective substances contained in this serum may be either antitoxic or bactericidal. The oldest example of the first case is the antidiphtherial serum, while the pneumococcus serum, for example, contains bactericidal substances.

The protective substances are of avail only against that kind of bacterium with which the immunization was produced. Since some bacteria act differently in different strains, some, in order to secure a specific immunization, have gone so far as to try to immunize an individual case of disease by means of cultures which they have obtained from the morbific germs derived from the case itself. But the preparation of cultures in sufficient quantities demands so much time that in acute cases one would usually be too late to effect a cure. [In acute cases, therefore, while the *autogenous* vaccines, i.e., those derived from the patient himself, are preparing, *heterogenous* vaccines of similar character derived from other sources are used. Or an autogenous serum may be used derived from a blister raised on the patient's skin (Römer).—D.] Another way of getting a specific serum with certainty consists in procuring it by immunizing animals with different strains of the same species of bacterium, so that one may expect that the special kind of strain by which the patient is rendered ill should be represented in it. Such a serum has been called polyvalent [better multivalent.—D.], but the same expression has been used in an

almost opposite sense, in that there has been attributed to individual sera (e.g., the diphtheria serum) the ability to act against all possible infections.

[When a specific germ cannot be isolated from the lesion in the eye itself or from the blood, *complement fixation tests* are used in order to determine the specific organism producing the disease and the one that should be used in immunizations. The oldest of these tests (that for the spirochæta pallida) is the Wassermann test, but in recent years complement fixation tests for the gonococcus, pneumococcus, various strains of staphylococcus and streptococcus, the influenza bacillus, the colon bacillus, and the micrococcus catarrhalis have been applied (Hastings, Reber).—D.]

The attempts at immunization which so far have proved most certainly effective in ophthalmology are that with the diphtheria serum by passive immunization and that with tuberculin by active immunization.

The *diphtheria serum* has only a limited application, namely, in diphtheria of the conjunctiva, in which it is successfully employed both by subcutaneous injection and by instillation into the conjunctival sac.

68. *Tuberculin* has enjoyed an extensive application in ophthalmology, particularly through the efforts of Hippel Senior. It is used for diagnostic and for therapeutic purposes. Its *diagnostic* application is designed to determine whether the patient is tuberculous at all and in particular whether his eye disease depends on his tuberculosis. The former is the case if the patient reacts in a positive way, i.e., with a rise of temperature, to the tuberculin injection; the second, if a local reaction occurs in the diseased eye.

The diagnostic application of tuberculin is performed by injecting the old tuberculin of Koch. Since in positive cases this excites fever, it is to be omitted as superfluous, if the presence of tuberculosis has been already made certain by the physical examination of the patient or by the examination of the sputum. In other cases, we inject 0.5 mg. beneath the skin of the upper arm. Before the injection, the patient should have his temperature taken every two hours for two days, so that we may know his ordinary temperature curve, and then measurement of the temperature should be continued for two days more. [It is well also to make sure that there is no cavity in the lung nor active tuberculous process in the joints or vertebræ, since in such cases the use of tuberculin is dangerous (Wilder).—D.] The rise of temperature usually occurs before the lapse of 24 hours, but sometimes not till later. If no fever occurs within 24 to 48 hours after the injection [3 mg. should be injected, and if in 24 hours more there is no reaction 5 mg. may be given. If then there is no reaction, active tuberculosis may be assumed to be absent. It is important to place the injections thus close together, since, if they are put too far apart, anaphylaxis may develop.—D.]. Besides the rise of temperature, there frequently develops, if tuberculosis is present, a local reaction at the site of injection, namely, an infiltration of the subcutaneous cellular tissue at the place

where the tuberculin passed from the point of the needle into the tissue. The skin at this place is a little reddened and beneath the skin is felt a hard and rather tender spot. [A focal reaction in the eye itself may also occur. This consists] either in increased injection or in fresh exudation; it occurs but seldom, and is unwelcome, too, since it means an aggravation of the eye disease [which in some situations, especially the cornea, may be quite dangerous]. It is true, though, that if a local reaction is absent, when a general reaction is present, the diagnosis that there is a local tuberculous trouble is only a diagnosis of probability.

Other diagnostic methods of employing tuberculin are the cutaneous methods in which the tuberculin is introduced, not beneath the skin, but into it and excites in the skin a local reaction without causing fever. In Pirquet's method the epidermis is scarified and tuberculin is placed on it; in Moro's method a tuberculin ointment is rubbed upon the intact skin. Finally Calmette's method consists in dropping tuberculin into the conjunctival sac; but since occasionally some not inconsiderable inflammations of the eyes have developed in consequence, this method has been properly given up.

The *therapeutic application* of tuberculin is performed by means of subcutaneous injections, for which we take either new tuberculin (TR), or the preparation known as bacillus emulsion. In this method, in contrast to what obtains in the diagnostic application, care must be taken that the patient does not get fever. We therefore begin with a very small dose and increase it, but very gradually; and if the patient, in spite of this, does get fever, we return to the former smaller dose. [We should do the same or lengthen the interval between injections if there is a focal reaction in the eye. To determine whether this is present or not, we should examine the eye exteriorly and with the ophthalmoscope after each injection (Jackson). According to von Hippel, the initial dose should be 1:500 mg., but it is now thought safer to begin with one of 1:10,000, 1:5000 or at most 1:2000 mg. and to increase the dose at each injection, the injections being made at intervals of a week or longer. At the Knapp Memorial Hospital the custom is to begin with 1:5000 mg. and give the injections 3 times a week; adding to each injection 1:5000 mg., until a dose of 1:500 mg. is reached; then adding 1:500 mg. till a dose of 1:50 mg. is reached; then adding 1:50 mg. till a dose of $\frac{1}{5}$ mg. is reached. Then at weekly intervals successive doses of $\frac{2}{5}$, $\frac{3}{5}$, etc., mg. are given until 1 mg. is reached. This is repeated every 3 or 4 weeks. In any event, whatever routine is adopted, the dose and interval must be regulated by the effect.—D.] In a few cases we have to continue these injections for many weeks in order to attain success.

69. [*Other infections* in which vaccine treatment has been applied with some success are those produced by the gonococcus, various strains

of the staphylococci and streptococci, the influenza bacillus, and the pneumococcus. Such treatment has been particularly applied in uveitis of gonorrhœal origin or derived from dental infection and the like; also to arrest a beginning suppuration after operations. As above stated, complement fixation tests may be employed to determine the specific organism involved and to indicate the culture that should be used in forming the vaccine. As in using tuberculin, the amount of vaccine used and the interval between injections are regulated by the effect, i.e., the presence or absence of a local reaction at the site of injection or a focal reaction in the eye.—D.]

70. Against the widespread parasitic disease, syphilis, no immunizing procedure has so far been found. Fortunately, in salvarsan, mercury, and iodine we possess specifically acting agents with which to combat this disease.

[Treatment in syphilis is largely regulated by the complement fixation or *Wassermann test.* A positive Wassermann may be found as early as the first week, but usually not before the fourth week of the disease. In secondary syphilis and in tertiary syphilis with lesions it is almost invariably present, but may be banished for the time being by treatment. Per contra, when absent it may sometimes be elicited if we give an injection of mercury or salvarsan (*provocative Wassermann*). It must be noted that it is found in some conditions besides syphilis (yaws, trypanosomiasis, some forms of malaria, scarlet fever). But this usually causes no confusion.

In syphilis of the nervous system, including general paresis and tabes, the blood examination may prove negative, and yet the spinal fluid, obtained by lumbar puncture, will almost always give a positive Wassermann reaction. The spinal fluid at the same time usually shows lymphocytosis and a positive globulin reaction (Noguchi's *butyric-acid reaction*).

The Wassermann reaction can be replaced by Noguchi's *luetin test* (cutaneous reaction produced by the intradermic injection of dead spirochætæ pallidæ).

Salvarsan is used in doses of 0.25 to 0.30 gm., increased if need be, given by intravenous injection every one or two weeks. In syphilis of the nervous system if salvarsan given intravenously is not effective, salvarsanized human serum may be administered by intraspinous injection.—D.]

71. [Besides treatment addressed to the infections there is that form of general treatment which is concerned with the metabolic processes and has to do with the regulation of the digestive functions and of the internal secretions; furthermore treatment designed to improve the conditions of circulation of the blood and lymph or to improve the sensory, motor, or trophic functions of the nerves. Many diseases of the eye require for their successful treatment careful consideration of one or more of these factors.

We may simply mention here the use of thyroid extract in parenchymatous keratitis and other forms of corneal disease; the use of small doses of calomel and careful regulation of the diet in phlyctenular keratitis and in uveitis; the prolonged administration of iodides to absorb vitreous and other opacities; and the exhibition of arsenic in disorders traceable to perverted nerve action (herpes zoster, corneal dystrophies).—D.]

PART II

EXAMINATION OF THE EYES

EXAMINATION OF THE EYES

CHAPTER I

OBJECTIVE EXAMINATION OF THE EYES

72. The examination of a patient's eyes is begun after ascertaining the history of the case.[1] In making this examination too much stress cannot be laid upon the necessity of proceeding systematically, since otherwise matters of importance may very readily be overlooked. We first, therefore, take a survey of the patient's appearance in general, as well as of the expression of his eyes and countenance; then we examine the eyes themselves, and in so doing proceed successively from the superficial parts—lids, conjunctiva, and cornea—to the deeper portions.

73. In respect to the *lids*, there are to be considered their position and mobility, the width of the fissure between them, and the way they shut together. The character of the skin lining the lids is examined, and especially at their margins, where pathological changes are most often found. Apart from the symptoms of inflammation, which is very apt to be localized at the borders of the lids, the things that we must look for are whether the palpebral edges have not possibly lost their sharply defined form and outline, whether the cilia are correctly placed, and also whether the puncta dip properly into the lacus lacrimalis. At the same time, we must not neglect to investigate the region of the tear sac. Should simple inspection disclose no alteration, it is yet often possible, by pressure with the fingers in this region, to make the contents of the diseased sac exude through the puncta. Furthermore we ascertain whether the small lymph gland in front of the ear is perceptible to the touch and is sensitive to pressure.

74. The examination of the *eyeball* itself is often rendered very difficult by strong spasm of the lids—blepharospasm. This is especially the case in children who, the more the physician attempts to draw the lids apart, squeeze them the more tightly together. In these cases the forcible separation of the lids calls for the greatest caution, since, if this is not observed, and a deeply penetrating ulcer is present, it is easy to cause a sudden perforation of the cornea, nay, even the extrusion of the lens from the eye. By dropping a solution of cocaine between the slightly parted lids we try to diminish their sensitiveness; and for separating the lids we can, with advantage, use Desmarre's elevator (see Fig. 2), with which we shall less readily inflict an injury than we should do if, by using the fingers, we exerted too

[1] [In taking the history inquiry should be made for the various symptoms, such as increased secretion, redness of the eye, pain, asthenopia, headache, vertigo, blurred vision, diplopia, metamorphopsia, head-tilting, etc., likely to occur in eye diseases (see pages 42–51), and also for the presence of any of the possible causal factors given on pages 20–41.—D.]

great a pressure upon the eyeball. Finally, in many cases it is only by means of general narcosis that we can obtain a sufficiently satisfactory view of the eyes. In spite of all these difficulties we should not be deterred from insisting upon an exact examination of the eyes at the patient's first visit, in order to establish the diagnosis and prognosis and to determine the treatment. [The best way to examine a small child is to put it on its back in the lap of the mother or an assistant, who is seated opposite the examiner and who controls the child's arms, legs, and body. The examiner, who is also seated, secures the child's head by gripping it tightly between his knees. The child is thus rendered perfectly immobile, and the examiner has both hands free to evert the lids, concentrate light on the eye, make applications, or do anything else that is necessary.—D.]

[Fig. 2.—D.]

In regard to the eyeball itself, we must first satisfy ourselves whether its situation in the orbit, its position with respect to the other eye, its size, and its mobility are normal or not.

[For the position of the eye in the orbit see § 710, and for the methods of determining the position of each eye with regard to the other and the absolute and relative motility of the two eyes, see §§ 673–675 and §§ 676–677.

It is often important to determine the distance between the two eyes. This is usually ascertained by taking the *interpupillary* (or *pupillary*) *distance*. This distance naturally varies according as the eyes are converged or not. To determine it when the eyes are not converging at all, we proceed as follows: Observer and patient face each other. The observer's right eye and the patient's left eye are closed or covered. The two uncovered eyes (patient's right, observer's left) look straight at each other. The observer holds a millimetre scale between himself and the patient, with its zero mark in line with the center of the patient's right pupil. Then without moving the scale he directs the patient to open his left eye and close his right, while he himself opens his own right eye and closes his left. The patient being now directed to look straight into the observer's right eye, the observer notes what part of the scale is opposite the center of the patient's left pupil. The reading will give the interpupillary distance in mm. when the visual axes are parallel. It therefore also gives the distance between the centers of rotation of the two eyes. This reading will be unaffected by the presence of a squint provided there is no absolute inability on the part of either eye to perform fixation. The interpupillary distance ranges between 53.5 and 71 (usually between 59 and 67) mm.

The *dimensions* of the adult eye in mm. are as follows (Merkel and Kallius):

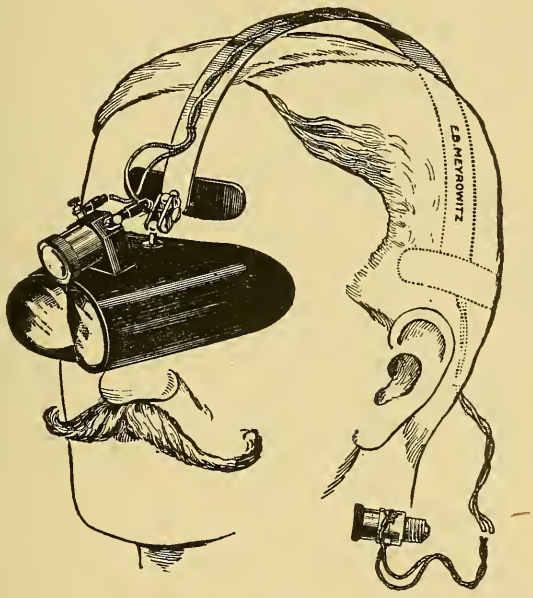
Antero-posterior diameter	24.3
Transverse diameter	23.6
Vertical diameter	23.3
Thickness of cornea at apex	0.9
Thickness of cornea at base	1.1
Diameter of cornea (diameter of anterior chamber)	11.9
Depth of anterior chamber	2.3
Thickness of lens (eye not accommodating)	3.7
Thickness of lens (eye accommodating)	4.3

The antero-posterior diameter of the eye is usually greater in myopes and less in hyperopes than that above given (see §§ 771 and 782).

In the newborn infant the antero-posterior diameter of the eye varies from 15.78 to 17.50 and the vertical diameter from 14.50 to 17.00 mm. The eye grows rapidly during the first few years of life, the vertical diameter growing faster. so that the eye becomes now more nearly spherical (Weiss).

The center of rotation of the eye is 13.5 mm. back of the apex of the cornea (Helmholtz).—D.]

75. The *conjunctiva of the lids* can be brought into view by everting the latter. With the lower lid, it is sufficient for this purpose simply to draw it down, while the patient is told at the same time to look up. [If in pulling



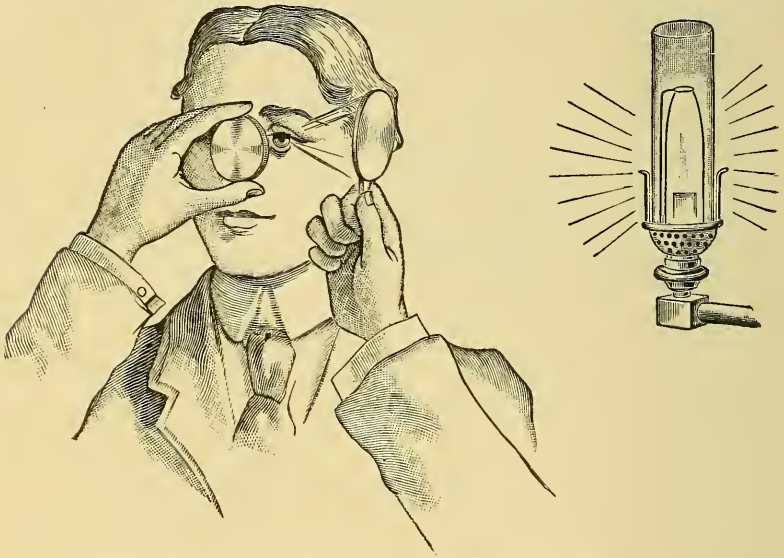
[Fig. 3.—Berger's Binocular Loupe with Electric Attachment.—D.]

the lid down we make slight pressure against it below with a somewhat rolling movement, so as to revolve the lid forward, not only will the lid itself be everted, but the retrotarsal fold will spring up and come into view.—D.]

With the upper lid, eversion requires a certain degree of skill, which must be obtained by practice. It is the more important to acquire this facility, since it is just the conjunctiva of the upper lid that generally affords the best evidence for the diagnosis of conjunctival diseases: the thickening of the conjunctiva, the uneven surface, the formation of cicatrices, which are characteristic of trachoma, are here most easily to be perceived. Further, the eversion of the upper lid is very frequently necessary for the removal of foreign bodies. [To evert the upper lid we direct the patient to look down, grasp the lashes between the thumb and finger, and draw the lid down and

somewhat away from the eyebrow. When the lid is well on the stretch, we place a pencil or similar object lengthwise in the groove beneath the brow and just above the tarsus. With the fingers that grasp the lashes we turn the lid sharply outward and upward, keeping it on the stretch all the time and revolving it on the pencil as a gate on its hinge.—D.]

76. In the examination of the *cornea*, besides a careful inspection with a good magnifier, such as Hartnack's spherical loupe or Zeiss's binocular microscope,[2] there are two main artifices in use,—examination of the corneal reflex and lateral illumination. To examine the *corneal reflex* signifies nothing more than to direct the eye in such a manner that the reflection of a window placed opposite it is visible upon the cornea (Fig. 78). By causing the eye to follow the movements of a finger held before it, the reflection is



[Fig. 4.—Oblique Illumination. (After Posey and Wright.)—D.]

brought successively upon different portions of the corneal surface, of whose curvature and smoothness we in this manner obtain an impression. [See also § 190.]

Lateral [or *oblique*] *illumination* consists in the concentration of light upon a certain portion of the cornea by means of a convex lens. This important method, although already employed by Himly, Mackenzie, and Sanson, was yet very little known formerly, and first obtained general currency through the efforts of Helmholtz. A light (candle or lamp) is placed beside and somewhat in front of the patient. [See Fig. 4.] Then, by the aid

[2] [Other corneal microscopes are those of Czapski and Howe. The binocular loupes of Jackson and Zeiss, which are attached to the observer's forehead by a head band, have the great advantage of leaving both hands of the observer free, and hence are particularly useful for finding and removing foreign bodies. An ordinary head-mirror often serves the same purpose very acceptably.—D.]

of a strong convex lens (of 15–20 D), the rays are concentrated to a cone of light, whose apex is made to fall upon the portion of the cornea to be examined. This method is designated as focal illumination, because the point to be illuminated is brought into the focus of the lens. This point stands out with special distinctness because, on the one hand, a great quantity of light is concentrated upon it, and because, on the other hand, the parts immediately surrounding it remain almost completely in darkness. On this latter ground, lateral illumination gives the most advantageous results if in its application the room is darkened. By lateral illumination we can recognize opacities in the cornea which are perceptible in no other way. The iris, too, and the lens as well, can be examined in this way by varying the depth to which the light is projected. By so doing we have not merely the advantage of getting very sharp images, but also, from the fact that we can at will vary the depth to which the apex of the conical sheaf of rays is projected, we get information as to the depth at which the changes that we observe are situated. [In applying oblique illumination it is well to use two lenses, one of from two to three inches focal distance, held in one hand, to concentrate the light, the other, held in the other hand, to magnify the image. By carefully focusing the two lenses and by making slight shifting movements with the condensing lens, we get the most accurate idea of the outlines of opacities, the markings of the iris, and other important details.—D.] A handy method of lateral illumination is furnished by the lamp of Priestley Smith. This carries in its center a small candle as a source of light; a strong convex lens let into the side of the lamp serves for the production of the cone of light. [For examining the surface of the cornea it is often advantageous to use oblique illumination by daylight. The patient with his head thrown well back reclines in a chair facing a window, and the observer stands behind him, focusing the daylight on the cornea with a lens and examining the cornea from above with a strong magnifying glass. If the lenses are focused accurately on the surface of the cornea so as to bring to view the flecks of mucus on it, and the patient then moves his eye about, the finest abrasions and also the slightest deformations of the corneal reflex (see Fig. 78) can be made out with ease.—D.]

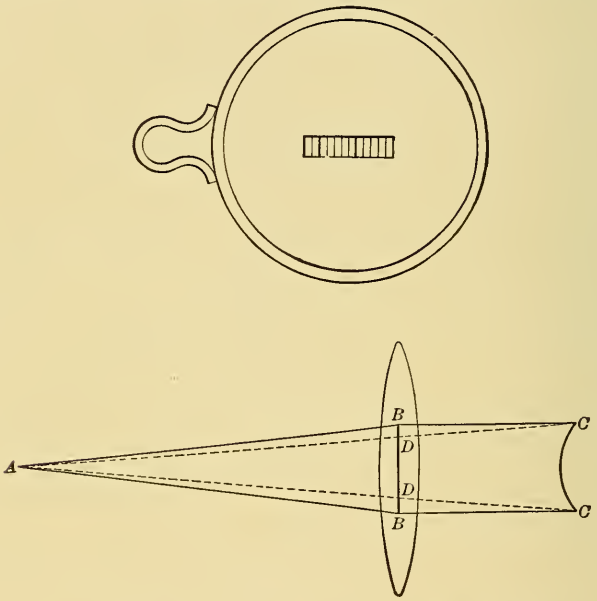
[The cornea can be examined with the ophthalmoscope (see page 93), and defects in the epithelium of the cornea can be detected by fluorescein (§ 193).—D.]

Besides examining the appearance of the cornea we have also to examine its sensitiveness, which is best done by touching it with the point of a thread.

77. The *anterior chamber* must be examined more especially in respect to its depth—that is, whether it is shallower or deeper, as a whole, or whether possibly it is of unequal depth. Further, we look for any abnormal matters which may be present in the chamber, such as an exudate, blood, foreign bodies, etc.

[The ophthalmoscope can be used both by the direct method and by direct illumination (§ 87) to demonstrate the presence of deposits, especially iritic precipitates in the anterior chamber. Furthermore, if the observer, standing to one side, holds the ophthalmoscopic mirror almost at right angles to the patient's line of sight, he can see deposits and adhesions in the angle of the anterior chamber, which ordinarily would not be visible because hidden by the limbus (Salzmann).—D.]

78. In the *iris* its color as well as the clearness of its markings must be observed. Special attention must be paid to the margin of the pupil, which should be examined—if need be, with the aid of a loupe—for irregularities (adhesions). In doubtful cases, in order to decide whether adhesions are present, a mydriatic (homatropine, atropine) should be instilled. At the



[Fig. 5.—Priestley Smith's Keratometer. (After Posey and Spiller.)

This consists of a lens containing a millimetre scale, *B*, *B*. If the observer places his eye at the focus of the lens, *A*, and looks through it at any object, as the cornea or iris, *C*, the size of the latter will be given accurately by the number of divisions that the object appears to cover on the scale.—D.]

same time we notice whether the iris quivers as the eye makes a sudden movement. Then we determine whether the pupil is round, whether its width is normal and equal to that of the other eye. (If the pupil is of abnormal size we do not neglect to inquire whether possibly some drug has been put into the eye which has caused an artificial alteration of the pupil.) Moreover, we see whether the pupil is centrally placed and of a clear black.

[The best way to measure the *width* of the pupil is with the Priestley Smith keratometer (Fig. 5), which, as its name implies, is also used to measure the cornea. For this may be substituted a + 3D lens, against which is held a millimetre scale. (The observer in this case should stand about a foot from the patient's eye).—D.]

79. Finally we investigate the *reaction of the pupil.* In order to determine the reaction to light, we have the patient look toward the window and make him cover one eye with his hand. We then cover with our own hand the eye that is being examined, and observe whether the pupil contracts when the hand is suddenly withdrawn. The patient is directed to fixate a distant object, since if he shifts his point of vision, the reaction for convergence might come into play too. This latter reaction must also be tested. This we do if we make the patient first look at a distance and then, holding an object (finger or pencil) close to his eyes, make him fixate that. [We must take care that the patient in making this quick change from distance to near does not shift his line of sight up, down, or sideways, nor do anything else that might change the conditions of illumination. We should also test the sensory reaction (§ 319). We do this by applying some sharp stimulus, e.g., by stroking the neck with a pin point, when, if the reaction is present, the pupil should dilate.—D.]

The light reaction of the pupil becomes still more clearly visible if we perform the test in a dark room, where after first shading the eye we suddenly concentrate the lamp-light with a strong convex lens upon the pupil, just as is done for focal illumination. [In doing this it is important that the patient look straight at the light, the eye not under examination being shaded. The observer standing to one side and watching the eye with a magnifying glass focuses the light right on the center of the cornea by another lens brought suddenly between the eye and the light (Uhthoff). If the faintest light reaction is present, the pupil will be seen to contract. On the other hand, there will be no reaction for accommodation or convergence, for the patient will have before his eye nothing but a blaze of light which by no possibility can form a clear image on his retina, so that he will make no attempt to focus it.—D.]

The contraction of the pupil in the illuminated eye resulting from the incidence of light upon it is the direct reaction, the simultaneous contraction in the pupil of the other eye is the consensual reaction. If the direct reaction is insufficient, the insufficiency may be due either to deficient light perception, or to changes in the iris or its nerves (adhesions, paralysis, etc.). Which of these is the case can be decided by means of the consensual reaction, provided the other eye is healthy. The consensual reaction is tested in two ways: (*a*) while alternately shading and illuminating the test eye we follow the movements of the pupil in its fellow. If the latter remains immobile this proves deficient light perception [or rather defective light conduction] in the former. (*b*) We alternately shade and illuminate the pupil of the [reacting] fellow eye and look for a reaction of the pupil in the test eye. If such reaction is absent the absence must be attributed to changes in the iris of the test eye.

[Sometimes, as in testing the hemiopic reaction (§ 546), it is necessary to illuminate only a portion of the retina of the examined eye. This is done with any apparatus that will throw a small concentrated pencil of rays.

The contraction which takes place in the pupil when suddenly exposed to light is succeeded by dilatation, and this may occur so quickly that the primary contraction escapes us.—D.]

80. Of the *lens* we see under ordinary circumstances only the small portion of the anterior surface which lies free in the pupil. If we wish to examine the lens more extensively, we dilate the pupil with homatropine [or cocaine] and use lateral illumination. As long as the lens is still transparent, the ophthalmoscope gives us the best conclusions in regard to its constitution. Whether the lens is present in the eye at all or not can be determined by investigating the Purkinje-Sanson reflex images. If a candle is placed before the eye and somewhat to one side of it, two brilliant reflections are observed. One of these at once attracts our attention by its size and brilliancy; it is the corneal reflex—that is, the erect image of the flame reflected from the anterior surface of the cornea (Fig. 6, *a*). It is this reflex which even from a distance is visible in every eye, and gives to the latter its fire and luster. The second reflex is quite as bright, but so small that we have to search for it in order to find it. It represents the very small inverted image of the flame which is reflected from the posterior surface of the lens (posterior lenticular reflex, Fig. 6, *c*). It is distinguished by moving in the contrary sense to the source of light when the position of the latter is shifted; if the candle is depressed, the shining point rises, and vice versa, in opposition to the corneal reflex, which moves in the same sense as the candle flame. It is this posterior lenticular image that is used in doubtful cases to demonstrate the presence of the lens in the eye. If the image is visible, the lens is present; if the image is not seen, either the lens is absent from its place or else is more or less opaque, so that a reflection can no longer be developed on its posterior surface. (Fig. 6 also shows a third reflex image, *b*, placed between the other two and originating from the anterior surface of the lens. The latter gives an erect image larger than the others, but so faint that it can be made out only with difficulty.)

Fig. 6.—Purkinje-Sanson's Reflex Images.

The black circle represents the dilated pupil of the eye that is being tested. The candle flame is supposed to be to the right, the observer's eye to the left of the pupil. *a*, image formed by the anterior surface of the cornea. *b*, image formed by the anterior surface of the lens. *c*, image formed by the posterior surface of the lens.

81. Finally, before proceeding to an examination with the ophthalmoscope, the *tension* of the eye is to be tested. The eye is closed and palpation is made by means of the two index fingers, which are placed upon the upper

lid. The tension that is thus appreciated is not, indeed, identical with the intra-ocular pressure, but is proportional to it, and may hence be employed as an expression of it. In so doing we must, however, take into consideration the fact that the sensation which the finger appreciates when it palpates the eyeball is not dependent simply on the tension of the tunics of the eye. What we appreciate is the depth of the impression which the finger makes on the eyeball and the force which we have applied in making it. But both depend not simply on the tension or the intra-ocular pressure, but also on the elasticity of the coats of the eye, and their flexibility or rigidity. Hence on account of the greater rigidity of the sclera, the eyes of old people feel harder than those of younger persons, even when the intra-ocular pressure is the same.

Furthermore, individual variations occur both in the intra-ocular pressure and in the elasticity of the coats of the eye, so that very slight variations from the usual tension cannot be denoted as surely pathological, except when we can make use of the second, normal eye of the same man for the purpose of comparison. Greater alterations in tension, however, make themselves evident at once. It has been agreed to denote the normal tension by the expression Tn (T = tension or tonus). Of increased tension (hypertony) we distinguish three degrees: T+1, T+2, and T+3, which are arbitrarily selected, and indicate approximately: tension noticeably increased; greatly increased; hard as stone. Similarly, we employ for diminished tension (hypotony) the designations T−1, T−2, and T−3.

The estimation of the tension by palpating the eye requires much practice, and even then is an inexact method. It does not suffice to determine in a positive way very small variations from the normal; and as little can it disclose with certainty small variations of tension which occur in the course of a case of disease, since one cannot designate in any precise way for later comparison the sensation which he receives in making palpation. Hence it is advisable to use a *tonometer*. Just as in the examination with the finger, the tonometer determines the tension by the fact that an impression is made on the eyeball; the force with which this is made is given by the weight used for the purpose; and the depth of the impression which is produced and which on account of its shallowness is better characterized as a flattening, is measured. In Schiötz's tonometer a collar (Fig. 7, *b*) bears at its lower end a concave plate (*c*) which is fitted to the curvature of the cornea, and which, as the patient lies upon his back, is placed upon the cornea, the latter being previously made insensitive. (For this purpose since cocaine diminishes the intra-ocular pressure somewhat, we use holo-

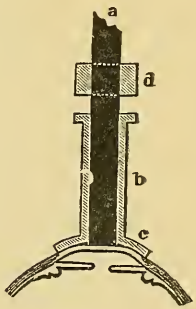
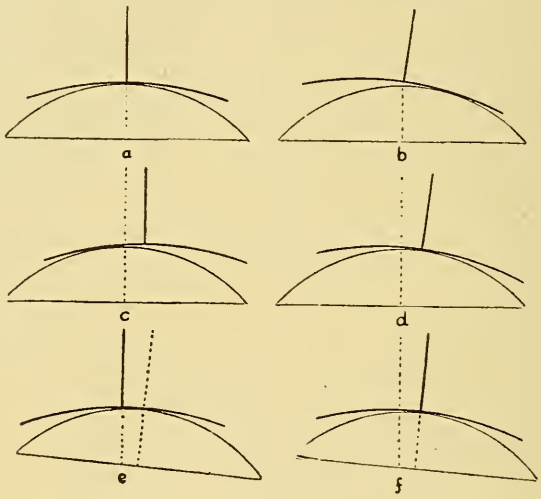


Fig. 7.

caine, which does not have this effect.) Through the collar and its foot-plate, which has a hole bored through its middle, passes the rod (*a*) which glides to and fro in them without friction. At its upper end the rod is loaded with a small weight (*d*), of which there are four different grades. When the instrument is placed perpendicularly upon the cornea the rod, owing to its weight, drops down upon the center of the cornea. If the rod cannot flatten the cornea its lower end is arrested at the level of the foot-plate, and a pointer which is connected with the upper end of the rod fails to show any vibration. The less tense the eye is, the more readily can the rod flatten the cornea and drops correspondingly far out of the collar, as is shown by the pointer.

[In applying the tonometer it is important that the patient's cornea should be directed straight up, that the plunger of the instrument should also be just vertical, and that its foot-plate should be concentric with the cornea (see Fig. 8).



[Fig. 8.—Application of the Tonometer. (After Priestley Smith.)
a, correct position; *b*, *c*, *d*, *e*, *f*, incorrect positions. *c* causes the most error; *e* and *f* cause the least.—D.]

When several tests are made with the tonometer in rapid succession, the later ones often show a lower value than the first. From this it is inferred that the repeated application of the instrument reduces the tension of the eye to below its usual amount. Too many tests, therefore, should not be made close together at one sitting and if the last of several readings is lower than the rest it should be rejected.

The tension as found with the tonometer in normal eyes varies between 13 and 27, and usually between 18 and 22, mm. of mercury. A tension above 25 is probably, and one above 30 quite certainly, pathological.

As stated above, the tension measured by the finger or tonometer does not precisely represent the intra-ocular pressure, being dependent partly on this, partly on the rigidity of the ocular envelopes. If the cornea is unduly rigid, the tension shown by the tonometer will seem too high, even when the intra-ocular pressure is really normal. If, on

the other hand, as in keratoconus, the cornea is unduly yielding, the intra-ocular pressure may be abnormally high, and yet the tension shown by the tonometer may be normal or subnormal. In this case there is said to be a *relative* increase of tension (Strebel and Steiger). And even if the intra-ocular pressure itself is no higher than usual, it must be regarded as relatively too high, if, as in keratoconus and progressive myopia, the ocular envelopes are so yielding as to give way before it.—D.]

Examination with the Ophthalmoscope (Ophthalmoscopy)

82. The invention of the ophthalmoscope by Helmholtz in the year 1851 was one of the most beneficent achievements in modern medicine. It has made the interior of the eye accessible to investigation; blood-vessels and nerves, which in the rest of the body are exposed only by surgical manipulation, here lie unveiled before us and permit us to study their minutest variations. In ophthalmology, the ophthalmoscope has produced a com-

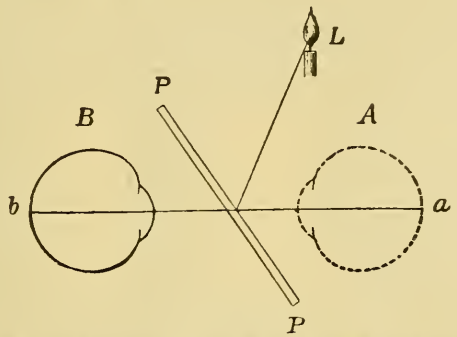


Fig. 9.—Principle of Helmholtz's Ophthalmoscope.

plete revolution, since it has thrown light into the dark region of what was formerly called black cataract, and has acquainted us with the manifold morbid processes which lie at the root of this dreaded malady. Many of these processes, if diagnosticated correctly and in time, would, at the present day, receive successful treatment. Furthermore, in general medicine the ophthalmoscope has become an indispensable aid to diagnosis, since many internal disorders produce characteristic changes in the fundus of the eye.

Principle of the Ophthalmoscope.—In order to see the fundus of an eye, we must throw light by the aid of suitable apparatus through the pupil and upon the fundus, and receive the light reflected from the latter into our own eye and unite the rays to form a sharp image. In the original ophthalmoscope of Helmholtz this end was obtained in the following way: Before the eye under investigation (*A*, Fig. 9) a glass plate, *P P*, is placed in an oblique position. A source of light, *L*, placed to one side of the eye, throws upon the glass plate rays, part of which are reflected at the surface of the plate and pass through the pupil into the eye, *A*. The rays reflected from

the fundus, *a*, arrive once more at the glass plate and are there in part reflected to the source of light, *L*, while another part goes through the glass plate and enters the observer's eye, *B*, which unites the rays upon its retina

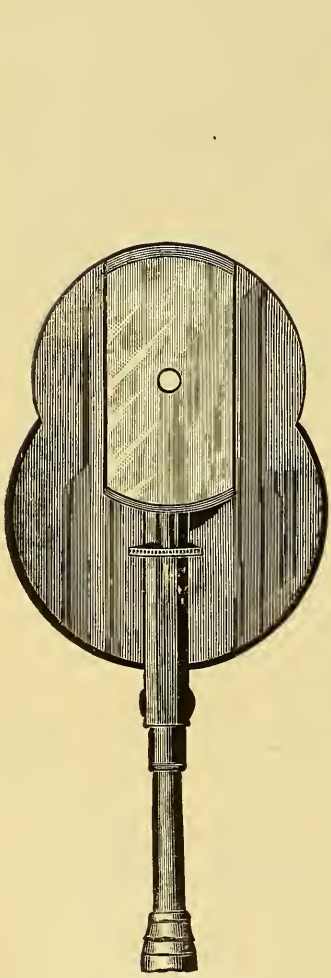


Fig. 10A.

Fig. 10B.

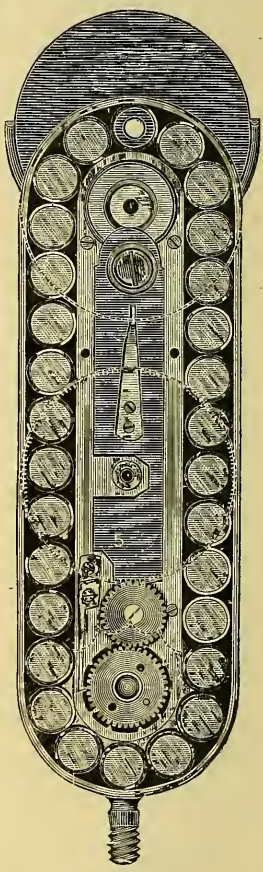


Fig. 10C.

[Ophthalmoscopes.

A.—Loring's ophthalmoscope; front view. The perforated mirror in this case is cut into a quadrangular shape, so that it can be rotated slightly from side to side (tilting mirror). This is to make the reflection of light less oblique in using the direct method. The lower disk contains a series of lenses which can be rotated by the pressure of the finger on the serrated edge of the disk, so that any desired lens can be brought in front of the sight-hole. Usually there is added a quadrant on the back, containing additional lenses, which being superimposed over those in the revolving disk make a great variety of combinations.

B.—Marple's modification of Dennett's electric ophthalmoscope.

C.—Morton's ophthalmoscope; rear view, showing the chain of lenses and the mechanism by which each one in succession can be brought in front of the sight-hole in the disk at the top which contains the mirror.—D.]

[Fig. 11.—Ophthalmoscopy. The Direct Method with Electric Ophthalmoscope. After Würdemann (from Posey and Spiller).—D.]

Fig. 12.—Ophthalmoscopy. The Indirect Method with Reflecting Ophthalmoscope. After Würdemann (from Posey and Spiller).—D.]

into a sharp image, *b*. In order to increase reflection at the surface of the plate and thereby illuminate the background of the eye more intensely, Helmholtz placed three such plates one behind the other. A later modification consisted in increasing the reflecting power of the glass plate by lining its posterior surface with a mirror coating, a round hole through the plate or at least through the mirror coating enabling the observer to see through it. Of this sort are the coated plane or weakly reflecting mirrors we use today; while we apply the term "strongly reflecting" to concave mirrors, which also are coated and are perforated through the center (first employed by Ruete). These, from the fact that they render convergent the rays springing from the source of light, throw a still greater quantity of light through the pupil into the observed eye. A device is placed on the posterior surface of the mirror, which renders it possible to bring different sorts of lenses before the aperture. In this way it is possible to give the rays of light which fall into the observer's eye any path that may be necessary in order to unite them into a sharp image upon the retina.

[Instead of having a separate light as a source of illumination we may attach a minute electric lamp to the ophthalmoscope itself. These *electric ophthalmoscopes* (see Fig. 10, B) give a very clear view of details and frequently may be used where the ordinary ophthalmoscope is difficult to manage, e.g., when the pupil is small, or the patient is restless, or when the examination has to be made with the patient lying flat in bed.

Demonstration ophthalmoscopes are also used, through which two or more observers can see the fundus simultaneously; also ophthalmoscopes specially designed for sketching the fundus.—D.]

83. Method of Examination.—The examination is conducted in a darkened room. The patient sits opposite the physician, and has on the side of the eye to be investigated a lamp as a source of light. Then there are two different methods to be employed for seeing clearly the fundus of the eye. In order to make the explanation of them simpler, we first presuppose that both the patient and the physician have a normal refraction (emmetropia, see § 752). In the examination with the *erect image* (*direct method*), the physician places himself and his mirror directly in front of [and very close to] the eye that he is observing. [See Fig. 11.] If now he holds the mirror obliquely in such a manner that he throws the light of the lamp into the pupil of the observed eye, he will immediately get a clear view of the patient's fundus. For (Fig. 13) a certain portion of the fundus of the eye, *A*, is illuminated by the mirror, *S S*. The rays reflected from any point, as *a*, of this illuminated region of the retina, leave the eye in a parallel[3] direction, pass through the central aperture, $o\ o_1$, of the mirror, and fall into the observer's eye, *B*. Here they are again united at a single point, *b*, upon the retina of this eye, so that here there is produced a sharp image of the point *a*. Since the same process is repeated for all the other points of the

[3] [Parallel, because if the eye is emmetropic, i.e., focuses parallel rays upon its retina, then, according to the principle of conjugate foci, rays emanating from the eye will also be parallel. Being parallel, such rays will also be focused sharply by the observer's eye, which is also emmetropic.—D.]

illuminated region of the retina of the eye, *A*, a sharp image of this portion of the retina is formed in the eye of the observer.

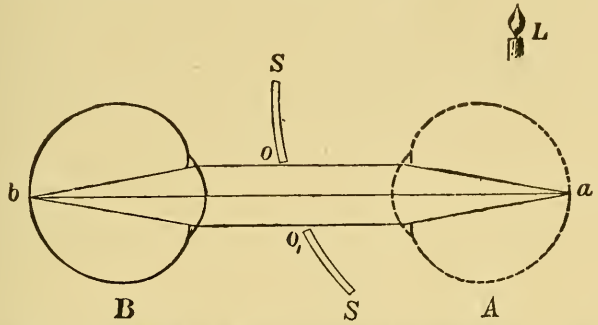


FIG. 13.—OPHTHALMOSCOPIC EXAMINATION WITH THE ERECT IMAGE.

The eyes are drawn of the natural size of an emmetropic eye having an axial length of 24 mm. The rays that are reflected from the mirror, *S S*, into the patient's eye are not shown, but only the rays that emerge from the latter.

[If not emmetropic, both patient and observer must be rendered so, if the fundus is to be clearly seen by the direct method.

This is usually effected by rotating the system of lenses behind the mirror of the ophthalmoscope until the proper correction is secured. A still better way, particularly when either patient or observer is quite astigmatic, is for each to wear his correcting glasses during the examination. Then the fundus is seen with great distinctness either with the simple aperture or, if either patient or observer accommodates, with a weak concave lens.

As stated below, the direct method may be used not only for examining the fundus at its normal level, but also for examining elevations or depressions in it (§ 100) and objects (opacities in the vitreous or lens) far in front of it (page 93). In this case we must interpose behind the mirror a suitable lens, convex or concave, as the case may be.—D.]

84. The examination with the *inverted image*, or by means of the *indirect method* (Ruete), is conducted with the aid of a strong convex lens

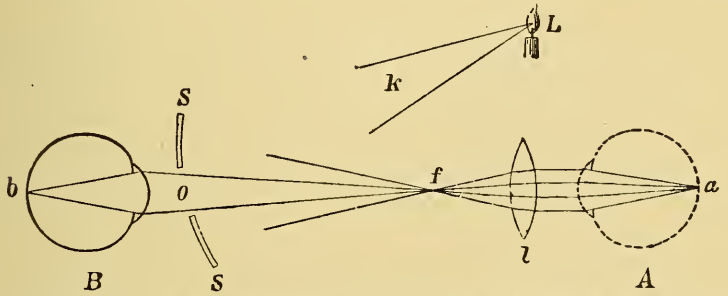


FIG. 14.—OPHTHALMOSCOPIC EXAMINATION WITH THE INVERTED IMAGE.

The illumination of the fundus is accomplished by means of the source of light, *L*, from which the cone of rays, *k*, falls upon the mirror, *S S*, and from this is thrown into the eye, *A*. In order not to confuse the representation, these rays are not shown, but only those which pass out of the eye, *A*, again.

of about six cm. focus. This lens, *l* (Fig. 14), is held at a distance of about six [seven] cm. from the eye (*A*) under examination. [See Fig. 12.] The

fundus of this eye is now illuminated by means of the mirror *S S*. The rays reflected from the illuminated region, *a*, of the retina pass out in a parallel direction, fall upon the lens and are united at the focus, *f*, of the latter. Thus there is formed at this spot an image of the point *a*. In like manner images from the other points of the illuminated region of the retina are produced in the focal plane of the lens, so that there is formed here an *inverted* image of this portion of the fundus. The observer's eye, *B*, now through the aperture, *o*, of the mirror examines this image at the ordinary reading distance (about thirty cm.), for which purpose the observer, unless he is myopic, must use a certain accommodative effort or else a corresponding convex glass [which he does by revolving the ophthalmoscope disk until a 2 or 3 D convex glass stands before the aperture].

85. Each of these two methods has its advantages. The erect image is highly magnified—about fourteen times—as opposed to the inverted image which is magnified but little (about four times). The direct method is therefore particularly adapted for the recognition of the finer details. The indirect method, on the other hand, affords a larger field of view, and therefore gives a better general prospect. The indirect method gives a more luminous image, and hence, when the refracting media are turbid, will still render the fundus visible when it is no longer to be seen in the direct image; and in myopia of high degree the indirect method is the only one practicable. In most cases, both methods are applicable, and then it is advisable to conduct the examination with the aid of both.

[The amount of magnification produced with the indirect method depends on the strength of the condensing lens that is used. If we employ a lens of two inches focal distance the details of the fundus appear magnified only two or three times. To compensate for this we get a very wide general view of the fundus. If we use a lens of three or four inches focal distance we get a much higher magnification—indeed one that approximates to that with the direct method. In all cases the lens ought to be held so that its focus is about half an inch in front of the eye; that is, a four inch lens should be held four and one-half inches from the eye, etc. If the lens is so held, the distortion of the details produced by astigmatism is reduced to the minimum. The corneal reflex, which, the beginner finds, interferes a good deal with his view of the fundus, can be got out of the way by a slight tilting of the lens, if he is using the indirect method, or a slight tilting of the ophthalmoscope itself when he is making a direct examination.

In the "reflexless" ophthalmoscopes of Thorner, Gullstrand, Wolff, and others, the elimination of the reflex is accomplished by making the light enter at one part of the pupil and emerge at the other.

In ophthalmoscopy, especially by the direct method, it is advantageous to keep making slight tilting movements of the mirror, so as to play the light a little this way and that over the fundus, the details and relief of which are thus better shown. Moreover, as Nagel first pointed out, details are often seen best if we so manage the light that the point to be examined lies at the edge instead of the center of the illuminated area in the fundus ("excentric illumination" of Burdon Cooper).—D.]

Luminosity of the Pupil.—Under ordinary circumstances the pupil appears black. This was formerly ascribed to the absorption by the dark background of the

eye of all the light entering the pupil from the outside. In reality, however, the cause of this phenomenon is as follows: If (Fig. 15) light from a source of light, *L*, enters the eye, *A*, and the latter is accurately focused for the source of light, the rays coming from *L* are united to form a sharp image upon the retina at *l*. *L* and *l* are called conjugate foci. For these the law holds good that they can be substituted for each other—that is, if the rays should start from the posterior focus, *l*, they would come together again at the anterior focus, *L*. Accordingly, the rays reflected from the illuminated portion of the retina, *l*, are returned to the source of light, *L*, and could be seen by an

Fig. 15.—Path of the Rays when the Eye is Focused for the Source of Light.

observer only in case the latter was in identically the same spot as the source of light. The solution of this problem is another of the discoveries that we owe to the genius of Helmholtz.

The conditions are different when the eye is not focused for the source of light before it. Suppose, for example, that the eye is hypermetropic (Fig. 16). Then the rays springing from the illuminated portion of the retina, *l*, leave the eye as a divergent beam, so that only a part of the rays are returned to the source of light, *L*, while another part passes to the side of the latter and can be seen by an observer stationed near it. Hence comes the striking luminosity of the pupil in so-called amaurotic cat's eye (see § 535), in which a markedly hypermetropic condition of the refraction is produced by the pushing forward of the retina. In like manner, luminosity is frequently apparent in eyes which are deprived of their lens by the operation for cataract and are therefore

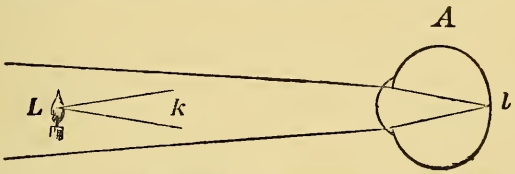


Fig. 16.—Explanation of Luminosity of the Eye.

The source of light, *L*, throws the conical beam of rays, *k*, into the eye. The further course of these rays is not shown in the figure, but only that of the rays reflected from the retina at *l*.

strongly hypermetropic. The enlargement of the pupil, which is at the same time present, and which is due to the iridectomy, renders it still easier to observe the luminosity. The luminosity of the eyes of many beasts, especially the carnivora, is also in part to be ascribed to the hypermetropic character of their refraction, although here the presence of a strongly reflecting layer (the so-called tapetum) in the chorioid of these eyes contributes to this result.

The luminosity of the pupils of *albinos'* eyes is to be explained in a different way. In such eyes the light passes not only through the pupil, but also through the unpigmented iris, and even through the sclera. Accordingly, in these eyes, not simply a limited district of the retina, but the whole fundus, is flooded with diffused light; and therefore rays from the different portions of the fundus pass out of the pupil in every direction and can very readily be caught up by the observer's eye. That this explanation is the correct one is proved by the fact that the pupil of an albino's eye looks black

as soon as we hold before the eye an opaque screen having an aperture that corresponds in size to the pupil. This shuts off from the eye any light which might enter it through other media than the pupil, and in this respect makes the eye of an albino like that of a normal person.

86. Application of the Ophthalmoscope.—In the ophthalmoscopic examination of the eye we invariably proceed by first carefully investigating the eye by means of lateral illumination, next testing the transparency of the refracting media with the ophthalmoscope, and not going on to the examination of the fundus itself until last of all. This last examination is best made first with the inverted, afterward with the erect image; and in examining the latter the refraction can be determined at the same time. If the pupil is narrow, the tyro will do well to dilate it with [cocaine, euphthalmine, or ½-per-cent] homatropine. Before doing so he must make sure that there is no reason to suspect glaucoma, in which case artificial dilatation of the pupil might have dangerous results, and therefore must not be employed.

[To be on the safe side it is well to instil pilocarpine or, in suspicious cases, ¼-per-cent eserine after the examination, in order to contract the pupil again.

The ophthalmoscope is also used to examine the angle of the anterior chamber (see page 80).—D.]

87. For testing the *transparency of the refracting media* light is thrown by the ophthalmoscope, held at the ordinary reading distance, into the eye under examination. If the refracting media are perfectly clear, the

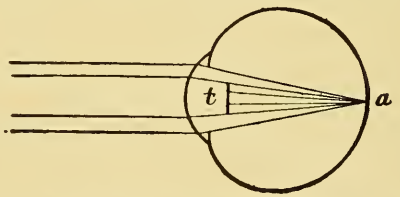


Fig. 17.—Visibility of Opacities in the Media by Means of the Ophthalmoscope.

pupil shines with a uniform red luster. If there are places in the refracting media that are opaque, such stand out upon the red background of the illuminated pupil as dark points or spots. For example, the rays which correspond to the opaque spot *t* (Fig. 17) are cut off on their return from the fundus, *a*, so that this spot is not illuminated and hence looks black. This is the case even when the opacities are actually, as seen by light thrown directly upon them, light colored—that is, white or gray. So also even a piece of chalk looks black if it is held in front of a flame.

When pronounced opacities are present, we make use of the concave mirror; slight opacities, on the other hand, are discovered only by means of the weakly reflective or plane mirror; and in this case it is often also necessary to dilate the pupil with homatropine. If the observer is emmetropic, and still more if he is hypermetropic, he ought to place a convex glass behind his mirror, so as to be able to get near enough to the eye

that he is examining. (The combination of a plane mirror with a convex glass is called a lenticular mirror.) A myopic observer will not need such a glass.

[The above method of examining opacities, in which the observer, using a weak convex glass, stands eight or ten inches from the patient, and views the opacities as shadows on a red ground, is called *direct* (as distinguished from oblique) *illumination*. We may also examine the opacities by the *direct method*. In this case we place our eye within an inch or so of the patient's and put on the convex glass that will bring the opacity sharply into focus. For opacities in the vitreous a convex glass of 3 to 18 D, for opacities in the lens and anterior chamber (especially deposits on the posterior surface of the cornea) one of 15 to 20 D will be required, the strength of glass depending a good deal on the distance of the observer from the patient.—D.]

In examining, we must not neglect to make the eye move in different directions, in order, on the one hand, to obtain a view of laterally placed opacities, and, on the other hand, to stir up in this way opacities which have sunk to the bottom of the vitreous humor. Smaller opacities look black; larger opacities appear gray, bluish-white, or even all white, since the light reflected from their surface is strong enough to shine out upon the vivid red background of the illuminated pupil. In order to recognize the site of the opacity,

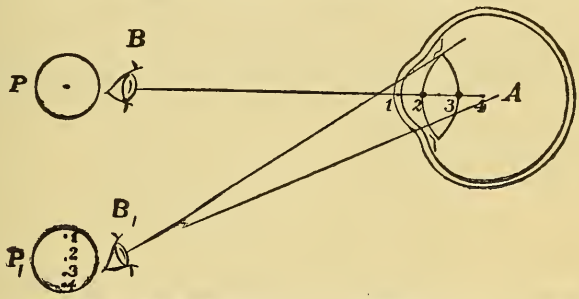


Fig. 18.—Diagnosis of the Site of an Opacity from Parallactic Displacement.

we decide, in the first place, whether the latter is movable or fixed. In the former case it can be situated only in the vitreous; in the latter case—that is, if the opacity moves only with the eye, and not spontaneously—it is probably situated in the cornea or in the lens; but it may still be in the vitreous, since here too fixed opacities are sometimes observed. In many cases this can be decided by employing oblique illumination. If we can obtain no result in this way, we then, in order to determine the site of the opacity, make use of the *parallactic displacement* of the latter with reference to the margin of the pupil. This is accomplished in the following way: In the eye, *A* (Fig. 18), suppose four opaque points to be present, which lie at different depths—namely, in the cornea (1), upon the anterior capsule of the lens (2), at the posterior pole of the lens (3), and in the anterior part of the vitreous (4). For simplicity's sake we assume that they are all disposed in the optical axis of the eye. Then, if the observer, *B*, looks into the eye from directly in front, he will see each one of these points precisely in the center of the pupil, *P*. Suppose, now, that the observer's eye passes from *B* to B_1. The position of the points with relation to the pupil will be changed at once. Point 1 approximates to the upper border of the pupil P_1; point 2, which is situated in the pupil itself, keeps its place unchanged; points 3 and 4 have approached the lower border of the pupil, and 4, on account of its greater depth in the eye, more so than 3. From this example the following rule for the determination of the site of an opacity can be deduced: We look into the eye from directly in front and note the position of the opacity in the pupil. Then, while the patient holds his eye fixed, we move slowly to one side and observe whether the

opacity remains in the same spot or not. In the former case, the opacity lies in the pupillary plane (upon or directly beneath the anterior capsule of the lens); in the latter case, in front of or behind this plane—in front of it, if the opacity shifts its place with a movement opposed to that of the investigating eye; behind it, if the opacity moves in the same sense as the eye. The more quickly this change of place occurs, the farther removed is the opacity from the pupillary plane. (Evidently we can also proceed by keeping our own eye still and telling the patient to move his. This way of examining has the disadvantage that, if the movement of the observed eye is rather extensive, a minute opacity, whose position has been marked, may disappear out of sight and then frequently is found again only with difficulty.)

[Another way of determining the site of an opacity is by its relation to the *corneal reflex*. We use the direct illumination; that is, we stand about a foot or so in front of the patient, and throw light directly into his eye. If his eye is directed straight at ours, we will see the bright corneal reflex about in the center of his pupil. If now he moves the eye in any direction, the reflex leaves the center of the pupil and approaches the margin of the cornea. At the same time we notice how any opacities that may be situated in the area of the pupil seem to move. An opacity, which appears close to the corneal reflex when in the center of the pupil and keeps close to the reflex in all its movements, must be situated at

[Fig. 19.—Würdemann's Transilluminator or Diaphanoscope.—D.]

or near the posterior pole of the lens. Such an opacity, for example, is produced by a posterior polar or posterior cortical cataract. On the other hand, the more remote an opacity is from the posterior pole of the lens, whether in front of it or behind, the further and more quickly will it appear to separate from the corneal reflex when the eye is moved.—D.]

Dark, ill-defined shadows on the red background of the pupil, which change their position suddenly on moving the mirror, are to be referred to irregularities of the refracting surfaces (most frequently to faceting of the cornea); the irregular astigmatism, so caused, further betrays itself by the fact that the image of the fundus appears irregularly distorted.

[The presence in the eye of a large-sized solid mass, especially a tumor, can be shown by *transillumination* or *diaphanoscopy*. This can be used even when the lens is opaque, so that the ophthalmoscope is not available. Special instruments for transillumination of the eye are those of Leber, Sachs, and Würdemann (see Fig. 19) and the ophthalmodiaphanoscope of Hertzell. In case of emergency these instruments may be replaced by a small narrow-tipped flash light. If these are applied to the sclera a red glow is seen in the pupil. If the light is interrupted in any place by a solid mass, the red glow is replaced by a more or less dense shadow, when the tip of the transilluminator is placed against the corresponding portion of the sclera. This test may fail with deeply pigmented eyes (e.g., those of negroes), since these even when otherwise normal may show no red reflex by transillumination (Griscom).—D.]

88. In making a systematic *examination of the fundus* we begin at the *papilla* [or *disk*]. In order to bring the latter into view we make the patient look, not straight in front of him, but a little inward (toward his nose). For the entrance of the optic nerve does not lie at the posterior

pole of the eye, but on the nasal side of it, and hence is brought directly opposite the observer only after a corresponding rotation of the eye inward. It then comes into view as a bright disk, whose color is a light grayish or yellowish red, contrasting strongly with the red of the rest of the fundus. The shape of the papilla is circular or oval; in the latter case generally an erect oval (Fig. 20). Its *size* apparently varies quite a good deal, which, however, is due to the varying degree of enlargement under which the papilla is seen. The true size of the papilla, measured in enucleated eyes, is, as a matter of fact, almost always the same—that is, about 1.5 mm. in diameter.[4] On account of this constancy we use the papilla for taking measurements in the fundus; we say, for example, that a diseased area is 2 papilla-breadths in diameter.

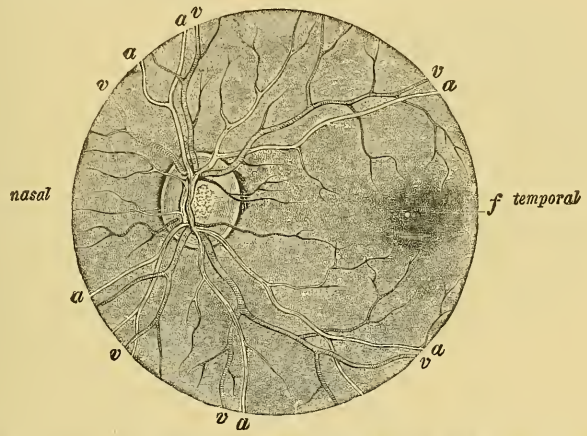


Fig. 20.—Normal Fundus of the Left Eye, seen in the Erect Image.

The optic disk, which is somewhat oval longitudinally, has the point of entrance of the central vessels somewhat to the inner side of its center. That portion of the papilla lying to the inner side of the point of entrance of the vessels is of darker hue than the outer portion; the latter shows, directly to the outside of the vascular entrance, a spot of lighter color, the physiological excavation, with fine grayish stippling, representing the lacunæ of the lamina cribrosa. The papilla is surrounded, first by a light-colored ring, the scleral ring, and externally to this by an irregular black stripe, the chorioidal ring, which is especially well marked on the temporal side. The central artery and vein divide immediately after their entrance into the eye into an ascending and descending branch. These branches, while still on the papilla, split into a number of arteries which in the upper half run mainly upwards and outwards (superior temporal artery and vein) and upwards and inwards (superior nasal artery and vein), and in the lower half run downwards and outwards (inferior temporal artery and vein) and downwards and inwards (inferior nasal artery and vein). Moreover, fine twigs (the macular vessels) run from the papilla over the temporal border of the latter directly to the macula lutea, while other twigs from the upper and lower temporal vessels push toward the macula lutea, which itself is devoid of vessels and is distinguished by its darker color. In its center a bright punctate reflex, *f*, is visible.

89. *Circumscribing the papilla*, we very often (especially in making the examination with the erect image) recognize two rings distinct in color. The inner ring, lying next the border of the papilla, is white (in Fig. 20 it runs all the way round the papilla, in Fig. 21 it appears only on the temporal side), and is called the scleral (or connective-tissue) ring, because it corresponds to the scleral bundles which jut in between the

[4] [This corresponds to an arc of 7.5° in the fundus. The blind spot (§ 112) which is the visual correlative of the optic disk naturally subtends the same angle on the perimeter.—D.]

margin of the chorioid and the head of the optic nerve (Fig. 24). At the margin of the aperture in the chorioid that gives passage to the optic nerve, the chorioid is often marked by a larger accumulation of pigment by which the second, exterior, ring is formed. This is apparent as a black, narrow, sometimes complete, sometimes incomplete, ring, which is called the *chorioidal ring* or pigment ring (Fig. 21; in Fig. 20 it is especially visible at the outer border of the papilla). The demarcation of the outline of the papilla, produced in this way, is generally much less sharp on the nasal than on the temporal side; for at the nasal side a greater number of nerve fibers pass over the margin of the papilla (Fig. 24) and thus obscure it. For the same reason the inner half of the papilla looks redder, the outer half paler, because the layer of nerve fibers in the latter situation being thinner, allows the lamina cribrosa to show through more.

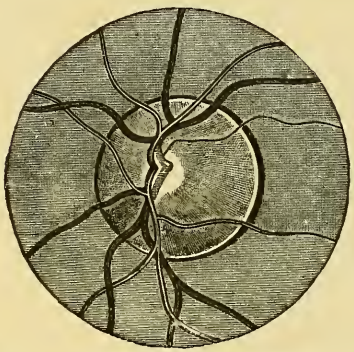


FIG. 21.—OPHTHALMOSCOPIC PICTURE OF THE OPTIC NERVE.

Somewhat to the nasal side of the center of the papilla emerges the central vein and to the nasal side of this again emerges the central artery. The main trunk of these vessels is itself not visible, since each divides at once into two main branches which run respectively upward and downward. The center of the papilla bordering the temporal side of the central vessels is rather lighter colored and somewhat depressed (the vascular funnel). On the temporal border of the papilla can be seen the white scleral ring. The papilla is surrounded by the black chorioidal ring.

Crescent or Conus.—The gap in the sclera and that in the chorioid, through which the optic nerve passes, together form a short canal, the sclero-chorioidal canal. The shape of this canal is by no means always exactly as Fig. 24 represents it, but it varies pretty considerably even in normal eyes, and to this are due the different pictures which the circumference of the papilla is accustomed to exhibit. The canal, as shown in Figs. 23 and 27, may keep narrowing all the way in its course toward the interior of the eye. But it may also happen that only one wall of the canal shows an oblique course pointing toward the axis of the optic nerve, while the other wall runs straight forward or is actually turned away from the optic nerve. In the latter case, which is represented in Fig. 22, the head of the optic nerve appears to be displaced bodily to one side within the sclero-chorioidal canal. This state of things occurs most frequently in near-sighted eyes (see §§ 413 and 771); but, since it is often enough also found in emmetropic and even in hypermetropic eyes and furthermore is visible with the ophthalmoscope, it must be considered here. If we should look at the optic nerve represented in Fig. 22, with the ophthalmoscope, we should on the temporal side look right into the scleral canal, because the tissue of the optic nerve-head itself is transparent. Then the temporal wall of the scleral canal would be visible in perspective foreshortening from *b* to the edge of the chorioid *c*, i.e., from the point where the canal begins to dilate (about corresponding to the place where the fibers of the lamina cribrosa are given off). With slight degrees of displacement the appearance is that of a rather broad scleral ring, but when the displacement is greater it is like a white crescent adjoining the margin of the papilla. Such a *scleral crescent*, which is also called a *conus* (Jäger), is most frequently met with at the temporal border of the nerve.

A light crescent at the border of the papilla may also be produced in another way. In the case represented in Fig. 23, the sclero-chorioidal canal is narrowest anteriorly, so that we could not see into it with the ophthalmoscope. But on the temporal side the pigment epithelium ceases at some distance from the margin of the opening of the optic nerve (at *d*). Over the area *bd* where this occurs, the chorioid, which is also itself somewhat rarefied, is exposed, and hence this spot appears as a light colored crescent adjoining the optic nerve. This *chorioidal crescent* or conus is distinguished from the scleral crescent, described above, by the fact that it is not pure white, like the latter, but shows remains of chorioidal vessels and pigment.

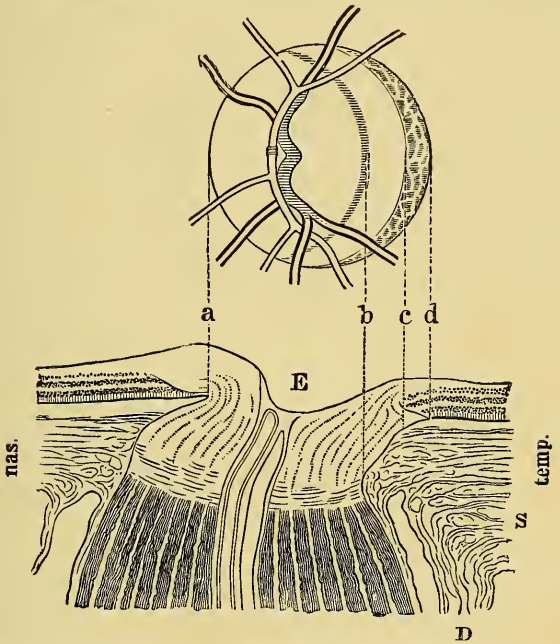


FIG. 22.—LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE IN A CASE OF SCLERAL CRESCENT AT THE TEMPORAL SIDE TOGETHER WITH THE CORRESPONDING OPHTHALMOSCOPIC PICTURE. Magnified 20×1.

In the section which has been stained by Weigert's method the nerve fibers of the trunk of the optic nerve being medullated have taken on a dark color. They become light colored at the spot where they lose their medulla, that is, at the level of the lamina cribrosa, which crosses the optic nerve in a slightly curved arch having its concavity forward. As far forward as the lamina the trunk of the optic nerve undergoes a uniformly conical narrowing. On the nasal side this narrowing is continued forward and even more markedly since here the wall of the sclero-chorioidal canal juts in more than ever toward the axis of the nerve. But by the same amount that this wall juts in toward the nerve is the temporal wall of the sclero-chorioidal canal separated from the nerve, so that the anterior border of the scleral opening lies at *c*, and the wall of the scleral canal becomes visible with the ophthalmoscope as a white crescent in the space *b c*. The margin of the lamina vitrea and of the pigment epithelium lies still further toward the temporal side, at *d*, and up to this point the fibers of the optic nerve are drawn over to the temporal side to form a pointed process. Between *c* and *d* the outer layers only of the chorioid are present, and these appear with the ophthalmoscope under the form of a spotted (chorioidal) crescent. On the temporal side the outer layers of the retina come to a stop sooner than do the inner ones, while on the nasal side of the nerve-head the reverse obtains. The nerve-head shows a shallow depression (physiological excavation, *E*). *S*, sclera, the inner layers of which extend up to the optic nerve while the outer ones even some distance down are reflected backwards into the dural sheath, *D*.

These two kinds of crescents are frequently found combined, as in the case shown in Fig. 22. For here the posterior layers of the chorioid extend pretty close to the optic nerve, in fact as far as *c*, while the lamina vitrea together with the pigment epithelium has receded to a greater distance from the nerve, *d*, at which spot also the fibers of the optic nerve are drawn out into a point through a gap in the border of the lamina vitrea.

Consequently, the posterior lamellæ of the chorioid are exposed from *c*, the anterior border of the scleral canal, to *d*, the anterior border of the chorioidal canal. The resulting appearance in the ophthalmoscopic picture is as follows: Close to the papilla is a pure white crescent (*bc*), and adjoining this is a narrower crescent (*cd*) which is lighter colored than the rest of the fundus, and presents some chorioidal vessels as well as a little pigment.

For colobomata of the optic nerve, see § 435.

Pseudoneuritis.—Not infrequently there are papillæ which are dark grayish red, and which, especially on the nasal side, have a very indistinct margin. Sometimes there is a cloudy gray areola about the papilla or a gray radial striation emanating from it which obscures the borders of the papilla; there may even be a slight protrusion of the papilla.

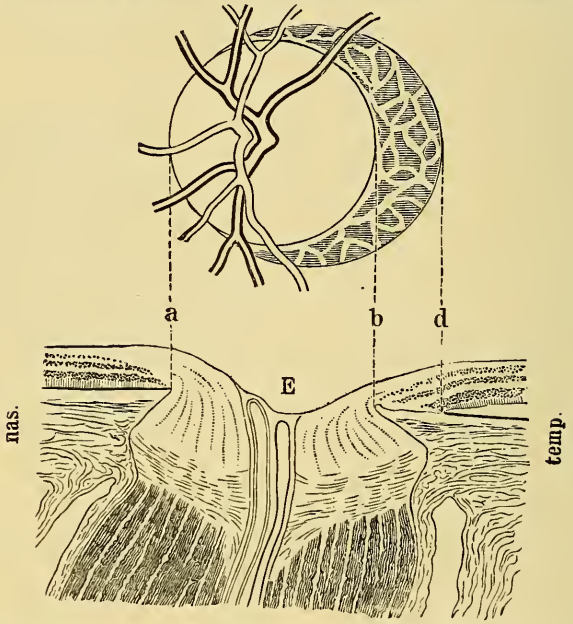


FIG. 23.—LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE IN A CASE OF CHORIOIDAL CRESCENT ON THE TEMPORAL SIDE TOGETHER WITH THE CORRESPONDING OPHTHALMOSCOPIC PICTURE. Magnified 20×1.

Here the medullary coating of the nerve fibers disappears further back in the central than it does in the peripheral bundles. In its course from behind forward the scleral canal shows first a dilatation then again a contraction which becomes still more marked in the chorioidal canal. At the temporal side the pigment epithelium comes to an end at *d*, some distance from the edge of the chorioidal opening. At *d* the layer of rods and cones of the retina also ends. The space from *d* to *b*, the border of the sclero-chorioidal canal, appears with the ophthalmoscope under the form of a crescent, in which is recognizable the structure of the chorioidal stroma. There is a shallow physiological excavation, *E*.

Such papillæ are sometimes found in perfectly normal eyes, but more frequently in eyes affected with marked hypermetropia, astigmatism, or congenital amblyopia. They are often found in conjunction with irregularity in the shape of the papilla, an abnormal division of the vessels, or a crescent at the lower border of the papilla, from which it follows that they are to be regarded as an abnormal formation of congenital origin. Since such cases are usually regarded by beginners as instances of neuritis, they have received the name of *pseudoneuritis*. [Still more likely to cause confusion with neuritis are the rare cases in which either from congenital or acquired anomalies there is excessive tortuosity of the retinal arteries and veins (see § 515).—D.] Another diagnostic mistake that is frequently made is to diagnosticate neuritis when the papilla, owing to the presence of astigmatism, appears hazy in the erect image.

90. The optic disk under normal conditions lies in the plane of the retina, and does not, therefore, form a projection upon it as the name papilla would lead one to suppose. On the contrary, it very frequently contains in its center a depression, which is produced by the fibers of the optic nerve separating from each other comparatively early and thus leaving a funnel-shaped space between them (*vascular funnel*, Figs. 21 and 24). The central vessels ascend on the inner wall of the funnel. The color of the vascular funnel seems white to us because we see the white lamina cribrosa at its bottom. Often, instead of a small funnel-shaped depression, a large excavation (*physiological excavation*, Fig. 22, *E*) is present. This is situated in the outer half of the papilla, to whose external border it often reaches. The blood-vessels come out upon the inner border of the excavation (Fig. 20), and at the bottom of the latter are seen grayish dots, the lacunæ of the lamina cribrosa. With the brilliant white of the excavated exterior half of the papilla the grayish-red hue of the unexcavated interior half is in vivid contrast. Sometimes the physiological excavation is so large that it takes in the larger part of the papilla, but, in contrast with the pathological excavation, it never takes in the whole. There is always a part (though it may be a small part) of the papilla that is unexcavated, this being, as a rule, on the nasal side, while on the temporal side the excavation does often extend to the border of the papilla, though it never has a sheer wall there like the pressure excavation (cf. § 439).

91. Vessels of Fundus.—The central vessels of the optic nerve divide at the head of the nerve into a number of larger and smaller branches, which pass over its edge into the retina, where they keep on branching in an arborescent fashion. They can readily be distinguished into arteries and veins. The former are of a brighter red, narrower, and run a straighter course (Fig. 20, *a a*); the latter are darker, of greater caliber, and more crooked (*v v*). The arrangement of the vessels in the retina is not always the same; most frequently it happens that two main branches run upward and two downward, while only small and short twigs pass to the outer and inner sides of the disk (Fig. 20). The region of the macula lutea is devoid of larger vessels; the larger trunks, running to the outside above and below, encircle it and send finer branches in toward it. [See Fig. 246.]

In the larger vessels we notice a shining white streak running along the center of the vessels. This streak, which is more distinctly visible in the arteries than in the veins, is called the *reflex streak* (Jäger).

92. A *pulsation* is frequently observed in the vessels at the spot where they first come to view upon the papilla. The way in which this displays itself is that that portion of the vessel which lies closest to the point where the vessels come out becomes alternately empty, so as to disappear from view entirely, and then full again. This is not, therefore, an instance of the ordinary pulse, in which there are simply variations in caliber, but a completely intermittent inflow of blood (in the case of the arteries) or an intermittent outflow (in the case of veins).

A venous pulse is a physiological occurrence: in the same eye it is sometimes present, sometimes absent. In the latter case, slight pressure upon the eye with the finger suffices to produce it.

An *arterial* pulse is present under pathological conditions only. In order to produce it artificially in a healthy eye, no inconsiderable pressure must be exerted upon the eyeball. When this is done, the person under examination notices a simultaneous obscuration of the field of vision, amounting finally to complete abrogation of sight, as a result of the obstruction to the retinal circulation produced by the pressure. In like manner a rise in pressure produced by pathological conditions (glaucoma) causes an arterial pulsation. The explanation of this is as follows: As a result of the increased pressure in the interior of the eye, the blood is able to enter the vessels of the retina only during the systole of the heart; during the diastole, when the pressure in the arteries falls somewhat, the pressure in the central artery of the optic nerve is no higher than the pressure in the interior of the eye, and hence the blood-wave cannot make its way into the latter. Such a disproportion between the intra-ocular pressure and the pressure of the blood in the central artery may also, of course, be produced by the fact that while the former remains normal, the latter is diminished. Accordingly, an arterial pulsation is observed in general anæmia and when syncope is imminent; also in local compression of the central artery within the optic nerve (e.g., in optic neuritis).

A *true arterial pulse* (i.e., one not due to a completely intermittent

EXPLANATION OF FIG. 24.—LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE AS SHOWN IN FIG. 21. Magnified 60×1.

In its passage through the sclero-chorioidal canal, the optic nerve shows an irregular conical constriction. The fibers of the optic nerve are gathered into bundles separated by septa, *s*. The continuation of the septa can be followed as far as the head of the optic nerve under the form of rows of nuclei belonging to the cells of the neuroglia. The axis of the optic nerve is occupied by the central vein, *v*, and the central artery, *a*, which lies to the nasal side of the vein. The optic nerve is crossed at right angles by the lamina cribrosa, which separates the trunk from the head of the nerve. The fibers of the lamina cribrosa arise from the wall of the scleral canal, pass through the optic nerve, curving slightly backward as they do so, and are inserted into the connective tissue that accompanies the central vessels. About at the level of the inner layers of the chorioid, the nerve fibers separate like a sheaf so as to form a funnel-shaped depression (the vascular funnel, *G*). More fibers go to the nasal side of the papilla than to the temporal, for which reason, the nasal side is higher. The fibers of the optic nerve pass over into the fiber layer of the retina (*1*), succeeding which toward the outside are the other layers of the retina, namely, the layer of ganglion cells (*2*), the inner granular or plexiform layer (*3*), the layer of inner granules or bipolar cells (*4*), the outer granular or plexiform layer (*5*), the layer of outer granules or bodies of the visual cells (*6*), the limitans externa (*7*), and the layer of rods and cones (*8*). The layers of the retina stop short at the head of the optic nerve, the outermost layer (*8*) extending the farthest. The innermost fibers of the sclera which form the wall of the scleral canal, accompany the optic nerve, backwards, under the form of the pial sheath, *P*, which is intimately attached to the nerve. The outer layers of the sclera are reflected backward at a greater distance from the nerve head and from the dural sheath, *D*, which envelops the nerve loosely. Between these two sheaths, lies a third, the thin arachnoid sheath *A*, which divides the intervaginal space of the optic nerve into the subdural space, *sd*, and the subarachnoid space, *sa*. Both spaces have a blind ending in the substance of the sclera. *b* is the oblique section of one of the numerous subarachnoid trabeculæ which connect the arachnoid with the pial sheath. In the wall of the scleral canal can be seen the cross section of some blood-vessels which belong to the circle of scleral vessels of Zinn (see § 315). Between the sclera, *S*, and the retina, *R*, lies the chorioid, *Ch*. The innermost layer of the latter, the lamina vitrea, *10*, extends further than the other layers in the direction of the optic nerve-head, the fibers of which are constricted by the margin of the lamina vitrea. Upon the lamina vitrea lies the pigment epithelium, *9*, which belongs to the retina and which at the nasal side reaches as far as the lamina vitrea, but on the temporal side stops somewhat short of it. On both nasal and temporal sides the pigment epithelium gets to be thicker and more strongly pigmented at its margin, and this thickening and pigmentation correspond to the pigment ring which can be seen with the ophthalmoscope. The next layers of the chorioid, the chorio-capillaris, *11*, and the layer of medium size and large vessels, *12*, do not extend on the temporal side quite up to the optic nerve, a layer of connective tissue which represents a continuation of the sclera being interposed between the two. In surface view this layer is visible with the ophthalmoscope, under the form of a white ring, the scleral ring. In this case it appears, with the ophthalmoscope, somewhat broader than it really is, because the layer of connective tissue which corresponds to it does not run forward in a precisely sagittal direction, but turns somewhat

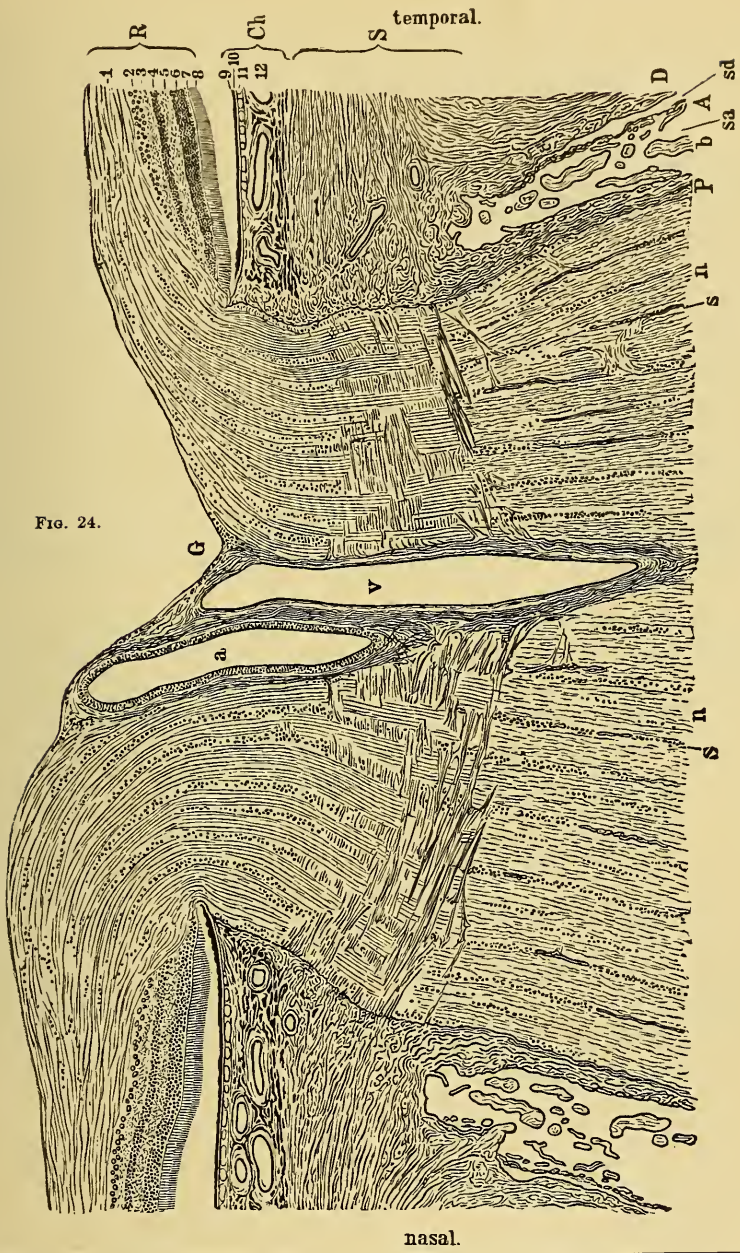


Fig. 24.

to the temporal side. Hence we see besides the front surface of the connective tissue ring its inner wall in perspective foreshortening lying close to the nerve-head (this because the substance of the nerve-head itself is transparent). On the nasal side the optic nerve keeps on contracting conically even within the chorioidal canal, because the inner layers of the chorioid come up closer to the optic nerve than do the outer. The connective-tissue ring which is interposed between optic nerve and chorioid, is narrow here, does not extend forward, and is covered by the pigmented chorioid and the pigment epithelium, for which reason no scleral ring can be seen with the ophthalmoscope on the nasal side.

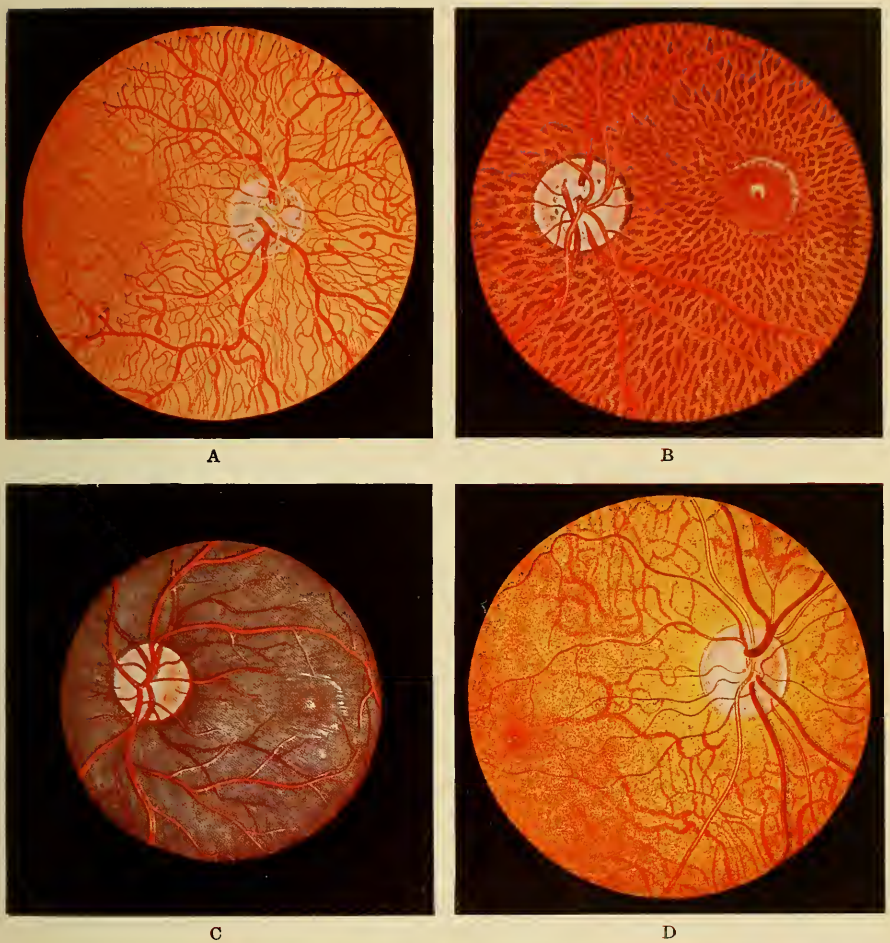
inflow, but to a translation of the variations of the pulse into the retinal vessels) occurs as one of the symptoms of an abnormally extensive diffusion of the pulse-wave all through the body, e.g., in insufficiency of the aortic valves or in Basedow's disease.

Donders gives the following explanation of the venous pulse: At each systole of the heart an additional quantity of blood is driven into the arteries of the interior of the eye, and the blood pressure in these arteries is consequently heightened. This increase in the arterial tension reacts at once upon the general intra-ocular tension, heightening the latter, so that it acts more vigorously upon the retinal veins and compresses them. It does this most at the spot where the blood pressure in the veins is lowest, which is at their point of emergence upon the papilla, since the blood pressure in the veins diminishes in proportion as we approach the heart and get farther away from the capillaries. The veins consequently are constricted at the spot where they dip down into the vascular funnel, while the portion of the veins directly adjacent swells up, because the blood is dammed up in it. But as a result of this damming, the blood pressure in the veins rises rapidly to the point where it is able to overcome the compression—doing this the more readily as now the diastole of the heart sets in, and with this the intra-ocular pressure diminishes. [As above stated, moderate digital pressure on the eyeball causes pulsation of the retinal veins. With greater degrees of pressure the retinal arteries narrow and the veins are emptied. This effect is most marked in children; it is less in elderly subjects; and in old persons with arteriosclerosis digital pressure may not produce even a venous pulsation (Black).—D.]

93. Appearance of Fundus.—Since in healthy living eyes the *retina* is transparent, we see no part of it with the ophthalmoscope except the blood vessels. At most we find the red fundus in the immediate neighborhood of the papilla covered by a delicate gray veil which shows a radiating fine striation and which is the representative of the layer of nerve fibers of the retina, which in this situation is still quite pronounced. In children vivid reflexes often exist which are especially pronounced along the vessels, change their place with every movement of the mirror, and give the retina a luster like that of watered silk. We must not regard these as pathological opacities. [In old people, on the other hand, we find the retina less transparent and lustrous, the vessels narrower, and the papilla paler (Leber).—D.]

The region of the retina that is most important for vision, the *macula lutea*, with the fovea centralis, is just the part that has very few distinctive ophthalmoscopic features. We find it with the ophthalmoscope if we go a distance of 1½ to 2 papilla-breadths outward from the outer border of the papilla. Here we come upon a region devoid of vessels which is somewhat darker than the rest of the fundus. Directly in its center, corresponding to the situation of the fovea centralis, we see, when examining the eye with the direct method, a bright point or a small, crescentic spot (Fig. 20, *f*). In the inverted image the macula lutea is represented by a fine white, not very sharply-defined curved line which forms a horizontal oval of about the size of the papilla. The region inclosed by the line is colored a dark brownish red and sometimes has in its center a little bright dot. These appearances are nothing more than reflexes produced by the light on the

PLATE I.



[Fig. 25.—Varieties of the Normal Fundus. (After Würdemann in Posey and Spiller.)

A, albinotic fundus; albino and light blonde (after Greef, modified by Würdemann). B, the tessellated fundus; brunette (after Greef, modified by Würdemann). C, the negroid fundus; negro (Würdemann). D, the yellow fundus; Chinese (after Oeller, modified by Würdemann).—D.]

inner surface of the retina, and are by no means constantly present. When the pupil is dilated they become less marked or disappear altogether.

94. Since the purple color of the living retina is not visible with the ophthalmoscope, the *color* of the background on which the appearances above described are to be seen is produced by the structures lying behind the retina, namely, the pigment epithelium, the chorioid, and the sclera. The following varieties of coloration in the background of the eye are found, depending on the amount of pigment contained in the two structures first named:—

1. If the pigment epithelium is very uniformly and profusely pigmented, it conceals the chorioid completely, and the background of the eye appears of a uniform brownish red (in very darkly pigmented men almost a dark gray), [see Fig. 25, C.—D.]. When the pigmentation is less uniform we observe in the erect image a finely granular appearance of the eye-ground which is caused by the cells of the pigment epithelium.

2. When the pigmentation of the pigment epithelium is less marked the chorioid shows through it and if this latter is profusely pigmented we can recognize the interspaces between the chorioidal vessels (the so-called intervascular spaces, Fig. 150, H) as dark elongated islands. The light red striæ running between the latter and anastomosing everywhere with each other correspond to the chorioidal vessels, which are chiefly veins. No sharp outlines of vessels, however, are visible, because the pigment epithelium spreads a veil over the chorioid. Such an eye-ground is said to be *tessellated* (Figs. 25, B, and 26); by beginners it is often confounded with chorioiditis.

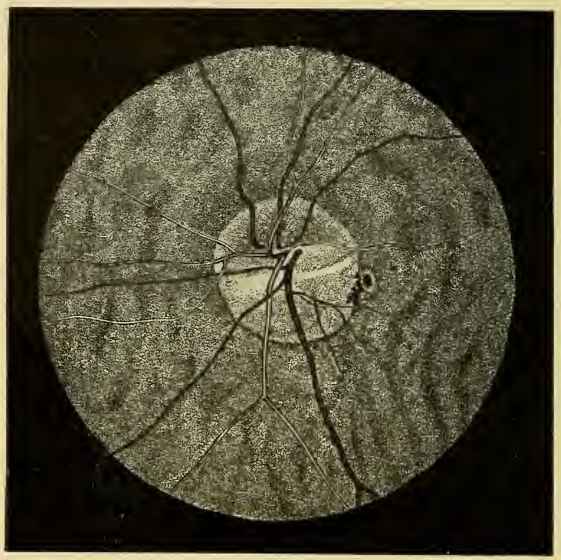
3. The less pigment there is contained in the pigment epithelium and in the chorioid the more the white sclera shows through, and consequently the lighter red is the eye-ground as a whole. In individuals with little pigment —i.e., in blondes and to the greatest degree in albinos—the pigmentation in the fundus is so scanty that we can see the vascular network of the chorioid distinctly. In contrast with the tessellated fundus, the intervascular spaces are lighter than the vessels, because the white sclera shows through. Such a fundus is called *albinotic* (Fig. 25, A, and Fig. 27; see also §437). The retinal vessels run over the chorioidal vessels, but are easy to distinguish from them. The chorioidal vessels are broader, less sharply defined, and look flat and ribbon-like; they lack the reflex streak. In opposition to the retinal vessels, which branch after the manner of a tree and do not anastomose, they form by their numerous anastomoses, a dense network with elongated meshes.

95. Determination of the Refraction by the Ophthalmoscope.[5]—This may be accomplished in three ways: with the erect image, with the inverted image, and by the shadow test.

(1) Determination of the refraction with the *erect image*. When the eye under examination is *emmetropic*, the rays emitted from the illuminated retina emerge parallel to each other (as shown in Fig. 13); hence the obser-

[5] The following remarks with regard to the determination of the refraction presuppose for their understanding an acquaintance with Part IV of this book, which treats of the refraction of the eye.

Nasal



Temporal

FIG. 26.—TESSELLATED FUNDUS.

(For the alteration of the papilla here depicted, which is due to congenital malformation, see the description attached to Fig. 202.)

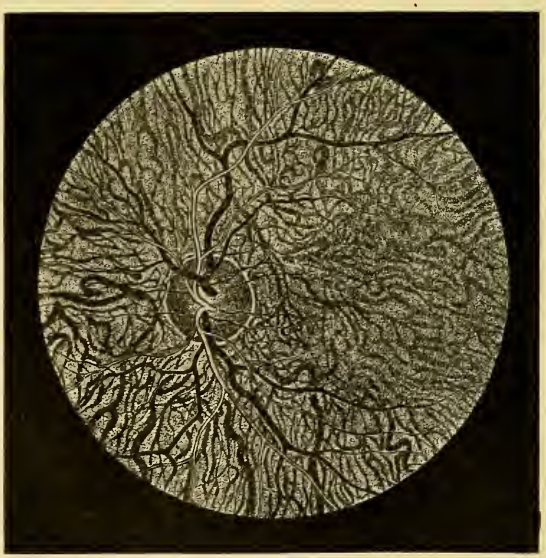


FIG. 27.—FUNDUS OF AN ALBINOTIC LEFT EYE SEEN IN THE ERECT IMAGE. (After Jäger.)

The papilla is surrounded by a light-colored scleral ring, and looks dark in comparison with the light hue of the rest of the fundus. The latter shows a thick network of chorioidal vessels and over them the retinal vessels, which are distinguished from the former by their better defined outline, their narrow caliber, and their straight course. Both the chorioidal and retinal vessels contrast by their darker red with the very light red of the background, the hue of which is produced by the white sclera shining through the chorio-capillaris. It is only at the macula lutea that the somewhat darker hue of the fundus gives evidence of the presence of a slight pigmentation of the chorioid.

ver's eye, which in all that follows we shall assume to be emmetropic, can without exercising any accommodation unite them into a sharp image. Emmetropia, however, represents the only condition of the patient's eye in which an emmetropic observer can without ulterior aid see distinctly the patient's fundus; when the patient's refraction is of any other nature the observer, to see clearly, must use either a glass or his accommodation.

In the first place, suppose the eye under examination (*A*, Fig. 28) to be *myopic* with its far point at *F* so that the rays coming from *F* are united on the retina at *f* (see § 765). *F* and *f* are conjugate foci, and the course of the rays would therefore be the same if they should proceed in the opposite direction—i.e., from *f* toward *F*; in that case, they would be united at *F*, as they emerge from the eye. A point *f* of the retina, illuminated by the ophthalmoscope, will then emit a bundle of rays converging at *F*; and at this distance there is produced a clear image of the illuminated fundus. The observer's eye, which is placed at a short distance (a few centimetres) from the eye *A*, would catch the rays emitted from the latter before they were united at *F*—that is, while they still had a certain amount of convergence. But the observer's eye, unless it were hypermetropic, is not in

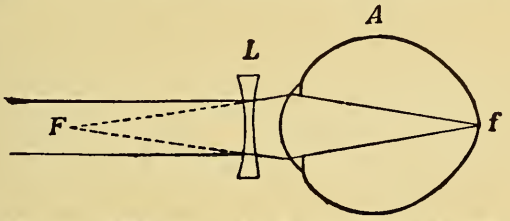


FIG. 28.—CORRECTION OF MYOPIA BY A CONCAVE LENS.
The eye is drawn of the natural size of a myopic eye having an axial length of 27 mm.

a position to unite convergent rays into a sharp image. If the eye is emmetropic, as we will assume it to be, the rays which fall upon it must first be made parallel, which evidently is accomplished by a concave lens, *L*, of the proper strength. This would be a lens of such focal length that its principal focus would coincide with the point *F*. Now, how is this lens related to the degree of myopia of the eye under investigation? If we imagine the course of the rays reversed, then parallel rays coming from in front and falling upon the lens *L* would be rendered by the latter so divergent that they would come to a focus upon the retina of the myopic eye; the myopic eye accordingly would get sharp vision with this lens for parallel rays—that is, rays coming from an infinite distance. *L* would therefore be the correcting glass for the myopia of the eye, *A*. We can hence say this: In order that an emmetropic observer should see clearly the fundus of the myopic eye, *A*, he must use the same glass that will correct the myopia of this eye. If, therefore, an emmetropic observer has to determine the refraction of a myopic eye with the ophthalmoscope, he keeps placing concave glasses before it until he finds one with which he gets a sharp view of the fundus in the erect image; the glass employed gives directly the degree of the myopia. The same principle holds good for *hypermetropic* eyes, only that convex instead of concave glasses are required. The rays emitted from the hypermetropic eye, *A* (Fig. 29), are divergent, and the more so the higher the hypermetropia is. The convex glass, *L*, which is required in a given case of hypermetropia in order to render parallel the divergent rays emerging from the eye and thus make it possible for the emmetropic observer to perceive the fundus, is the same as that which renders rays falling upon the eye in a parallel direction so convergent that they are united upon the retina, and is therefore the glass which corrects the hypermetropia. Hence the degree of hyperme-

tropia of the eye under examination is given immediately by the convex lens with which the emmetropic observer sees the fundus distinctly.

An emmetropic observer can also, it must be noted, cause divergent rays to focus upon his retina by calling his accommodation into play, and in this way can see the fundus of a hypermetropic eye distinctly even without the aid of a convex glass. But as one cannot estimate precisely the degree of accommodation thus applied, it is impossible to determine the amount of hypermetropia with precision by proceeding in this way.

How is it in those cases in which the physician himself is not emmetropic? In that case he must simply correct in addition his own ametropia. If, for instance, an emmetrope is examining an eye having a myopia of 2 D, he needs for this purpose − 2 D. If the observer himself should have a myopia of 3 D, he would have to take in addition − 3 D for himself; hence he would employ a glass of − 5 D. If the observer were a hypermetrope of 1 D, he would need + 1 D for the correction of his own ametropia; this, in combination with the − 2 D which are required for the eye under examination, gives a lens of − 1 D. A similar procedure must be adopted in those frequently occurring cases in which the observer is indeed emmetropic, but cannot completely relax his accommoda-

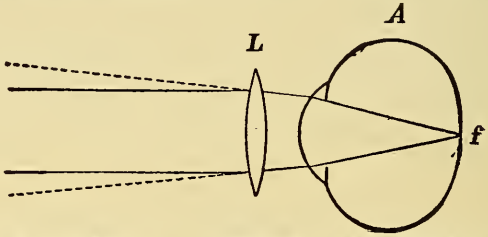


Fig. 29.—Correction of Hypermetropia by a Convex Lens.
The eye is drawn of the natural size of a hypermetropic eye having an axial length of 21 mm.

tion during the ophthalmoscopic examination. He is then to be regarded as a myope, inasmuch as he has to neutralize his residual accommodation by a corresponding concave glass.

[We have to do the same thing if the patient uses his accommodation. The fact that very few observers are able to relax their accommodation completely when examining the eyes with the ophthalmoscope and that we can never be sure that the patient himself is not using some accommodation, renders the determination of the refraction by the direct method pretty uncertain, and there are very few ophthalmologists who can measure the refraction in this way to within one D.

If we examine by the direct method an eye with a notable amount of *astigmatism* some details of the fundus, e.g., the vertical vessels, will come out distinctly with one glass while the vessels at right angles will be seen best with another—the difference in strength between the two glasses being approximately equivalent to the amount of the astigmatism. In this case we must recollect that the glass with which we see one set of vessels gives the refraction of the meridian at right angles to them. Thus, if we see the vertical vessels with a + 3 D, and the horizontal vessels with a plane glass, the vertical meridian is emmetropic and the horizontal meridian is hypermetropic 3 D.—D.]

(2) *The determination of the refraction with the aid of the inverted image* is done by the method proposed by Schmidt-Rimpler, the principle of which is as follows: The concave mirror, *S S* (Fig. 14), forms at its focus a sharp image of the flame that is used as the source of light in making the ophthalmoscopic examination. This image lies between the mirror and the convex lens (*l*). Rays emanating from it are by means of the lens thrown upon the retina of the patient's eye to form there a new image of the flame, which the observer sees upon the fundus. Whether this latter image is sharp or not depends upon various circumstances: upon the strength of the mirror and the lens; upon the distances between the lamp, the mirror, the lens, and the eye; and lastly upon the refrac-

tion of the latter. By taking all these factors into consideration we can determine the refraction, provided we ascertain the distance at which the observer has to be in order to see the image of the flame upon the fundus of the patient's eye distinctly. [Cf. § 99.]

96. (3) The determination of the refraction by means of the *shadow test* was discovered by Cuignet, who called it keratoscopy. It is also known as pupilloscopy, retinoscopy, and skiascopy (σκιά, shadow). In using it, the observer places himself at a distance of rather more than one metre from the patient, and throws light into his pupil by means of a concave mirror. When the mirror is in a certain position, the whole pupil appears a vivid red; then if the mirror is turned a little on its vertical axis, a black shadow appears at the edge of the pupil and, as the mirror is rotated still more, passes over the whole area of the pupil, until the latter is completely dark. From the direction in which the shadow travels the refractive state of the eye under examination can be ascertained. To accomplish this, we must, to start with, have a clear comprehension of the significance of the illuminated portion of the pupil and of the shadow respectively.

By means of the mirror a point of the fundus is illuminated, and from this point the rays are returned in such a way that a portion of them pass out again through the pupil. The direction which these rays take on emerging is determined by the refraction of the eye. If *myopia* is present, we know that the emergent rays will converge so as to meet at the far point of the eye. In Fig. 30 let $J\ P$ and $P_1 J_1$ be the iris, and $P\ P_1$ the pupil of the patient's eye. The rays emerging from the pupil unite at the far point R of the eye. If we suppose that by means of the mirror a point of the retina is illuminated which lies somewhat to the right of the line connecting the pupillary centers of the observer's and of the patient's eyes, R will be situated correspondingly far to the left of this line. From R the rays (now become divergent) continue on their way toward the observer's eye, which we will now suppose to be beyond R. Let $i\ p$ and $p\ i$ represent the iris, and $p\ p$ the pupil of this eye. Now $p\ p$ does not take in all of the conical sheaf of rays emanating from R, but only a portion of it, having $p\ p$ as its base. The remainder of the cone falls upon the iris $p\ i$. Since the rays constituting this part of the cone are not seen by the observer, the portion of the pupil which is opposite to them, and from which they come (represented in Fig. 30 by the lines of shading), appears unilluminated; the only portion of the pupil that does appear illuminated being that which is here shown as unshaded, and from which the observer receives rays that enter his own pupil. The dark and the luminous portions of the patient's pupil are separated by a curved line, since the boundary between the two is formed by the pupillary edge p of the observer's eye. Thus the circle at the bottom of Fig. 30 represents the pupil of the patient's eye seen from in front; the portion of it left unshaded in the figure corresponds to the illuminated part of the pupil.

Now suppose that by a rotation of the mirror the spot of illumination in the fundus shifts in such a way that R travels farther to the left. Then more and more of the emergent beam will fall upon the iris, and less and less of it will fall upon the pupil of the observer's eye, and the shadow in the pupil of the patient's eye will, as the arrow in the circle indicates, advance farther and farther toward the left pupillary margin, until finally the whole pupil appears dark. The shadow, therefore, moves in the same direction that R does.

We have now to determine how the movements of R are related to the movements of the mirror. If a concave mirror is employed, it forms at its focus an image of the lamp flame which lies between the mirror and the patient's eye and serves to illuminate the

latter. If the mirror is rotated to the left, the image of the flame also travels to the left. But as the portion of retina illuminated by the image of the flame must always lie on the side diametrically opposite to the latter, it must, with the movements of the mirror, move in a sense opposed to that of the image of the flame—i.e., to the right (from B to B_1 in Fig. 30). But the point of union, R, of the emergent rays lies diametrically opposite to that occupied by the illuminated portion of the retina; hence R will move to the left—i.e., in the same direction as the mirror. Now since R moves to the left when the mirror is rotated to the left, and as the shadow in the pupil travels to the left when R moves to the left, we may say,

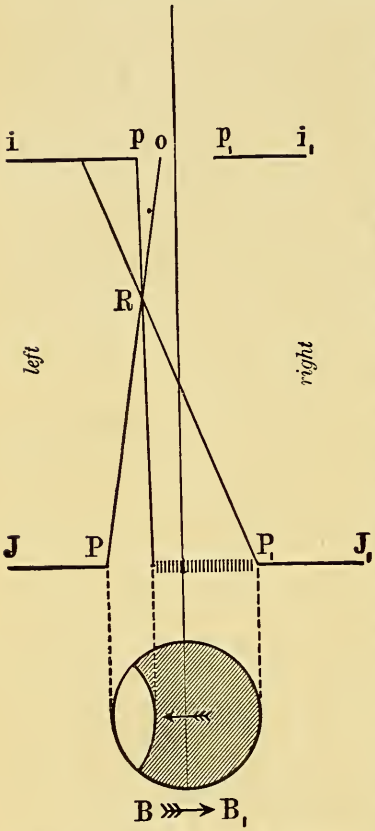


Fig. 30.—Shadow Test in Myopia.

When a concave mirror is used the shadow in the patient's pupil moves in the same sense as that in which the mirror rotates, provided the far point of the patient's eye lies between his eye and the observer's.

These relations are reversed when we come to examine a myopic eye whose far point is beyond the observer's eye. By constructing Fig. 30 so that R lies beyond $i\ p$, it will be found that in this case the illuminated portion of the pupil lies on the right side, and that as R shifts to the left the shadow goes to the right. The like is true for those cases in which the far point of the patient's eye lies behind the latter, as is the case in *hypermetropia*. This will be clear from Fig. 31. $P\ P_1$ represents the pupil of a hypermetropic eye, from which the rays that emanate from the retina pass out, taking a divergent course. They thus form a cone whose apex lies behind the eye at its far point, R. If the illuminated portion of the retina lies to the right of the line connecting the pupillary centers of the two eyes, R is also found to the right of the latter. The pupil, $p\ p_1$ of the observer's eye takes in only that part of the conical sheaf of rays which corresponds to the right-hand portion of the patient's pupil (the portion left unshaded in Fig. 31). The left-hand portion of the patient's pupil (represented by the lines of shading in the figure) remains unilluminated, because the rays that come out from it no longer fall upon the observer's pupil. The more R moves to the right, the more the unilluminated portion of the pupil is displaced to the right in the direction indicated by the arrow in the circle below. The shadow, therefore, travels in the same direction that R does, as is also the case in myopia when the far point lies in front of the observer's eye (Fig. 30). The difference between the two cases lies in the different relation that the movement of R has to the rotation of the mirror. If the concave mirror is rotated to the left, the image of the flame produced by it travels likewise to the left, and the spot of illumination upon the retina travels to the right (from B to B_1). Then the beam of rays returning from this spot of illumination shifts to the left, but R, since in the hypermetropic eye it lies at the prolongation of these rays backward, moves to the right. R, therefore, moves in a direction opposite to that in which the mirror rotates and the same is true of the pupillary shadow, which always moves in the same way that R does.

Hence when a concave mirror is used, the shadow moves in a direction opposite to that in which the mirror is rotated, provided the far point of the patient's eye lies behind the observer's eye(in low degrees of myopia) or behind the patient's eye (in hypermetropia).

The way in which the shadow moves, therefore, depends upon the relative situation of the far point and the observer's eye. If the observer stations himself at a distance of rather more than 1 m. (say 120 cm.) from the patient's eye, *R* lies between the two eyes when there is myopia of 1 D or more, because then the patient's far point lies at 1 metre or less from his eye. In myopia amounting to less than 1 D the far point lies behind the observer's eye, and the same is true of emmetropia, in which the far point lies at infinite distance. In hypermetropia, on the other hand, the far point lies behind the patient's eye. From these facts are derived the following rules for conducting the shadow test:

The observer, standing at rather more than 1 metre from the patient, illuminates the eye with a concave mirror, and notices the way in which the shadow moves in the patient's pupil as the mirror is rotated. If the shadow moves in the same direction that the mirror rotates, there is myopia of 1 D or more. Then successively stronger concave glasses are set before the patient's eye in a trial frame[6] until a glass is found with which the shadow travels in the reverse direction. This glass carries the far point of the eye to just beyond 1 metre (corresponding to a refractive power of 1 D); and the last glass, *n* D., with which the shadow still moves in the same sense as the mirror, corrects the myopia of the patient's eye up to approximately 1 D. Hence the total myopia of this eye is *n* D + 1 D.

If, when the mirror is rotated, a movement of the shadow takes place in the opposite direction, there is then in the eye that is being examined either myopia less than 1 D, or emmetropia or hypermetropia. In this case a series of convex glasses is placed before the patient's eye until the shadow begins to move in the same direction as the mirror. If this glass is *n* D, the refraction of the patient's eye is *n* D − 1 D.

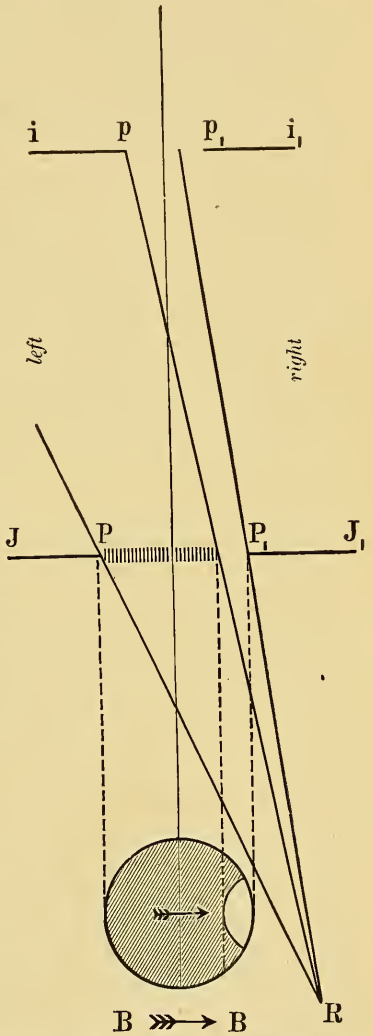


Fig. 31.—Shadow Test in Hypermetropia.

[If we give the name *reversing* glass to the lens which in any case just suffices to give us a movement with the mirror (any lens that is more concave or less convex giving us a movement against), we may enunciate the above rules more simply as follows: If we add − 1 D to the reversing glass we shall get the glass that corrects the patient's refraction.

[6] [Instead of placing a number of glasses one after another in a trial frame, we may also employ a revolving disk, a set of lorgnette frames, or an oblong slide in which a series of such glasses is permanently fixed after the fashion of an ophthalmoscope. Such an appliance, which is called a *skiascope*, allows the glasses before the patient's eye to be shifted rapidly either by the patient himself or by the physician, without the latter having to move from his station.—D.]

The observer may stand at any distance from the patient other than a metre. For example, he may stand at two metres. In that case, since the reversing glass obviously puts the patient's far point just in front of the observer's eye, the patient with the reversing glass on will be myopic 0.5 D and to find the true correcting glass we shall have to add − 0.5 D instead of − 1 D to the reversing glass. So if we stand at half a metre we shall have to add − 2 D to the reversing glass to get the proper correction.—D.]

97. [We determine *astigmatism* with the shadow test by ascertaining the refraction of the two principal meridians separately. For example if with the concave mirror at one metre we obtain reversal in the vertical meridian with +2.5 D and in the horizontal meridian with +4.5 D there is an astigmatism of 2 D and the correcting glass is + 1.50 D sph. ⊂ + 2.00 cyl. ax. 90°.

A more accurate way of determining the astigmatism by the shadow test is to ascertain the refraction of one of the principal meridians with a spherical glass; then leaving this glass in situ, to place a cylindrical glass with its axis in the meridian thus corrected and keep on changing the cylinder until with the two glasses, spherical and cylindrical, a complete and uniform reversal is obtained in all meridians at once. This method has the advantage of indicating both the axis and the amount of astigmatism with great precision.

Regular astigmatism of any amount is shown in the shadow test by the presence of a band of light which always runs in the direction of one of the two principal meridians. This appears as follows: When a glass is put on which is about equal to the reversing glass for either one of the principal meridians, the illuminated area of the pupil instead of appearing round is transformed into an elongated oval or into a band of light which runs in the direction of the meridian thus corrected. Then, no matter how the mirror is moved, the movements of the shadow tend to take place at right angles to this band. This band of light is an important indication since it runs in the precise direction in which the axis of the correcting cylinder is to be placed.

The presence of irregular astigmatism is shown in the shadow test by confused shadows which move irregularly and in various directions across the pupil; often by the presence of two shadows which come simultaneously from opposite sides of the pupil to meet each other (scissors movement).—D.]

98. The shadow test can also be conducted with the *plane mirror*. With this the image of the flame lies behind the mirror, and hence, when the latter is rotated, moves, not with the mirror, as is the case when this is concave, but in the opposite direction. Accordingly, the movement of the pupillary shadow with relation to the rotation of the mirror is just the reverse of that which obtains with the concave mirror.[7]

In either case this method is of great simplicity; of all methods it is the easiest to learn, and has the advantage that in it the refraction and accommodation of the observer do not need to be considered. Withal it gives as exact results as any one of the other methods. [The shadow test is one of the most accurate of our methods of determining objectively the refraction, and of checking the results of the subjective examination, and should be carefully studied and practiced. It usually requires dilatation of the pupil and relaxation of the accommodation with homatropine, although often quite accurate determinations can be made without this. If the pupil is dilated we must try to observe the movement of the shadow in the area which corresponds to that occupied by the pupil when normally contracted. This *visual area*, or central

[7] [I.e., the shadow moves in the opposite direction to that in which the mirror is rotated—moves to the right when the mirror is rotated to the left, or, as the phrase is, moves "against" the mirror in myopia of such a degree that the patient's far point is between him and the observer; and in all other cases—in less degrees of myopia, in emmetropia and in hypermetropia—moves "with" the mirror.—D.]

portion of the pupil, which the patient habitually uses for seeing, sometimes differs markedly in refraction from the peripheral area of the pupil. Unless attention is paid to this point and the movement of the shadow in the visual area is taken instead of that in the periphery, a false estimate of the refraction may be made.—D.]

99. *Determination of Refraction by Direct Illumination.*—A superficial estimate of the refraction of an eye can be got whenever an observer at some distance [using direct illumination] is able to see portions of the fundus.

This is the case both in marked myopia and in marked hypermetropia. In marked myopia the rays emanating from the eye come together in front of and very close to it at its focus *F* (Fig. 28), where, accordingly, an inverted image of the fundus is produced, and that without the aid of a convex glass. The observer can see this image if he places himself at a suitable distance—e.g., the ordinary reading distance. That it is an inverted image which he sees is obvious from the fact that it moves to the right when he moves his head and mirror to the left, and vice versa.[8] If the observer approaches the patient's eye, the image of the fundus rapidly becomes indistinct and soon disappears altogether, because the observer then gets so close to it that he can no longer accommodate for it.

In marked hypermetropia, too, the image of the fundus can be seen at some distance from the eye, but in this case it is an erect image. It moves in the same direction that the observer moves, and remains distinct when the latter approaches the patient's eye. We can thus determine whether we are dealing with a high degree of myopia or of hypermetropia.

100. *Apparent Deformation of Disk in Ametropia.*—The presence of astigmatism may be recognized from the change of shape which the papilla undergoes.

In regular astigmatism one meridian of the dioptric system of the eye is more refractive than the one at right angles to it; may be said, in fact, to correspond to a stronger convex lens than does the latter. Hence with the erect image the papilla will be seen under a greater enlargement in the more refractive meridian. If the latter, as is generally the case, is vertical, a round papilla will appear like a vertical oval. But the papilla often has in reality an oval form; and, in order to distinguish whether we are dealing with a papilla that is anatomically oval or with an astigmatic distortion of a round papilla, we must resort to a comparison with the inverted image. If the papilla is really a vertical oval, it must appear so with the inverted image also. If, however, there is astigmatism, the distortion due to it in the inverted image will be the opposite to that produced in the erect image—i.e., in the example given the papilla in the inverted image would appear transversely oval. (This, however, is only the case when the convex lens used for forming the inverted image is held close to the patient's eye. If the lens is gradually carried off, the papilla appears first round, and at length vertically oval.)[9]

[8] [This is not so much an evidence of the image's being inverted as of its being in front of the eye. The image of the fundus, in fact, appears to move to the right when the observer moves his head to the left, because it lies in front of the plane of the pupil to which its movements are referred. On the other hand, in hypermetropia the image of the fundus appears to move in the same way that the observer does, because the image then lies behind the plane of the pupil. The conditions are the same as when we are on a moving train, and looking out at the telegraph poles near the track, backed by trees in the distance. The trees appear to move in the same direction as the train, and the telegraph poles in the opposite direction, the movements of both being projected upon some plane intermediate between the two.—D.]

[9] [The fact that the papilla appears to change its *shape* as the convex lens is withdrawn from the patient's eye is of itself a proof of astigmatism. On the other hand, an apparent change in size of the papilla, without change of shape occurring when the convex lens is gradually withdrawn, points to simple hypermetropia or myopia—to the former if the disk appears to diminish, and to the latter if the disk appears to enlarge. If the convex lens is placed so that its focal point is a little farther from the eye than the anterior focus of the latter (i.e., is rather more than half an inch in front of the cornea), there will be no distortion of the image of the disk from astigmatism, and no apparent increase or diminution in size due to myopia or hypermetropia. As this is the position of the lens which gives an undistorted view of the fundus, it is the one that should habitually be used in making examinations by the indirect method—i.e., a 2-inch lens should be held 2½ inches and a 3-inch lens 3½ inches in front of the cornea.—D.]

101. Determination of Differences of Level in the Fundus.—Differences of level cannot only be appreciated, but also precisely measured by means of the ophthalmoscope. This is effected with the aid of the erect image, by means of which the refraction can be determined for every point separately of the visible fundus. If a point in the fundus projects above its surroundings, as, for instance, the swollen papilla in neuritis, the axis of the eye corresponding to this point is shorter—that is, there is a hypermetropia. By determining the degree of the hypermetropia we can compute the height of the prominence. Conversely, a point of the fundus which lies farther back (for example, the bottom of an excavation) possesses a myopic refraction, from which the linear measure of the amount of depression can be found. As the basis of this computation the rule holds that a difference of level of about 1 mm. corresponds to a difference of refraction of 3 D.

Differences of level of the fundus are also made apparent by *parallactic displacement*. This is the displacement that under ordinary conditions of vision all objects show which do not lie in a plane parallel to the frontal plane. With the erect image we appreciate this parallactic displacement if during the examination we move a little from side to side. In making the examination with the inverted image we move the

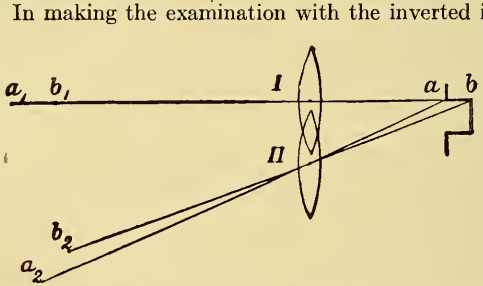


Fig. 32.—Parallactic Displacement of the Inverted Image of Points of the Fundus, Lying at Different Levels.

convex lens which serves for the production of the inverted image a little up and down during the examination. If the points of the fundus which we have fixed upon lie all in the same plane, they do not change their relative position to each other with the shifting of the convex lens. If, on the contrary, a difference of level exists between them, we notice a displacement with relation to each other, so that they now come nearer together, now go farther apart. Figure 32 may elucidate what takes place. Let a be a point on the edge, b a point behind it on the bottom of an excavation of the optic nerve. If the convex lens stands at I, the images of the two points a_1 and b_1 fall behind each other and are superimposed. If the convex lens is now brought to II, the image of the point a is reproduced at a_2, that of b at b_2; the points appear to have separated from each other. Had the convex lens been carried in the opposite direction, the apparent displacement of the two points would have taken place in the opposite sense; it would have looked as if the edge of the excavation had been drawn over the bottom of it. From the magnitude of the displacement the difference of level of the two points can be estimated, but cannot be exactly computed, as is possible with the aid of the erect image.

CHAPTER II

FUNCTIONAL TESTING

102. Besides instituting the objective examination, we have further to test the function of the eye. In doing this we are limited almost entirely to the statements of the patient, so that in this respect we are quite dependent upon the latter's intelligence and good will.

Our visual sensations are of three different kinds, inasmuch as in looking at objects we take cognizance of their form, their color, and their brightness. The faculty by which we recognize the form of objects is called the space sense, and finds its numerical expression in the visual acuity; the faculty by which we distinguish colors constitutes the color sense; the faculty by which we distinguish different degrees of brightness constitutes the light sense. These faculties are resident in the retina throughout its entire extent, although in very different degrees. In this regard we must distinguish between central and peripheral vision.

103. *Central* or direct vision is vision with the fovea centralis. When we wish to see an object distinctly, we "fix" [or "fixate" or "sight"] it—that is, we turn the eye in such a way as to make the image of the object fall upon the fovea centralis, as the latter, on account of its peculiar anatomical structure, gives us the sharpest vision that we are capable of. It is with reference to central vision that we test the refraction, the accommodation, and the visual acuity. For more precise particulars in regard to these tests, see Part IV of this book, which treats of the optical defects of the eye.

104. *Peripheral* or indirect vision is vision with those parts of the retina which do not belong to the fovea centralis and which comprise by far the greatest part of the retina. Vision with the peripheral portion of the retina affords a less distinct,[1] a duller sensation, of which we can best get an idea by holding the outspread fingers of our hand to one side of the eye, while the latter is looking straight ahead. The farther from the fovea centralis is the image produced upon the retina, the more indistinct is the perception of its shape. For perceptions of movement, on the other hand (Exner), as well as of slight differences of luminosity, the periphery of the retina is actually more sensitive than the center.

Of what use, then, is peripheral vision, if we can get no well-defined perceptions with it? We can best understand this by observing persons who have lost peripheral vision to such an extent that only the fovea centralis and its immediate vicinity retain their functional activity, as happens

[1] [According to Burckhardt, the visual acuity of an excentric portion of the retina is 1 divided by $3n$, where n=the distance in degrees from the fovea (Leber).—D.]

in many diseases, especially in retinitis pigmentosa. Such persons can sometimes still read the finest print, and yet are in no condition to go about alone. We can put ourselves in this condition if we fasten in front of the eyes a long tube which allows us to see only the point lying directly in front of our line of vision. We cannot go about with such an apparatus because we strike against objects everywhere. Peripheral vision, therefore, is of service in orientation. How? If, as we are walking, we look straight before us and there is a stone lying in our path, the latter forms an image in the periphery of the retina of our eye, in this case in the upper part of it. The stone, to be sure, is not distinctly perceived, but still it excites our attention. Our gaze is then directed at it; it is seen directly; we recognize it as an obstacle and avoid it. The same thing happens if we go out upon the street and men come toward us from one side, etc. The images falling upon the periphery of the retina give us warning signals which make us cast our eye directly upon the objects which produce the images. And it is precisely moving objects that are most sure to attract our attention, since, as just stated, the peripheral portions of the retina have a high degree of sensibility for the perception of movement.

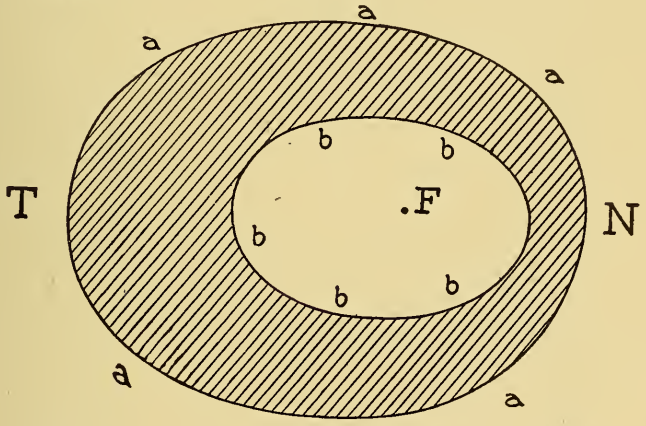
105. Examination of the Field of Vision.—The examination of the field of vision—that is, of the limits of indirect vision—must be made for each eye separately. The eye examined is directed at a fixed point, in order that it may thus remain steadily in the same position, while the other eye is kept closed.

The simplest way of investigating the extent of the field of vision is by using the *hand* as a test object. The physician places himself directly in front of the patient and at a short distance from him; the patient looks with one eye at the physician's eye directly opposite. The physician now closes his other eye (as does the patient), and gradually moves his hand from the periphery inward over the limits of the field of view; the patient must tell as soon as he sees the hand. In this way the physician has in his own eye a means of judging the field of view of the patient; if this is normal, the patient must see the hand at the same time that the physician does with his eye. This method is sufficiently exact for the recognition of the larger encroachments upon the visual field; but small defects cannot be thus recognized. It is the only method of testing applicable in those cases in which smaller test objects are no longer made out because the vision is too poor.

[If instead of the hand a small test object be used, this method, which may be called the *confrontation method*, is quite precise and is also as accurate for clinical purposes as the more seemingly scientific methods described later. A suitable test object for this purpose is a white card with a round black spot 1 to 3 mm. in diameter on each side of it, or for color tests a small colored square on each side of a gray card, the gray having the same light value as the color that it bears (Holden). The observer stations

himself opposite the patient and in such a way that the background and the illumination are alike for both. The patient closes his left eye and with his right looks straight into the observer's left eye. The observer closes his own right eye, looks with his left straight at the patient's right eye, and holding the test card midway between the patient and himself, carries it from the periphery to the center, noting the point where he himself first sees the dot and then the point where the patient sees it. If these two points coincide, the patient has a normal field in that special meridian; otherwise his field is abnormal. Thus, if under the given conditions the observer begins to see the spot at a certain distance from the center but the patient does not see it until it is carried half way in, the patient's field is contracted one-half in that meridian.

By repeating this test for the chief meridians in succession, quite an accurate plot of the field may be made as compared with the field of the normal eye (see Fig. 33).—D.]



[Fig. 33.—Plot of Field taken by the Confrontation Method.

The observer with one eye shut gets an idea of his field of vision in the other as a space bounded by an oval, *a*, *a*, *a*, which, reckoning from the point of fixation, *F*, extends apparently about twice as far to the temporal side, *T*, as to the nasal, *N*, and also extends somewhat further below than above. The patient in the present case sees the test object only half as far out as the observer does to the temporal side, and two-thirds as far out to the nasal, while his field above and below is contracted about one-half. His field, therefore, has the extent shown by the curved line, *b*, *b*, *b*.—D.]

106. If the patient is no longer in condition to see even the hand, we must make use of a *candle flame* which we carry about through the field of vision. In this way, for instance, we test the visual field of a person who is blind from cataract. [In doing this we place the patient in a dark room, blindfold with a towel or handkerchief the eye not under examination, and direct the patient to keep the other eye fixed steadily in one direction. We then carry a candle in the different parts of the field, and, as we do so, alternately shade and uncover it. The patient, if his field is normal, must be able to tell not only when it is light and when it is dark, but also just where the light is at any moment (if necessary, by pointing to it or trying to grasp it).

The reason why it is essential that the patient should not only declare that he sees the light, but also say where it is, is this:] It not infrequently happens that he recognizes the glimmer of the light as soon as the candle flame appears in the periphery of the visual field, but that he tells its place wrong. He says every time, for instance, that it is on the right hand even when it is held in some quite different spot. This is accounted for as follows: When an eye with transparent media is tested with a candle flame in a dark room, there is formed upon the retina, at a point opposite to the flame, an image of the latter, while the rest of the retina is not illuminated and has a sensation of darkness. If that part of the retina which is situated directly opposite the light were insensitive, no light would be seen at all. The case is different with an eye whose media are cloudy. In this the rays emanating from the light are so dispersed by the cloudy media that the whole retina is illuminated no matter where the light is placed. To be sure, the illumination of the retina is not perfectly uniform. There are always more rays falling upon that part of the retina which lies opposite the light than upon the other regions of the retina, and thus the patient is able to tell where the light is; but he would also see the light if the portion of the retina lying opposite the latter were insensitive, since the rest of the retina likewise receives light upon it. Let us assume that the whole retina has become insensitive, with the exception of a region situated on the temporal side. This latter region, no matter where the light may be, will receive diffused light and will perceive it, too. The patient will locate the source of this sensation in the portion of the external world lying opposite to this region of the retina, and will therefore believe always that what he sees is on his nasal side. Hence the mere statement that a light is seen is not sufficient proof of the possession of functional capacity by all parts of the retina. For this it is requisite that the situation of the light be told correctly every time.

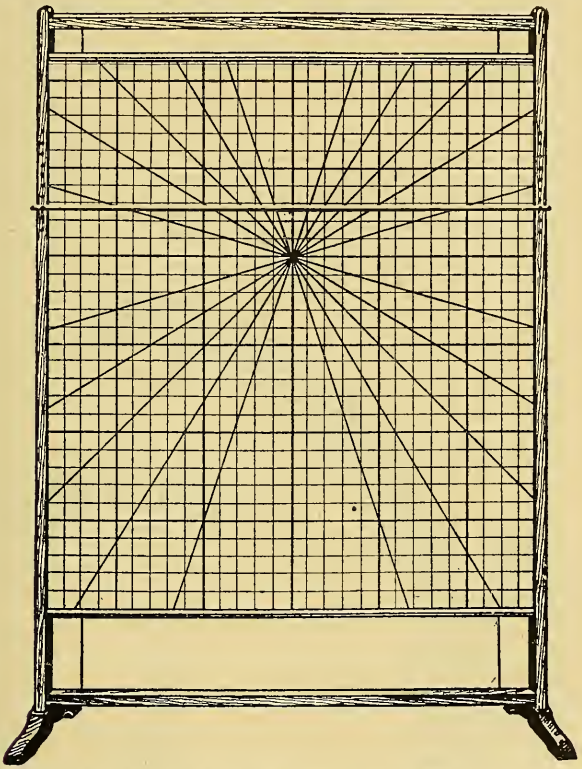
107. We get at the field of vision more exactly by means of the *blackboard.* We place the patient before this and take care that during the examination the distance between the eye and the board remains always the same (e.g., 30 cm.). Directly opposite the patient's eye we make a mark on the blackboard with chalk, and direct him to fix his gaze on this mark during the examination. The chalk is now gradually brought from the edge of the blackboard to its center, and the patient is to tell at what moment he first sees it. By marking on the blackboard the limits of the visual field in every direction and connecting the points thus determined, we fix the extent of the field of vision. The size of the latter is, of course, in direct proportion to the distance at which it is taken.

[The blackboard may be conveniently replaced by a curtain (*Bjerrum's curtain*), one form of which is shown in Fig. 34.

This is used at two distances, viz. 30 inches (0.75 metre) and 60 inches (1.50 metres). The former distance is used in determining limitations of the field lying anywhere within 50° from the center (especially sector-like and hemiopic limitations), in determining limitations of the color-field, in mapping the field of fixation (§ 676) and in plotting double images (§ 662). The 60-inch distance is used when we wish to delimit central and paracentral scotomata (§ 113) and enlargements of the blind spot (§ 112). Diagrams for plotting defects at these two distances are shown in Figs. 38 and 326.—D.]

108. Even this method is not entirely free from drawbacks which spring from the difficulty of projecting a hollow sphere like the retina upon a plane. One important drawback is that unequal distances in the field of

vision correspond to equal distances on the retina. Thus, in Fig. 35, the distances *m a* and *b c* upon the retina are equal, each corresponding to an angle of ten degrees. In the field of vision projected on the board, *T T*, however, the section (from 70° to 80°) that corresponds to the second region of the retina is many times greater than that (from 0° to 10°) which corresponds to the first. Hence, a spot upon the retina of definite size that has become insensitive to light would in such a visual field appear as a gap, the



[Fig. 34.—Tangent Plane for Plotting Central and Paracentral Scotomata, the Field of Fixation, and the Double Images in Paralysis.

The figure shows the reverse, or white side of the curtain forming the tangent plane, the other side, turned toward the patient, being a dead black and without markings except for a white-headed pin right at its center. The curtain can be raised and lowered, so as to bring this pin directly in front of the patient's eye. The small squares of the checkerboard in the figure denote two-inch (5 cm.) intervals, the larger squares intervals of 10 inches (25 cm.). Pins are thrust into the black side of the curtain, to show the outlines of scotomata, the limits of the field of fixation, the exact site of double images, etc. The plot outlined by these pins on the reverse side of the curtain may be transferred at once to a chart (Fig. 38) which has printed on it a diagram like that shown in the figure.—D.]

size of which would be quite different according as it is nearer to or farther from the center, and thus mistakes might be caused. A second evil is that the whole of a normal visual field does not find a place on a plane, be the latter ever so large. The normal field of vision, that is, extends outward to 90° and more. Therefore, as is evident from Fig. 35, the temporal limit of the visual field can never be projected on the board.

After what has been said, therefore, there is only one exact method of representing the entire visual field, and that is by projecting it upon a hollow sphere (Aubert). Upon this principle different *perimeters* are con-

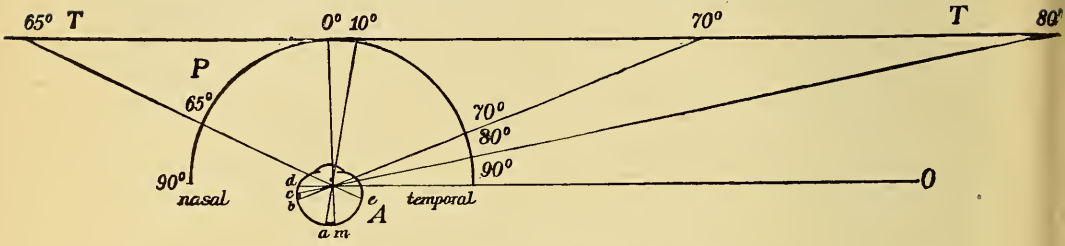
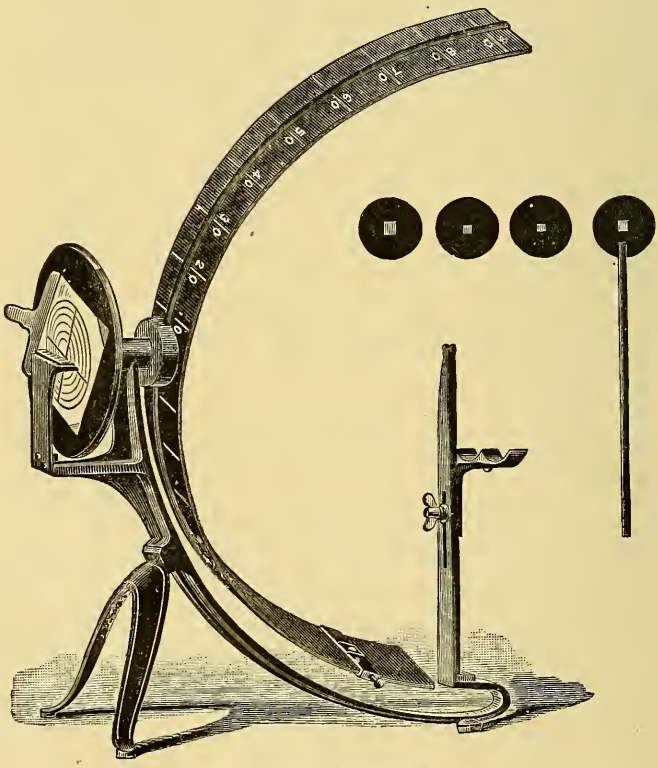


Fig. 35.—Projection of the Visual Field.

The field of vision of the eye, *A*, projected in the semicircle of the perimeter, *P*, extends from 65° on the nasal side to 90° on the temporal side, corresponding to the points, *e* and *d*, of the retina. These mark the anterior border of the sensitive portion of the retina, which extends farther forward on the nasal side than on the temporal. On a flat surface, *T T*, the field of vision cannot be represented as far as its temporal limit, since its projection, *O*, falls outside of the surface.



[Fig. 36.—A Perimeter.

The perimeter consists essentially of a graduated arc along which the test object is carried. This arc is capable of rotation through all meridians, the meridian which it occupies and the position of the test object on the arc being marked upon the diagram, which, as shown on the left side of the cut, is attached to the instrument. The patient sits with his chin in the chin rest sliding in the upright, shown on the right-hand side of the cut.—D.]

structed. To Förster belongs the credit of having introduced this instrument into ophthalmic practice. Förster's perimeter consists, not of a complete hemisphere, but of a metallic semicircle (Fig. 35, *P*; see also Fig. 36) which represents, as it were, one meridian of the hemisphere. The semicircle is capable of being revolved so as to take the direction of each meridian in succession. The patient supports his head on a chin rest which is so placed in front of the semicircle that the eye to be examined is situated in the center of curvature of the latter. In the examination the eye must be fixed upon the middle point of the semicircular arc, while the mark that serves for the test is carried to and fro along the latter. A scale of degrees marked upon the semicircular arc enables us to read off directly the situation of the boundary of the visual field, and the result obtained is transferred to a diagram (Fig. 37).

[The perimeter or the confrontation method will show gross alterations in the field, and must be employed in any event when the field is of anything like normal extent. For very contracted fields and particularly for mapping out central or paracentral scotomata (see page 124) the perimeter has too small a radius. In this case it is much better to use the Bjerrum curtain (§ 107).—D.]

Von Graefe was the first to call attention to the importance of testing the visual field in ophthalmic practice. He showed that for many intra-ocular diseases there are special varieties of contraction of the visual field, which are more or less characteristic of these diseases, and can be utilized for their diagnosis. Since then the study of the visual field has been much cultivated, so that at present its examination has great significance, both for diagnosis and prognosis.

109. Extent of the Field of Vision.—The normal field of vision, as a glance at the appended diagram (Fig. 37) shows, does not by any means extend equally far in all directions. It stretches farthest toward the external (temporal) side, where it has an extent of over 90°. Accordingly, we can still see objects on the temporal side, although they lie in, or even somewhat behind, a plane passing through the pupil (for example, the point *O* in Fig. 35). This is rendered possible by the fact that the rays from such a point undergo such strong refraction at the surface of the cornea that they can still enter the pupil. The field of vision is much less extensive in other directions, especially in directions inward and upward. The cause of this is to be sought for in the fact that the nose and the eyebrows project into the field of view and limit it. This obstacle can indeed be partially overcome by making suitable rotation of the head while the field of vision is being examined, but even then we never find the field of vision as extensive on the nasal side as it is on the temporal. The cause of this is that the margin of the percipient layers of the retina does not extend as far forward on the temporal side as on the nasal (Fig. 35, *d* and *e*).

110. [The field that we obtain in any given case, either with the perimeter or with other methods, will vary according to the size and conspicu-

ousness of the test object, the background against which the object is projected, the way the light falls on it, the intensity of illumination, the state of adaptation of the patient's eye, and other circumstances. This is especially true of pathological cases, in which we shall often find that the field shows considerable variations from day to day without there being any act-

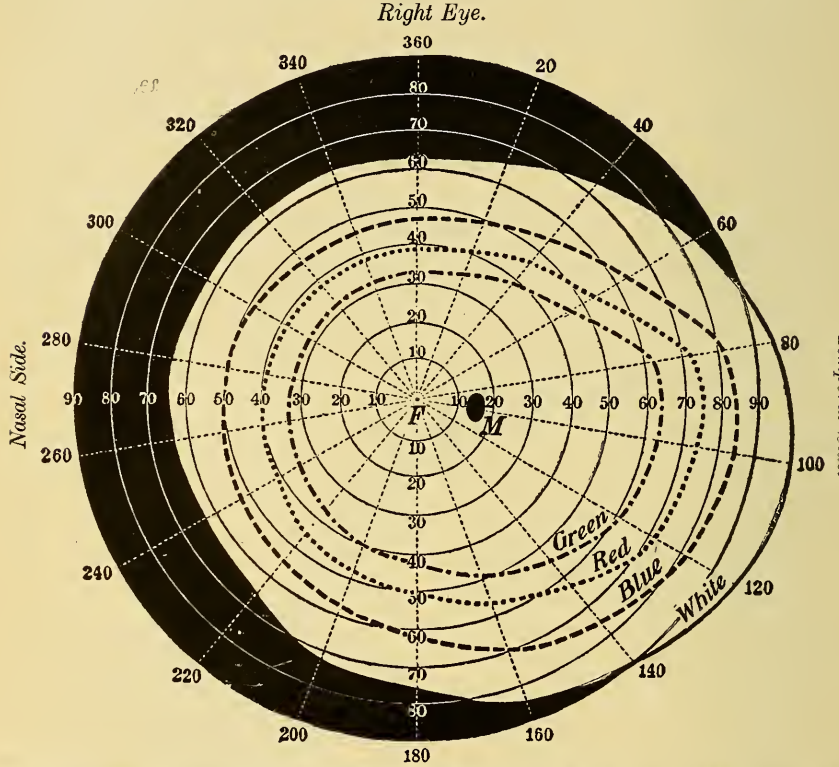


FIG. 37.—FIELD OF VISION OF THE RIGHT EYE FOR WHITE, BLUE, RED, AND GREEN, FOR A TEST-OBJECT 20 MM. SQUARE. (After Baas.)

[The size of the field of vision varies, owing to the encroachment of the brow or nose. This encroachment can be removed by getting the patient to fix an excentric part of one perimeter arm, while the test-object is carried along the arm opposite. Thus a field, somewhat larger than that usually given will be delimited (true field—Mauthner). In normal eyes, the field for white is essentially the same with smaller (even 2 or 3 mm.) test-objects. The field for colors diminishes as the size of the test-object is reduced, and in any event varies so considerably with the tint of color used, the illumination, the adaptation of the eye, and other circumstances, and also varies so considerably in different subjects, that no standard limits can be indicated. The limits given in the illustration must be regarded as maximal, being in many cases 15° or 20° less. Particularly it is the case that the field for red is uncertain, owing to the fact that toward the periphery red changes to orange and then yellow. This zone of uncertain coloration is some 10° in width, and hence the patient's estimates of the limits of the field for red may vary by this amount. Again it not infrequently happens that the field for green equals or exceeds that for red, especially when spectral colors are used. These facts render tests of the color fields doubtful in any case and worthless unless the precise conditions of the test are noted. The field for any color, however, should in general be roughly concentric with the field for white, and any marked variation in this regard would be abnormal. With sufficiently large objects, the limits of the color fields are practically co-extensive with those for white.—D.]

ual change in their condition itself to cause it. In fact the comparison between two perimetric records either of the same case or of different cases may be quite misleading unless we are sure that the conditions under which

the two records were taken were identical. Hence, a perimetric record is not complete unless the attendant circumstances, and particularly the size and kind of test object employed, are noted.

In taking the field it is important that the test-object used be not too large. Unless the vision is quite defective, it is better not to use an object larger than 5 mm. square, and it is well to supplement this with examinations made with objects 1 and 2 mm. square. Tests made with these minute squares sometimes reveal relative defects, which are not apparent when the larger objects are used. They thus afford the same sort of information that is given by tests made with colors (see § 115), and often in a more trustworthy fashion (Walker).

So also in using the Bjerrum curtain, especially for determining faint scotomata, it is best to employ white balls from 1 to 3 mm. in diameter, and supplement these only in case of necessity with larger objects (7 mm. ball).—D.]

111. The *pathological alterations* of the visual field consist in its curtailment. This is either produced by a pushing in of the boundary of the visual field at some point, or it occurs under the form of gaps lying like islands inside of the field of vision.

Narrowing of the visual field *at the periphery* presents varying characters. If the limits of the visual field are brought nearer to the center from all points alike we speak of a concentric contraction. When this is considerable, it results in that incapacity for orientation which has been already described, although it may be that direct vision (visual acuity in the narrower sense) is still quite good. In other cases, the contraction extends from one side only of the periphery into the visual field. If it has the shape of a triangle whose base corresponds to the periphery of the visual field, it is called a sector-shaped contraction. A peculiar variety of contraction of the visual field is the hemiopic, in which exactly one half of the field is wanting (see § 545 and Figs. 272 and 273).

Concentric contraction of the visual field, associated with retention of good central vision, we meet with especially in retinitis pigmentosa, and sometimes also in glaucoma simplex. In other diseases which are frequently accompanied by concentric contraction of the visual field, as, for instance, in atrophy of the optic nerve or of the retina and diffuse chorio-retinitis, central vision is also simultaneously and markedly affected. Finally, concentric contraction, often of very high degree, is characteristic of hysterical amblyopia. [Fig. 281.]

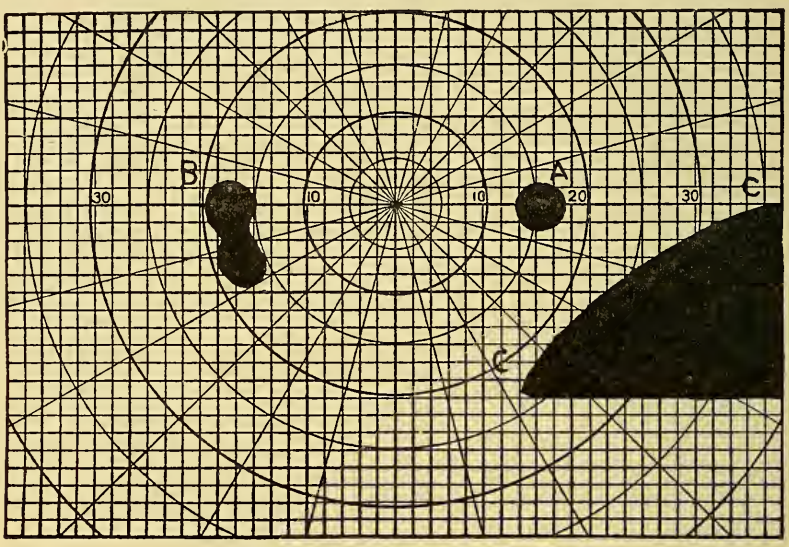
We find the *sector-shaped deficiencies* especially in atrophy of the optic nerve; also in occlusion of one of the larger retinal arteries, when the sector-shaped district of the retina supplied by such an artery has its function abrogated. We observe more extensive, although not triangular, contractions of the visual field in detachment of the retina, and these most often extending in an upward direction, since the detachment, if of long standing, generally involves the lowermost part of the eye. In glaucoma a contraction of the visual field toward the nasal side is of relatively frequent occurrence.

112. Island-like gaps in the visual field are called *scotomata*.[2] One of these exists in the healthy eye at that point of the visual field which corresponds

[2] From σκότος, darkness.

to the entrance of the optic nerve, and is known as Mariotte's blind spot. In the field of vision it lies 15° to 16° to the outside of the point of fixation, *F*.

[It varies in size and shape, but has usually the form of an erect oval, between 5° to 6° broad and 7° to 8° high (thus corresponding to the dimensions of the optic disk; see § 87 and Fig. 38). This area of complete invisibility is surrounded by a narrow rim ¼°—¾° wide, of relative obscuration (penumbra) in which large objects are still well seen, but small objects and colored objects are made out dimly or not at all (Haycraft, Van der Hoeve). Any marked extension of this penumbra or any notable enlargement of the blind spot indicates disease and particularly chronic glaucoma or degenerative or inflammatory changes in the optic nerve. This enlargement may be the only or at least the determining diagnostic sign present in these conditions. Hence in suspected cases



[Fig. 38.—The Blind Spot.

A, blind spot (of normal size) of right eye. It differs from the usual form in being horizontally instead of vertically oval. B, blind spot (enlarged) of left eye. C, C, area of limitation of nasal field in left eye. Taken from a case of glaucoma in the translator's practice. Field plotted on Bjerrum curtain. Figure shows card used for making plot when patient is placed 1.50 metres from curtain.—D.]

the blind spot should be carefully plotted on Bjerrum's curtain, and preferably with minute objects at a distance of 1.5 metres. It is mapped out in the same way as other scotomata (see § 114).—D.]

113. The scotomata which occur as the result of disease have a very different significance for vision according to their situation; and, according to the latter, we distinguish them into central and peripheral. A central scotoma is one which involves the point of fixation (cf. Fig. 278). In this case direct vision is either greatly diminished or is abrogated altogether. The patient can no longer do any fine work, although his power of orientation remains intact. Peripheral scotomata cause little disturbance of sight, especially if they lie far from the point of fixation, in which case they may not come to the patient's knowledge until his visual field is being examined.

A special variety of scotoma is the annular, which encircles the point of fixation like a ring (which is not always completely closed), but leaves intact the point of fixation itself.

Scotomata are most frequently met with in diseases of the fundus with focal lesions; especially, therefore, in chorioiditis disseminata, in which, as a rule, the gaps in the visual field correspond to the separate spots visible with the ophthalmoscope. So long as these gaps affect the periphery only of the visual field, they cause little disturbance of sight. If they are very numerous, the visual field acquires a sieve-like character. If, finally, one of the chorioiditic foci is localized at the region of the chorioid corresponding to the yellow spot, the visual power is very considerably reduced by the formation of a central scotoma in addition to the scotomata in the periphery.

Isolated *central scotomata* occur in diseases of the retina and chorioid at the posterior pole of the eye, especially as a result of syphilis, or myopia of a high degree, and of senile changes. In all these cases there corresponds to the scotoma a change in the region of the macula lutea, visible with the ophthalmoscope. In another series of cases, on the contrary, a central scotoma exists, but the macula on ophthalmoscopic examination looks normal; the cause of the scotoma is then to be looked for in the optic nerve. In the latter it is just those fibers which supply the region of the macula lutea that are the most favorite seat of disease (in retrobulbar neuritis and in toxic amblyopia, glaucoma simplex, etc.). [Normal eyes will develop a relative central scotoma if placed in a room that is sufficiently darkened. If the room is darkened still more, the scotoma becomes absolute—under-sensitiveness of the fovea to light in eyes that are adapted to the dark (Hess).

A *central scotoma*, even when so minute as to be demonstrable with difficulty, usually causes much trouble in near work. A person with such a scotoma may have comparatively good vision (20/40) for distance, and yet may be utterly unable to read even large type; while a person with similar vision but no central scotoma can read quite well. A person with central scotoma reads letters in a characteristically hesitating way, picking out the letters slowly and often missing a letter altogether.—D.]

As the expression scotoma is used in different senses, it will require here a more precise explanation. We distinguish between positive and negative scotomata (Förster).

By a *positive scotoma* we understand a dark spot which the patient perceives in his visual field—projects, that is, upon some portion of his visual field. The cause of a positive scotoma lies either in the refracting media or in the retina. It may have its basis in fundus changes, e.g., an exudate or a hæmorrhage into the retina. Such lesions throw a shadow on the rods and cones, as these are the furthest back of all the layers of the retina, and this shadow is perceived and is projected exteriorly. Scotomata of this kind are best brought to light by making the patient fix his gaze upon a uniformly bright surface (e.g., a sheet of white paper). They are often more readily perceived if the illumination is at the same time diminished (as by letting down the window curtains). We can direct the patient to make a copy of the dark spots that become visible upon the paper, and from this we can determine the position and extent of the diseased portions of the retina. Opacities in the refracting media also throw their shadow upon the retina, and are therefore visible as dark spots. If the opacities lie in the vitreous they are motile (muscæ volitantes) and the scotomata caused by them are characterized as motile scotomata. It is better not to use the expression scotoma for cases of this sort.

We characterize as a *negative scotoma* a hiatus in the visual field, an isolated spot within the confines of which the patient does not perceive any external objects. Such a scotoma is usually not discovered until the visual field is examined. But there is nothing to prevent a negative scotoma from being at the same time a positive one, too; the same

diseased regions of the retina that are insensitive to external luminous impressions can at the same time be themselves perceived as dark spots and be projected exteriorly.

Negative scotomata are divided into absolute and relative according as the object used in examining the field of vision becomes altogether invisible or simply indistinct in the confines of the scotoma. Hence an absolute scotoma for white is present if a white object disappears entirely at one spot, but there is a relative scotoma for white which at that point simply appears of a less pure white, or indistinct in outline. But even when a white object shows no change whatever, this does not prove that there is no change in the field of vision. We must then make the examination with colored objects also. For with a certain diminution of the visual power the ability to distinguish colors accurately disappears, while the objects themselves, owing to differences in their luminosity, can still be recognized. For example, in a recent case of chronic poisoning by nicotine, the visual field, measured with the aid of a white test object, may seem quite normal; but if a small red paper disk is chosen for the examination there is a small region in the center in which the disk is either not recognized as red at all (absolute scotoma for red) or less vivid there (relative scotoma for red).

114. [Scotomata (including the blind spot) are best mapped out on Bjerrum's curtain (preferably at a distance of 1.50 metres).

Scotomata, especially central scotomata, are often hard to delimit, because the patient, on account of his poor central vision, fails to see distinctly the central spot on the curtain which he is asked to fixate, and hence lets his eye wander about, so as to project his scotoma on successively different portions of the curtain. To obviate this, we employ some device by which the other eye, presumably sound, maintains fixation and yet does not see the test-object. This may be effected by a specially devised form of stereoscope (Haitz's apparatus). Another device is the *complementary-color test.* This is based on the fact that when a sufficiently dark colored object is looked at through a darkish glass of complementary color it appears black and hence, when placed on a black background like the Bjerrum's curtain becomes practically invisible. Hence to map out a scotoma in the left eye, we place a dark green glass before the right, and carry a dark red object over the curtain—directing the patient at the same time to look steadily at the center pin of the curtain with his right eye. At points outside of the scotoma, the test-object appears red, because still seen by the left eye, but when it reaches the portion of the curtain corresponding to the scotoma, it suddenly disappears, because it is now invisible to both eyes.

As above stated, scotomata, especially faint positive scotomata, are often best projected on a white surface, instead of the black curtain. In this case the complementary-color test may be replaced by the *homonymous-color test.* In this the sound eye is covered with a red glass, and a light spectral red is used as a test-object. This, seen through the red glass appears white, and hence is practically invisible against the white background.

Another method of delimiting scotomata, and especially the blind spot, with accuracy is to use *two test-objects* which are carried from opposite sides towards the limits of the scotoma, then brought within the latter, and then separated until both are just simultaneously visible. This fixes one diameter of the scotoma, and by repeating the test in different diameters, a fairly accurate plot may be made. The result may be checked by putting in a series of white pins round the periphery of the scotoma as found. If when the eye is properly directed these all disappear at once, the plot may be regarded as accurate.—D.]

115. Color Fields.—Even in the normal visual field the perception of *colors* is not everywhere the same. Just as in regard to the visual acuity, so also in regard to the color sense a distinction must be made between central

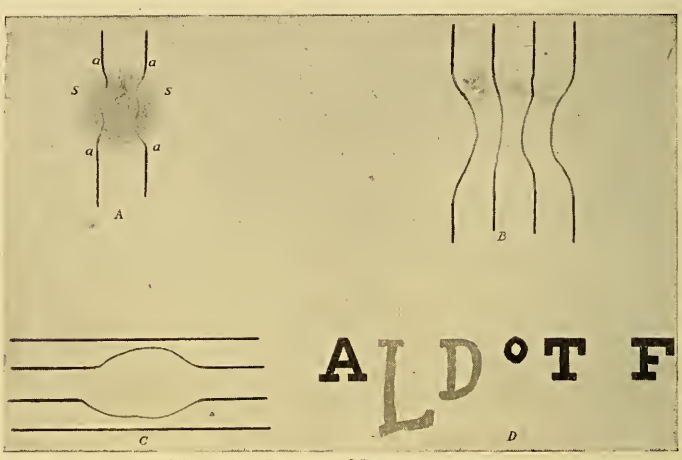
and peripheral color perception. While the former is tested by simply placing samples of different colors before the patient, the latter must be tested like the visual field by using for test objects colored marks which are moved to and fro on the board or on the perimeter. The bigger, more vivid, and better illuminated the colored surfaces used for this purpose are, the further toward the periphery will their color be distinguished, and they may even be distinguished up to the extreme limits of the field. But when the examination is made with the ordinary test objects used with the perimeter (colored squares of paper 3 to 10 mm. in diameter) the most peripheral portions of the retina are found to be color blind. If such a colored square is pushed from the periphery of the visual field toward the center, the person examined at first recognizes only the presence of a moving object. It is not until the square approaches nearer the center of the visual field that its color is correctly given. The moment when this occurs is not the same for all colors, some being recognized farther out from the center than others. The visual field for green is the smallest, that for red somewhat larger, that for yellow still larger, that for blue the largest (see Fig. 37).

The examination of the visual field with colored objects is of great practical importance. For instance, we find in one case the visual field normal when tested with white, while the examination with colors shows already a considerable introcession of its borders at one spot. After some time, if the disease has progressed, we now, on testing with white, establish the same deficiency in the visual field that had before existed for colored objects only. The examination with colors is accordingly a more delicate test than that with white; it betrays a diminution of the visual power before it has advanced so far that a white object can no longer be recognized. If, therefore, we take two cases in which the visual field for white is equally large, but the visual field for colors is unequal, that case in which the visual field for colors is smaller affords the worse prognosis, since here a still further diminution of the general visual field is to be expected. Rapid diminution of color perception is pre-eminently associated with the progressive lesions of the optic nerve that lead to blindness. The examination of the visual field with colors is also requisite for the recognition of central scotomata, so long as they are not absolute. Furthermore, the way in which the color sense is diminished gives us a clew as to the site of the morbid changes. Thus diminution in the perception of blue corresponds to a lesion of the percipient elements (rods and cones) of the retina, such as occurs in chorioiditis, retinitis, detachment of the retina and hemeralopia; diminution in the perception of red and green to a lesion of the conducting elements, as in affections in the optic nerve. If, however, the vision is impaired simply by opacities in the media, the perception of colors is normal. [Owing to the difficulty of standardizing the color tests and the conditions under which they are made, the results obtained by them are, as a rule, quite unsatisfactory, and more reliable information is furnished by tests made with white objects of varying sizes (see page 121).—D.]

116. [**Metamorphopsia.**—Metamorphopsia is the condition in which objects appear distorted.

It may be due to defects in the refractive media (astigmatism, especially of the irregular kind)—*refractive* metamorphopsia—or to displacement of the retinal elements (*retinal* metamorphopsia). The latter occurs when the retina is lifted up by an exudate (retinitis, chorioiditis) or an underlying liquid or growth (detachment of the retina, tumor

of the retina or chorioid). If the process is such that the retinal elements are spread apart, as for instance occurs in a recent chorioiditis or retinitis, an object looked at will look smaller than it is (*micropsia*). This is because a retinal image, that would normally occupy three retinal elements, now, owing to the distention of the retina, occupies but two, and hence affords an impression corresponding to that afforded by an object of only



[Fig. 39.—Retinal Metamorphopsia.

A, *Micropsia*, evidenced by an irregular bowing in of the parallel lines at *a*, *a*. Due to some cause producing distention of the retinal elements (detachment of retina, recent central chorioiditis or retinitis). The case depicted was one of central chorioiditis with a positive central scotoma, *s*, *s*. Within the scotoma the lines are curved in and are distorted. B, metamorphopsia in same case nine years after; positive scotoma still present. C, *Macropsia*, evidenced by an irregular bowing out of the parallel lines, *c*. Due to any cause producing shrinking of the retinal elements (atrophic chorioiditis, retinitis). The case depicted was one of solar retinitis in the translator's practice. D, the distortion and change in hue produced in a line of test-types by the metamorphopsia in this case. The letters are all really of the same height and blackness.—D.]

two-thirds the actual size of the object looked at. On the other hand, when the retinal elements are abnormally crowded together, as occurs in the atrophic stage of retinitis and chorioiditis, objects look larger than they really are (*macropsia*).

The best test for retinal metamorphopsia is a series of parallel lines like the lines of music (Fig. 39).

Retinal metamorphopsia if due to chorioiditis or retinitis is usually combined with a complete or partial scotoma.

Micropsia and macropsia also occur in disorders of accommodation (see § 797) but are not then associated with distortion like that shown in Fig. 39.—D.]

117. Light Sense.—Let us assume that we have before us two persons who in ordinary daylight have the same visual acuity; both under equally good illumination read print of the same size at the same distance. We now gradually lessen the illumination. As a result of this, the difference in brightness between the black letters and the white paper diminishes and the letters are distinguished with greater and greater difficulty. At a certain stage in the process of obscuration, one of the two persons ceases to recognize the print while the other is still able to read, and the darkening has to be carried further in order to make reading impossible for him. In this case we say: The two persons have the same space sense, i.e., the same susceptibility of the retina for impressions of forms, but they have a different light

sense (L)—i.e., a different susceptibility for impressions of brightness and of differences of brightness.

The light sense can be tested in various ways. We determine either the lowest limit of illumination with which an object is still visible (minimum stimulus) or the smallest difference in brightness which can still be appreciated (minimum of differentiation). The most usual method of measuring the light sense is with Förster's *photometer*, which gives the minimum stimulus. This instrument, which is represented in horizontal section in Fig. 40, is placed in a perfectly dark room. A box, A, blackened on the inside, bears on its anterior wall two apertures for the two eyes, a and a_1, which look through these apertures at a plate, T, which is placed upon the posterior wall, and upon which large black stripes upon a white ground are placed as test objects. The illumination is produced by a normal candle,[3] L, the light from which falls through a window, F, into the interior of the box. In order to make the illumination perfectly uniform, the window is covered with paper which is made translucent (by impregnating it with fat). By a screw, S, the size of the window can be altered from complete closure up to an aperture of five centimeters square. In this way the illumination of the plate is varied. The patient is first made to look into the apparatus with the window closed and the plate therefore unillumined. Then the window is slowly opened until the stripes upon the plate can be recognized. The size of the opening requisite for this purpose gives a measure of the light sense of the person examined. In conducting this examination the precaution must be adopted of making the person that is examined stay beforehand in darkness. If we come from daylight into a moderately darkened room we see so little for the first moment that we cannot move about without stumbling over the objects in the room. The longer we remain in the latter the better we see, and at last perhaps see well enough to be able to read. This [complete adjustment of the eye to the surrounding illumination] we call *adaptation* of the retina [and, according as the eye is adjusted for the dark or for bright light, we speak of dark-adaptation or *scotopia* (Parsons) and light-adaptation or *photopia*]. In the examination of the light sense, a period of adaptation of ten minutes, which the patient must pass with bandaged eyes in a perfectly dark room, is sufficient for practical purposes. [A better instrument than Förster's is the *adaptometer* of Nagel.—D.]

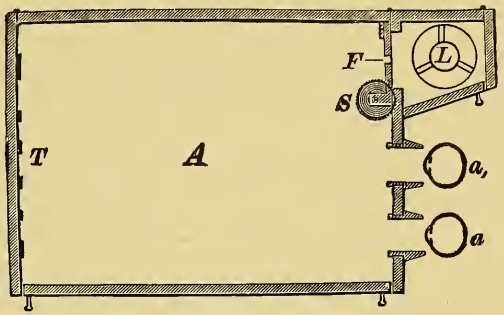


Fig. 40.—Photometer of Förster.

The examination of the light sense in different diseases has shown that it is not always by any means diminished in proportion to the visual acuity, but is sometimes but little diminished, sometimes excessively so, a circumstance from which diagnostic points may be gathered. The diminution of the light sense is greatest in those cases which are characterized as hemeralopia (see § 569).

[The sensitiveness of the fovea to light is less than that of the periphery of the retina. (See §§ 113 and 569).—D.]

118. Simulation of Blindness.—In testing the function we shall at times have to reckon with the fact that the patient is purposely trying to lead the physician astray by simulating blindness or weakness of sight when

[3] [I.e., one of one-candle power.—D.]

these do not exist. This most frequently occurs with those persons who wish to be relieved of military service or who wish to get damages for an accident. Quite similar are the symptoms in hysteria, in which, however, it is not a question of intentional deception, but of unconscious imagination.

We are first led to suspect simulation by the lack of agreement between the results of the functional testing and of the objective examination; an eye, for example, which is alleged to be perfectly blind presenting no pathological changes of any sort. Or the tests of the individual functions give contradictory results, inasmuch as the visual acuity, the field of vision, the color sense, etc., do not stand in the right relation with each other and with the result of the objective examination. In children it often happens that the vision, ostensibly very poor, is at once rendered normal by placing the weakest concave glasses or even plane glasses before the eyes. Various methods of examination have been proposed for furnishing a certain proof of simulation; we shall accomplish this more or less readily by their aid according to the degree of skill of the simulant. Only some of these methods need be here adduced.

Complete blindness of both eyes is rarely simulated; much oftener it is simply unilateral blindness that is alleged; and still more frequently a feebleness of sight actually present in one eye is exaggerated. In the case of an alleged complete blindness of one or both eyes we regard in the first place the reaction of the pupil to the light. If this is well preserved, it will always afford a strong ground for suspecting simulation, although there are rare cases in which in the presence of actual blindness the pupillary reflex for light is still retained (see §318). Schmidt-Rimpler recommends the following procedure: The patient is made to look with the blind eye at his own hand, which he holds in front of him. A blind man will do this without hesitation, since he is informed of the position of his hand by the sense of feeling; a malingerer will perhaps look purposely in the wrong direction. Simulated unilateral blindness can also be discovered in the following way: A lighted candle is brought in front of the good eye and is slowly carried toward the side of the blind eye. The patient is detected if he declares that he still sees the candle at the moment when it is just concealed from the sound eye by the dorsum of the nose (Cuignet).

A number of methods are of service in detecting the simulation of unilateral blindness or amblyopia. These mostly depend on the fact that one does not perceive with which of his two eyes he is seeing, if both eyes are kept open when the tests of sight are made. A man could get at this, if his two eyes were equally good, only by quickly closing one eye or the other for a moment while the sight was being tested, and care must, therefore, be taken that this is not done by the person who is being tested for simulation.

1. We make the patient read, and then hold a pencil in a vertical direction between the eye and the book. If there is vision with only one eye the pencil conceals certain words from it, and thus interferes with reading. If, however, there is good vision with both eyes, those letters which are concealed from one eye by the pencil are visible to the other, and vice versa, and reading is carried on without difficulty (Cuignet).

2. A convex glass of 6 D is placed before the sound eye. In this way the eye is made artificially myopic, so that its far point lies at a distance of about 17 cm. (it being presupposed that the eye is emmetropic). The eye can therefore read fine print only at a distance of 17 cm. or less, but no farther. After placing the glass before the eye we first make the patient read at quite a short distance, and then slowly and imperceptibly move the book farther and farther away. If it is possible in this way to withdraw the book considerably farther than 17 cm. without the patient's ceasing to read, it proves that he has been reading with the eye alleged to be bad. That is, he began reading with the good eye and, when the book was carried too far off for that, continued with the other eye, without noticing the alternation in the employment of the two eyes.

3. We make a show of occupying ourselves with the sound eye only. We take a strong prism (one of 18 Δ), with the base up, and, first holding it in front of the cheek, push it gradually up in front of the eye. Before the base has reached the center of the pupil the eye will see double. For two images of every external object will be thrown upon the retina, one transmitted through the free half of the pupil, the other through the half covered by the prism, and the eye sees double the object upon which it is fixed (monocular diplopia)—a fact which the patient will admit without hesitation, since, of course, it is the sound eye only that is concerned in the matter. Now the prism is imperceptibly pushed along until it covers the entire pupil. Now the eye that is provided with the prism again has only one single retinal image, which, however, is thrown upon a higher point of the retina than is the case in the other eye. If now there is still double vision (binocular diplopia), it is a proof that both eyes see. If we use the test types for this examination and compel the person under examination to read sometimes the upper, sometimes the lower of the two double images, we can determine directly the visual acuity of each eye separately without the patient being aware of it (Alfred Graefe and Baudry).

4. Snellen has constructed a board with test types which are alternately red and green. Before the patient is allowed to read it, a pair of spectacles is put upon him, in which are introduced a red glass for one eye and a green glass for the other. Through the red glass the red letters alone, and not the green, can be seen, because green is the complementary color of red, and therefore green rays are not transmitted through red glass. For the same reason the red letters cannot be perceived through the green glass.[4] If, therefore, any one who is blind in one eye looks through these spectacles at the test types, he will read off only the red, or only the green letters, according as the red or the green glass of the spectacles is placed in front of the eye which alone can see. He will not once suspect that still other letters of a different color lie between the letters that he has read. Should the patient, on the other hand, read all the letters, it proves that he sees with both eyes and in such a way as to recognize the red letters with one eye and the green with the other.

[5. Letters are written on white paper with a black and a red pencil alternately. The subject under examination is then told to read the writing rapidly, while a red glass is held before the sound eye. If he reads the whole correctly, it is a proof that he is able to read with the eye alleged to be blind, for the sound eye, looking through the red glass, cannot see the red letters since these now offer no contrast to the background upon which they are viewed, which appears as red as they.—D.]

[6. In the methods above given the malingerer may readily outwit us if during the examination he slyly closes the alleged poor eye and thus gets an idea of how he ought to see were it really poor. We can prevent this trick if we make him read out loud, and then, while he is reading quite fast and is wholly occupied with what he is doing, suddenly place a prism of 3 Δ or 4 Δ with the base down before the poor eye. Of course, we make sure in doing so that the eye is open at the time. Now, if the eye is really very poor sighted, it will make little difference to him whether the prism is placed before it or not, and hence he will read on as before; but if he sees fairly well with this eye the interposition of the prism will cause very great confusion by the production of superimposed double images and he will at once involuntarily pause and stumble in his reading and perhaps will be unable to go on at all.—D.]

For the tests of the motility of the eye and of binocular vision, see § 672.

[4] [In this test of Snellen's the red and green letters are transparent and placed upon an opaque ground and are hung up before a window so as to be seen by transmitted light. In this case, as stated in the text, only the red letters are seen through the red glass, and only the green through the green glass. The same will occur if the red and green letters are opaque, and placed on a dull black ground, and viewed by reflected light. In either case the conditions are opposite to those which exist in the test next mentioned, in which opaque red letters are viewed by reflected light on a white ground.—D.]

PART III

DISEASES OF THE EYE

DISEASES OF THE EYE

CHAPTER I

DISEASES OF THE CONJUNCTIVA

Anatomy

119. The *conjunctiva* coats the posterior surface of the lids and the anterior surface of the eyeball. It forms a sac, the conjunctival sac, which is slit open anteriorly in a line corresponding with the palpebral fissure. In the conjunctiva we distinguish three divisions. That part of the conjunctiva which covers the posterior surface of the lids and which is closely adherent to the tarsus is called the conjunctiva tarsi; that division which coats the anterior surface of the eyeball is the conjunctiva bulbi. The connection between the two is formed by the third division, which we name the transitional portion of the conjunctiva (conjunctiva fornicis). That region where the conjunctiva is reflected from the lids to the eyeball and which forms the bottom of the conjunctival sac is called the fornix conjunctivæ.

We get a view of the *conjunctiva tarsi* in the living eye by everting the lids. It has a smooth surface and is intimately and immovably adherent to the subjacent tarsus (Fig. 41, *k*). (It is therefore impossible to cover up losses of substance of the palpebral conjunctiva by performing an operation to draw the adjacent conjunctiva over them, as is often done with the conjunctiva bulbi.) On account of its thinness, the conjunctiva tarsi allows the Meibomian glands, which lie in the tarsus itself, to be seen through it clearly.

The microscope shows that the conjunctiva of the lids as well as that of the fornix is covered with a laminated cylindrical epithelium. The mucous membrane proper is of adenoid character—that is, even in the healthy state it contains an abundant quantity of lymphocytes, which notably increase in number with every inflammation of the conjunctiva. The palpebral conjunctiva contains small glands, which are found partly along the convex border of the tarsus (Fig. 41, *w*), partly in the fornix conjunctivæ (Krause's glands, Fig. 41, *kr*). In structure these resemble the lachrymal gland.

The conjunctiva of the upper lid obtains its blood supply from two arterial arches, the arcus tarseus superior and the arcus tarseus inferior.

The arcus tarseus inferior (Fig. 42, *ai*) lies on the anterior surface of the tarsus close to its lower border. To reach the conjunctiva its branches perforate the tarsus through its entire thickness from before backward, 2 to 3 mm. above the free edge of the lid (Fig. 42, *rp*). The line along which the vessels come out along the tarsus is marked by a shallow furrow

(sulcus subtarsalis) on the conjunctival surface of the lid. The arcus tarseus superior (Fig. 42, *as*) lies a little above the upper border of the tarsus upon the fascia tarso-orbitalis, which runs from the tarsus to the margin of the orbit and through which the branches of the arcus superior pass to the conjunctiva. On the lower lid there is but one arterial arch.

In the epithelium of the conjunctiva, particularly in its uppermost layers, are found cells which are undergoing a mucous metamorphosis (*beaker cells*). They occur but sparsely in the normal conjunctiva, but multiply to a great extent in inflammatory disorders of the latter.

The conjunctiva of the tarsus rises in low papillæ, over which, however, the epithelium passes undimpled so that the surface of the conjunctiva is smooth. This continues so up to the convex border of the tarsus, where not only are the papillæ higher, but the surface of the epithelium dips down between the papillæ, forming sulci so that the papillæ become visible as distinct structures and the surface of the conjunctiva acquires a slightly velvety appearance. The extent to which these papillæ are developed shows great individual variations, and the line between a physiological and a pathological state cannot be sharply drawn. The conjunctiva is exposed to external influences more than any other mucous membrane, and hence in every man attacks of hyperæmia occur in it often in the course of life, and these may ultimately result in a permanent alteration of the membrane. This is probably also the cause of the variations in degree of the adenoid structure of the conjunctiva. Small quantities of plasma cells, and small nodules consisting of lymphocytes, are present in the healthy conjunctiva, but these become increased as a result of the state set up by repeated irritation. The like is true of the inconstant, but still very frequently seen tubular depressions lined with cylindrical epithelium which are called, after their discoverer, Henle's glands (Fig. 51, *d*). Furthermore, in middle and advanced life there are found, frequently in the retrotarsal fold, less often in the palpebral conjunctiva, little yellow dots resembling the infarcts of the Meibomian glands (§ 606). This again is an instance of the new formation of tubular glands, in which develop concretions that are visible through the conjunctiva under the form of yellow dots.

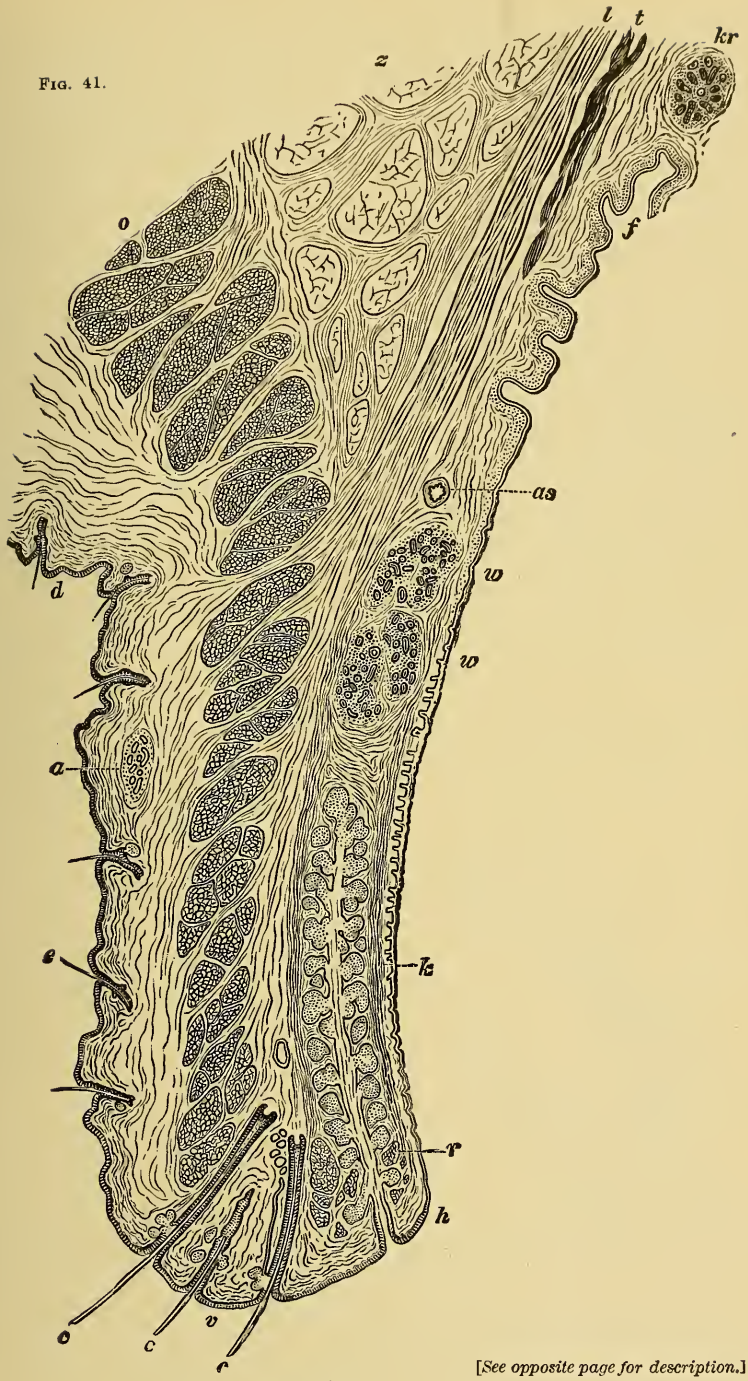
120. The conjunctiva of the *region of transition* [retrotarsal fold] is very readily brought to view in the lower lid by drawing the lid down while the eye looks up. In the upper lid the retrotarsal fold is harder to see. To see it, we draw the lid by the lashes, well down and [then] away from the eyeball, and then, by means of a slender rod placed beneath the eyebrow, press the skin of the lid down until the retrotarsal fold protrudes. [See also page 77.]

The retrotarsal fold is the most lax portion of the conjunctiva, the latter being here so abundant that it lies in horizontal folds. This arrange-

EXPLANATION OF FIG. 41.—PERPENDICULAR SECTION THROUGH THE UPPER LID. Magnified 6×1.

The skin of the lid presents in the upper part a sulcus, defining the overhanging fold, *d;* below, it covers the anterior edge of the lid, *v*. In the skin are found minute hairs, *e*, sweat-glands, *a*, and on the anterior edge of the lid the cilia, *c, c, c*. Adjoining the latter are sebaceous glands (Zeiss's glands); in front of the hair papilla of the most posterior cilium is seen the transversely divided tube of a modified sweat-gland (Moll's gland), the excretory duct of which runs down along the cilium and empties into the hair follicle. Beneath the skin lie the transversely divided bundles of fibers of the orbicularis muscle, *o*, of which those placed most posteriorly at *r* form the musculus ciliaris Riolani. The posterior surface of the lid is covered by conjunctiva which is intimately adherent to the subjacent tarsus, *k*, and over the latter presents isolated papillæ especially in the space between *k* and *w*, corresponding to the upper (convex) border of the tarsus. Still higher up in the vicinity of the fornix, *f*, the conjunctiva shows an adenoid character. The Meibomian glands have their orifices in front of the posterior edge of the lid, *h;* above them lie the branched tubular glands, *w, w*, and still higher up Krause's glands, *kr*, and in front of the latter Müller's musculus tarsalis superior, *t*, and the levator palpebræ superioris, *l*. From the latter leashes of fibers pass between the muscular bundles of the orbicularis to the skin of the lid. *z*, lax connective tissue; *as*, the arcus tarseus superior. Above the roots of the cilia is seen the cross section of the arcus tarseus inferior.

Fig. 41.



[See opposite page for description.]

ment insures the eye its free power of movement. If the conjunctiva were to pass directly from the lid to the eye, as is sometimes observed in consequence of disease of the conjunctiva, every movement of the eyeball would be transmitted to the lids; and if one of the lids was held still with the finger, the eyeball would be hampered by it in its movements. But the conjunctiva is present in such quantity at the fornix that the eye is able to move in complete independence of the lids, the folds in the region of transition being smoothed out or crumpled together, as the case may be. Appearing through the lower fold of transition are the extensive subjacent plexus of veins and also the white glistening fascia. Its lax character and also its abundant blood supply render the fold of transition particularly liable to great swelling in inflammations of the conjunctiva.

121. The *conjunctiva bulbi* covers the anterior surface of the eyeball. It has no aperture corresponding to the cornea, but continues, even if with altered character, over the latter. This continuity of the conjunctiva makes it plain to us why morbid processes of the latter do not stop at the margin of the cornea but are continued upon the surface of the latter, as we see very clearly in trachoma and in conjunctivitis eczematosa. The two divisions of the conjunctiva bulbi are distinguished as the conjunctiva scleræ and conjunctiva corneæ. The conjunctiva corneæ is perfectly transparent, and is so intimately adherent to the cornea proper that it must be regarded as the uppermost layer of the latter, and is better treated of at the same time with the cornea itself (see § 188).

122. The *conjunctiva sclerœ* covers the anterior segment of the sclera in the form of a thin pellicle. It is connected with the sclera by lax connective tissue (the episcleral tissue) so loosely that it can readily be moved about from side to side upon the sclera. It is only at the periphery of the cornea, where it ends in a sharp edge, the limbus[1] conjunctivæ, that the conjunctiva scleræ is intimately adherent to its substratum. It is very thin and elastic and lets the white sclera be seen through it plainly, thus forming the "white of the eye." In old people there is an area at the inner and the outer margins of the cornea which contrasts by its yellow color with this whiteness. This has the shape of a triangle with its base at the corneal margin, and projects a little above the rest of the conjunctiva. It is called the interpalpebral spot or the pinguecula, and is produced by the fact that that part of the conjunctiva which, being included in the interpalpebral fissure, is constantly exposed to atmospheric influences, has undergone an alteration in its tissues.

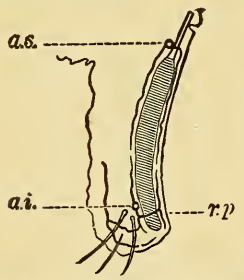


FIG. 42.—ARTERIES OF THE UPPER LID. Magnified 3 × 1.

as, arcus tarseus superior; *ai*, arcus tarseus inferior; *rp*, rami perforantes.

[1] *Limbus*, hem.

The conjunctiva scleræ is covered with laminated pavement epithelium and contains no glands. At the inner angle of the eye it forms a crescentic duplication, the semilunar fold (plica semilunaris), which represents an abortive remnant of the palpebra tertia (nictitating membrane) of animals. To the inside of the semilunar fold is a small, reddish, nipple-like prominence, the caruncle (caruncula[2] lacrimalis), which occupies the bottom of the horseshoe-shaped excavation at the angle of the eye (Fig. 60, *C*). This is shown to be histologically a small island of skin, containing sebaceous glands, sweat glands, and small glands like Krause's glands and having its surface covered with minute light-colored hairs.

This interpalpebral spot owes its name pinguecula[3] to its yellow color, which was formerly referred to deposition of fat in the conjunctiva. What actually is present, however, is a thickening of the conjunctiva, due chiefly to an increase in the number and size of its elastic fibers. Associated with this is the formation of numerous concretions of a yellowish hyaline substance, to which in fact the pinguecula owes its yellow color. As a result of these changes, the conjunctiva in this place becomes less transparent, for which reason the pinguecula appears most prominent when the conjunctiva bulbi is markedly reddened, whether from injection or from extravasation of blood. In this case the pinguecula does not allow the red color of the blood to shine through as plainly as does the adjacent conjunctiva that is not thickened, and the former, therefore, stands out from the red substratum in the form of a light-colored triangle, so that by beginners it is easily confounded with a diphtheritic infiltration of the conjunctiva, or, when the yellow color is pronounced, with a small pustule.

123. Conjunctival Vessels.—The conjunctiva of the eyeball receives its *blood-vessels* chiefly from the vessels of the retrotarsal fold—the posterior conjunctival vessels (Fig. 43, *h* and h_1). Furthermore, the anterior ciliary

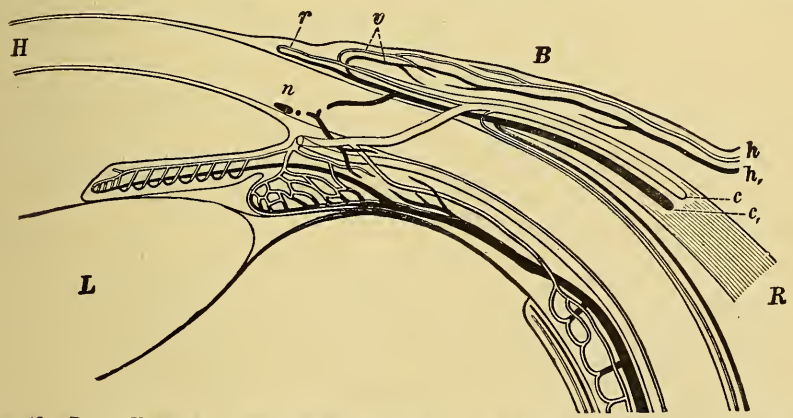


Fig. 43.—Blood-Vessels of the Anterior Segment of the Eye. Schematic. (After Leber.)

The posterior conjunctival vessels, *h* and h_1, communicate with the anterior conjunctival vessels, *v*, which come to meet them and which are branches of the anterior ciliary vessels, *c* and c_1; and with the latter form the marginal network in the limbus. *n*, Schlemm's canal; *H*, cornea; *B*, conjunctiva; *R*, one of the recti muscles; *L*, lens.

vessels (Fig. 43, *c* and c_1) take part in supplying the conjunctiva with blood. These vessels come from the four recti muscles (*R*, Fig. 43) and run under the conjunctiva (through which they are visible, shining with a bluish

[2] *Caruncula*, dim. of *caro*, flesh. [3] *Pinguis*, fat.

luster) until near the edge of the cornea, where they suddenly disappear, since they pass through the sclera into the interior of the eye. But before this happens, they give off branches which end in vascular loops, in the limbus conjunctivæ directly at the margin of the cornea (marginal network of the cornea,—Fig. 43, *r* and Fig. 44). This latter is of great importance for the cornea, which is chiefly dependent upon it for its nutrition. Other branches of the ciliary vessels (anterior conjunctival vessels, Fig. 43, *v*) run backward in the conjunctiva toward the posterior conjunctival vessels (hh_1) and anastomose with them.

FIG. 44.—MARGINAL NETWORK IN THE LIMBUS. (After Leber.)
The arteries are drawn in light color, the veins dark. In the marginal loops can be distinguished the thinner arterial and the thicker venous segments.

We have therefore in the conjunctiva two vascular systems—that of the posterior conjunctival vessels and that of the anterior ciliary vessels. According as the one or the other system is overdistended with blood, the conjunctiva has a different aspect, which we designate respectively as conjunctival and as ciliary injection.

Conjunctival injection presents to us a superficially disposed network of larger and smaller vessels, whose situation in the conjunctiva is proved by the fact that when it is moved about they move with it. The color of the injection is a vivid scarlet or brick-red; the individual vascular meshes are plainly to be recognized. This injection is characteristic of diseases of the conjunctiva itself.

Ciliary injection occurs as a rose-red or pale-violet zone round the cornea (hence the designation peri- [circum-] corneal injection), in which we are unable to recognize clearly any individual vessels. With injection of greater intensity we see, still farther removed from the cornea, a coarser network of vessels, which are to be recognized as deeply placed by their

violet color and their hazy appearance; by the fact, moreover, that when the conjunctiva is displaced over them they do not move with it. Ciliary injection is most markedly distinguished from that of the conjunctiva by its violet hue as well as by the diffuse appearance of the redness, due to the fact that the individual engorged vessels can be made out but indistinctly or not at all. Ciliary injection most frequently accompanies diseases of the cornea, and also of the iris and the ciliary body, parts which belong to the vascular district of the anterior ciliary vessels. On account of the numerous anastomoses between the two vascular districts of the conjunctiva, we find both injected in every inflammation of any great violence in the anterior section of the eyeball; even then, however, it is still generally possible to recognize, along with the superficial conjunctival injection, the ciliary injection, more deeply situated and directly surrounding the cornea.

[The distinction between deep and superficial injection may be made with adrenaline, which temporarily dispels the latter and does not greatly affect the former.—D.]

[Inflammation of the Conjunctiva

124. Inflammation of the conjunctiva—conjunctivitis—comprises many forms, some etiologically distinct, like gonorrhœal and diphtherial conjunctivitis, others clinically well defined but of complex or uncertain etiology. The clinical classification is followed here.—D.]

I. Conjunctivitis Catarrhalis

(*a*) *Conjunctivitis Catarrhalis Acuta*

125. Symptoms.—Acute conjunctival catarrh, in the *lighter* cases, chiefly affects the conjunctiva of the lids and of the region of transition. The conjunctiva of the lids presents a vivid redness and is relaxed. The injection is usually reticulate—i.e., the separate vessels can still be distinguished as such; it is only when the injection is especially dense that the conjunctiva acquires a uniformly red appearance. The surface of the conjunctiva is smooth; catarrh is thus distinguished from some other forms of inflammation of the conjunctiva in which the latter is infiltrated and subsequently hypertrophied, as shown by the unevenness of its surface. The retrotarsal fold (as well as the plica semilunaris) is likewise greatly reddened and is somewhat swollen, while the conjunctiva bulbi shows little or no change.

The *severe* cases are distinguished from the lighter ones by the fact that the process invades the conjunctiva bulbi [and sometimes the cornea, too. These severe forms occur especially in epidemics of conjunctivitis]. The redness and swelling of the palpebral conjunctiva are greater, and

moderate œdema of the lids is often present at the same time. The conjunctiva of the eyeball shows both a dense reticulate reddening and a slight degree of swelling. Very frequently [especially in the form caused by the Koch-Weeks bacillus] we find in the midst of the reticulate injection red-colored spots—i.e., small hæmorrhages, ecchymoses of the conjunctiva—produced by the rupture of small vessels. The severer cases, in which the conjunctiva is affected throughout its entire extent, are designated under the name of *ophthalmia catarrhalis*, to distinguish them from the lighter forms, which are named simply conjunctivitis catarrhalis; and if at the same time there is a specially marked swelling of the retrotarsal fold, as occurs particularly in the epidemic form, it is called "tumid catarrh."[4] [Such chemosis is specially marked in cases due to the pneumococcus (Butler). Moreover, in severe cases of pneumococcus infection and sometimes also in other forms of infection, if specially virulent, there may be a formation of false membranes.—D.]

Inflammation of the conjunctiva is accompanied by increased conjunctival *secretion*. This secretion appears under the form of flakes of mucus, swimming in the abundant lachrymal fluid. The more intense the inflammation the greater the secretion, and the more the character of the latter changes from mucous to purulent. Violent cases of ophthalmia catarrhalis, therefore, are in their inception often hard to distinguish from an acute blennorrhœa of slight intensity, although, of course, the subsequent development of the case makes the diagnosis clear. The secretion which exudes from the palpebral fissure dries at night upon the edges of the lids and glues them together.

The *subjective* symptoms consist of photophobia, and of itching and burning of the eyes. The intensity of the annoyance given depends naturally upon the degree of inflammation. Violent pain, however, is but rarely present, and then, as a rule, is excited, not by the catarrh itself, but by its complications (especially ulcers of the cornea). [The same is true of photophobia, which occurs most frequently in cases with corneal involvement and as a result of improper treatment, such as bandaging the eye.] A very troublesome sensation frequently present is that of a foreign body being in the eye, and is caused by flakes and filaments of tough mucus in the conjunctival sac. If such filaments lie upon the cornea, they produce the disturbances of sight of which the patients sometimes complain. These are distinguished from visual disturbances of more serious character by the fact that clear vision is immediately restored by brushing the mucus off with the lids. It is a characteristic feature of catarrh that all its disagreeable characters are least pronounced in the morning, and afterward gradually increase until they reach their highest point in the evening.

[4] [There is no good English equivalent for the German "Schwellungskatarrh."—D.]

126. Course and Prognosis.—The prognosis is favorable in uncomplicated cases, the inflammation disappearing spontaneously after from eight to fourteen days. Not infrequently, however, there remains a condition of chronic inflammation (chronic conjunctival catarrh), which, to be sure, causes less annoyance than the acute stage but, on the other hand, is protracted over a long time. In the majority of cases acute conjunctival catarrh attacks both eyes, and either both simultaneously or one eye a few days after the other.

The *complications* which are observed in catarrh are corneal ulcers and iritis. The development of corneal disease is manifested by an increase in the pain and photophobia. At first we recognize, in the neighborhood of the corneal margin, small gray points, which are arranged in a row concentric with the corneal margin. The next few days, these punctate infiltrations of the cornea become more numerous and at length confluent, so as to form a small gray crescent. By a process of superficial disintegration an excavation is produced, so that finally a crescentic ulcer is formed, situated very near the corneal margin and concentric with it. Such ulcers are characteristic of conjunctival catarrh and are hence called *catarrhal ulcers*. Ordinarily the ulcer becomes clean quickly and heals, leaving behind it a slight arcuate opacity; in cases, however, that are of special intensity, perforation of the cornea may occur. [For other varieties of corneal ulcers occurring in conjunctival disease see § 206.—D.]

From the clinical picture of acute conjunctival catarrh, as sketched above, we sometimes find variations forming what are described as special varieties of catarrh. Among these variations belongs the devlopment of so-called *follicles*, which will be described more precisely in § 133. Another variety of catarrh is *vesicular catarrh*, in which the conjunctiva of the tarsus is covered with numerous minute elevations, looking as if fine sand had been scattered over a moist glass plate (Arlt); according to Mayweg what we have to do with here is very small follicles. A third variety of catarrh is that to which is given the name of the *pustular* form. In this, flat elevations develop upon the conjunctiva bulbi, mostly near the margin of the cornea. These break down into pus on their surface, and in this way are formed grayish or yellowish ulcers with somewhat elevated base and of the size of a millet seed or more. These have a great resemblance to the efflorescences occurring in conjunctivitis eczematosa (§ 157). The distinction between the pustular form of catarrh and conjunctivitis eczematosa consists in the fact that in the former the phenomena of catarrhal inflammation are present in the conjunctiva of the lids and of the retrotarsal fold, while in conjunctivitis eczematosa these divisions of the conjunctiva take little or no part in the inflammation. Many regard this form as a mixture of conjunctivitis catarrhalis and conjunctivitis eczematosa. And, as a matter of fact, we must take this view into consideration in our treatment, inasmuch as in the beginning of the disease the application of the silver solution ordinarily proves to be the best thing, but later, after the more violent inflammatory phenomena have run their course, calomel is of the most service.

The three varieties of catarrh just named are seen chiefly in children or in adolescents. In adults, on the contrary, we encounter much more frequently the crescentic ulcers of the cornea which result from catarrh, but which are rarely observed in children.

[Special varieties of conjunctivitis, marked by their severity, are: *Squirrel-plague*

conjunctivitis, a very severe conjunctivitis transmitted from squirrels and attended with general symptoms (fever, glandular enlargements) and with the formation of necrotic ulcers in the palpebral conjunctiva; and *Samoan conjunctivitis* (conjunctivitis samoensis), an acute infectious disease, marked by rapid onset, severe pain, photophobia, intense conjunctival inflammation, soon becoming purulent, and a tendency to corneal destruction (Ely).—D.]

[In Europe and America the *course* of conjunctivitis is that described above. In the Near East, where conjunctivitis is extremely prevalent, untreated cases are apt to drag on for months, and cases due to pneumococcus or mixed infection resist treatment. Cases due to the Koch-Weeks bacillus are readily cured, but tend to relapse; those due to the Morax-Axenfeld diplobacillus tend to become chronic, but are amenable to treatment (Butler).—D.]

The *complications* above mentioned are observed only in severe cases—that is, only in ophthalmia catarrhalis. They very often owe their existence to faulty treatment of the catarrh. Among the laity, all sorts of household remedies are in use for inflammation of the eyes, such as the application of raw meat, or of bread soaked in milk, or of cooked onions, or a bathing with urine, etc. Such remedies are well adapted to increase the inflammation and produce complications. [Involvement of the cornea is specially common in infection by the pneumococcus and in certain mixed infections. Pneumococcus infection quite often produces the very serious ulcus serpens corneæ (see § 217).—D.]

127. Etiology.—Acute conjunctival catarrh in the majority of cases is certainly produced by bacteria, whose multiplication in the conjunctival sac induces the inflammation of the mucous membrane. The source of the morbid germs varies. They may be transferred from a diseased to a healthy eye; that is, the catarrh may be produced by contagion. This is eminently probable in the case of epidemics occurring in the same family or the same house. [Infected handkerchiefs, towels, etc., may spread the disease; in other cases the transfer may be effected by flies.] At certain times, especially in the spring, when so many people are attacked by catarrhs of the air passages, by coryza, coughs, etc., conjunctival catarrhs, too, are generally present in especially great number and many patients with conjunctival catarrh also have at the same time a coryza, in the secretion from which not infrequently the same bacteria are demonstrable as in the conjunctival secretion. In that case the disease, apart from direct transfer, can be spread by particles thrown out in sneezing, coughing, etc. At such times real epidemics of conjunctival catarrh occur, and under these circumstances it is the violent form (ophthalmia catarrhalis) which is prevalent. [In Palestine, conjunctivitis, which occurs endemically during the winter months, assumes in the summer an epidemic form with marked aggravation of the symptoms. Both forms seem to be produced by the same causal agents (Koch-Weeks bacillus, pneumococcus, gonococcus), which, however, in summer receive an access of virulence (Butler).—D.] It is further possible that pathogenic germs which are sometimes already present in the normal conjunctival sac may spontaneously multiply so greatly as to excite inflammation. But in not a few cases of acute conjunctival catarrh the examination of the secretions for bacteria proves negative.

Acute catarrhal conjunctivitis, also called conjunctivitis simplex, may be caused by different kinds of bacteria. In the severe cases of ophthalmia catarrhalis, particularly in those which occur in epidemics, there is found as the cause of the trouble a very small bacillus described first by Koch, afterwards by Weeks (Fig. 45). Cases of less severity, particularly those which are associated with reddening at the angles of the lids (blepharitis angularis) [and which usually run a chronic course], are caused by the diplobacillus described by Morax and Axenfeld (Fig. 46). These two bacteria are the germs most often found. The pneumococcus occurs in cases of acute conjunctivitis in small children, rarely in adults. [It is more frequent in the Near East, where it often occasions epidemics.—D.] The streptococcus has been found in cases of catarrh with simultaneous disease of the tear sac; the influenza bacillus in epidemics of influenza; and the meningococcus in cerebro-spinal meningitis. [The micrococcus catarrhalis, which resembles the gonococcus in appearance and staining properties, occurs in some cases, especially of epidemic conjunctivitis, and the diplococcus samoensis is said to occasion

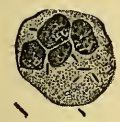


Fig. 45.

Fig. 46.

Fig. 45.—Koch-Weeks Bacillus. (After Weichselbaum-Müller.) Magnified 925×1. The bacilli are very small, as is shown by comparison with Fig. 46, which is drawn under a magnification only half as great. For the most part the bacilli lie upon the pus corpuscle; at a distance from this can be seen a chain of four bacilli.

Fig. 46.—Morax-Axenfeld's Diplobacillus. Magnified 480×1. In the coagulated secretion lies an epithelial cell and the nuclei of two pus corpuscles. The bacilli are long and thick and are connected either in pairs or in fairly long chains.

Samoan conjunctivitis.—D.] In a few cases the cause of the trouble seems to be the staphylococcus, the bacterium coli, the bacillus subtilis, Petit's diplobacillus liquefaciens, the pneumobacillus, [sarcinæ, and the bacillus tularensis (in squirrel-plague conjunctivitis). Petit's diplobacillus resembles the diplobacillus of Morax-Axenfeld, but differs in biologic reactions. It also differs in attacking the cornea primarily (Chaine), while with the Morax-Axenfeld diplobacillus involvement of the cornea, if it occurs at all, is secondary.—D.]

To the fact that the flora of the diseased conjunctiva is so manifold must be added the further fact that the bacterial finding also varies greatly with the time and place. [For example, the Koch-Weeks bacillus is frequent in New York and apparently much less so in Chicago, where, on the other hand, the Morax-Axenfeld bacillus appears to be common.—D.] Moreover, even the normal conjunctival sac not infrequently contains pathogenic germs (page 30).

While the infection which, in all probability, excites the catarrh of the conjunctiva comes to the latter from without in most cases, there are also instances in which a poisonous principle circulating in the blood causes the conjunctival inflammation. This is the case in the conjunctival catarrh which accompanies measles, and indeed often forms the first prominent symptom of it (see § 164 [and compare also remarks on metastatic gonorrhœal conjunctivitis (page 156)]).

In a high degree dependent upon atmospheric influences is that form of acute conjunctivitis which accompanies *hay fever*. This affection, which is pretty frequent in certain countries, attacks individuals who are predisposed to it in the beginning of

the summer, and makes itself apparent by fever and also by violent catarrhal inflammation of the conjunctiva and of the air passages. It is probably the result of the direct action of a poisonous substance which is contained in the pollen grains of many gramineæ.

128. Therapy.—By suitable treatment the duration of a conjunctival catarrh can be considerably shortened and the development of a chronic catarrh prevented. The sovereign remedy in all the more intense cases of catarrh is the cauterization of the conjunctiva with *nitrate of silver*. This should come into contact with the conjunctiva only, and not with the cornea. In applying it, we evert the lids so that their conjunctival surface looks forward. The latter is then brushed over with a 2-per-cent solution of silver nitrate and the excess of the solution is quickly washed off with lukewarm water or with a weak solution of salt. We now find the surface of the conjunctiva covered with a delicate bluish-white pellicle. This is the superficial slough which the solution has produced. The immediate result of this procedure, which is called painting the conjunctiva, is violent burning and marked irritation of the eye, an increase, in short, of all the inflammatory phenomena (stage of exacerbation). After this has lasted from a quarter to half an hour, according to the energy of our application, improvement gradually sets in. An examination of the eye at this time shows that the thin slough is separating and is being thrown off in the form of shreds. When this is completed, we find the eye paler and the patient feels relieved and much less annoyed by his catarrh than was the case before the application of the brush (stage of remission). This improvement lasts from half a day to a day, according to the intensity of the catarrh. Then the troubles gradually increase again (recrudescence). This is a signal for repeating the application. As a rule, it is sufficient to make the application once a day, and best in the morning.

Beginners must particularly avoid making the application too energetically. If this has been done, the pain that follows the application lasts unusually long (for hours), and we find that even after a pretty long time, indeed even on the following day, the slough is still adherent in places. This is a proof that the sloughing process has penetrated too deeply. If, in spite of this, we should repeat the application, we would produce a progressively deeper and deeper sloughing of the tissue, and increase the inflammation instead of curing it. We must omit the application, therefore, as long as the slough is still adherent to any part of the conjunctiva.

[For silver nitrate may be substituted with advantage one of the organic salts of silver, especially argyrol and protargol. *Argyrol* is said to have no bactericidal action, yet clinically it has proved its efficacy, particularly when used frequently and in strong solutions. The surgeon may apply it with a dropper or with a swab in 30- or even 50-per-cent strength, and the patient may drop in a 10- to 20-per-cent solution 2 to 6 times daily. Some have thought that the best results are secured by keeping the con-

junctiva continually flooded with the solution (immersion method—Bruns). So far from having a cauterant or even irritative effect, it is distinctly sedative, relieving irritation in acute conjunctivitis.—D.]

[*Protargol* is more irritating than argyrol, but not as a rule excessively so. The surgeon may apply it once a day in 10- to 33-per-cent strength, and in addition a 5-per-cent solution may be instilled from 2 to 5 times a day. Clinical tests show that protargol is more efficient than argyrol and when applied in strong solution is more efficient than even nitrate of silver (Butler). It is distinctly bactericidal.—D.]

[In conjunctivitis due to the pneumococcus good results are obtained from *ethylhydrocupreine* (*optochin*). A freshly prepared 2-per-cent solution of the hydrochloride may be applied with a cotton-tipped applicator or a 1-per-cent solution may be dropped into the eye every hour or two.—D.]

For less severe cases of acute catarrh (especially for those produced by the Morax-Axenfeld diplobacillus) *zinc sulphate* is used. This is instilled once a day in ½-per-cent solution. [It is sometimes effective in ¼- or ⅕-per-cent solution, and is often ordered to be instilled two or three times a day. On the other hand, not a few cases require the use of 1- or occasionally even a 2-per-cent solution. In this strength it is almost a specific for cases of diplobacillus infection.—D.] This method of treatment has the advantage over the application of the silver solution in that it can be employed in the case of patients who are not able to visit the physician every day.

[*Corneal ulcers* complicating conjunctivitis are treated like corneal ulcers in general, but if there is much secretion a bandage should be avoided, as, in fact, it should be in conjunctivitis under any condition.—D.]

When the acute stage of catarrh has started to decline, we change to the use of *astringent collyria*, such as are employed for chronic catarrh, for which see page 150. For the agglutination of the lids during the night we order an ointment (of boric acid or of white precipitate) to be rubbed at bedtime upon the borders of the lids—the eyes being closed.

In addition to our medicinal treatment of conjunctival catarrh, we must not forget to enjoin upon the patient general *hygienic* rules; telling him to keep the eye clean by washing it with lukewarm water, and to avoid smoke, dust, and bad air in general, and recommending him instead to pass his time in the open air. He must also refrain from straining the eyes much, especially in the evening by artificial light. In consideration, too, of the possibility of spreading the disease by contagion, the patient must take care not to use the same wash basin, towels, etc., as other people. [Physicians and nurses handling many cases of acute conjunctivitis should be careful to keep their hands clean and protect their own eyes from any germ-carriers (soiled handkerchiefs, flies). Where but one eye is affected it is well, following Butler's suggestion, to drop a 5-per-cent solution of protargol into the other as a prophylactic.—D.]

Nitrate of silver, our most important remedy in catarrh, was first employed for inflammations of the conjunctiva by St. Yves in the eighteenth century, although it was in the last century that it first found general acceptation. People had a natural dread of instilling so irritating a liquid as a nitrate-of-silver solution into a violently inflamed eye. In fact, in a perfectly sound eye this solution excites violent irritation of the conjunctiva, and it is quite possible to produce an artificial catarrh by too frequent application of it. How then does it happen that the nitrate-of-silver solution has such a beneficial action in conjunctival catarrh? The delicate bluish-white pellicle which covers the conjunctiva directly after the application is due to coagulation of the albumin of the cells in the upper layers of the epithelium by the nitrate of silver, so that these layers become opaque and die. The escharotic process acts like an irritant which increases the existing hyperæmia. This not only gives rise to an increase of the annoyance suffered (exacerbation), but also induces a transudation under the eschar, so that the latter is loosened and finally cast off. But when this takes place the micro-organisms contained in the upper layers of the epithelium are thrown off with the eschar and so eliminated from the eye.

The silver solution finds an extensive application not only in catarrh, but also in other affections of the conjunctiva. In regard to it the following hints may be laid to heart: (*a*) Many physicians apply weaker or stronger solutions according to the effect which is to be obtained, but we can always succeed with a 2-per-cent solution, since we have it in our power to regulate the effect by making a light or penetrating application. The application is made with wooden toothpicks wound at one end with cotton, which, acting like a brush, takes up the silver solution. The toothpick is thrown away, after being used once. [(*b*) Too frequent or too energetic application not only causes sloughing of the conjunctiva with the formation of permanent cicatrices but in infants may cause profuse and persistent hæmorrhage.—D.] (*c*) The application should not be made at night, because the secretion, which is poured out more abundantly after the application, would be retained in the conjunctival sac by the closure of the lids in sleep. For the same reason the eye ought not to be bandaged immediately after the application. (*d*) Corneal ulcers do not constitute a contraindication for making the application; on the contrary, they furnish a direct indication for it, in case they prove to be catarrhal ulcers. Only still greater care than would otherwise be necessary must be taken to prevent the caustic from coming into contact with the cornea. (*e*) If the treatment of the conjunctiva with the silver solution is kept up too long (for some months or a year), there is produced little by little a dirty-gray coloration of the conjunctiva, which never afterwards disappears. This phenomenon, called *argyrosis* or argyria,[5] is caused by the fact that silver is deposited in the form of an oxide or an albuminate in the tissues of the conjunctiva (in its elastic fibers), and can never afterwards be removed. Argyrosis is produced even more readily by the constant instillation of the silver solution than by the application of the brush, since in the former case the excess of the solution is not removed by being washed off but remains in the conjunctival sac. This coloration of the conjunctiva is also observed when the conjunctiva is constantly exposed to the action of silver dust, as occurs, for example, in many of those who work in silver.

The organic silver salts may cause argyrosis in the same way as the nitrate if their use is kept up too long [for two or three weeks].

[One of the great advantages of both *argyrol* and *protargol* over silver nitrate is that, being non-irritating, they can be applied at any stage of a conjunctival inflammation—for example, before suppuration has set in—and that their administration can be largely intrusted to the patient himself. Another advantage over silver nitrate and also over indifferent detergent solutions is that both argyrol and protargol penterate into all the recesses of the conjunctival sac, lodge in them a long time (so as to produce a continuous

[5] From ἄργυρος, silver.

action), and as they gradually exude drive before them the secretion with its contained bacteria. They also force their way down the nasal duct, and help to keep the latter pervious, thus preventing stagnation of secretions and consequent multiplication of bacteria in the conjunctival and lachrymal sacs.—D.]

[The secretions mixed with argyrol form coagula, which sometimes act like foreign bodies. Hence before instilling argyrol it is well to flush out the eye first with an indifferent (saline or boric) solution in order to remove all the secretion possible, and a few minutes after the argyrol has been instilled wash the eye out again in order to remove the coagula. After this another drop of argyrol should be instilled and left in.—D.]

Acetate of lead is sometimes used in conjunctivitis, but should never be employed, since it produces an intensely white, very disfiguring opacity (*lead incrustation*), wherever the cornea is denuded by ulceration or erosion.

(*b*) *Conjunctivitis Catarrhalis Chronica*

129. Symptoms.—In chronic conjunctival catarrh the changes *objectively* perceptible are on the whole but slightly pronounced. A moderate degree of redness of the conjunctiva is present either over the tarsus alone or in the retrotarsal fold also. The conjunctiva is smooth and not swollen; it is only in old cases that hypertrophy with thickening and a velvety appearance of the conjunctiva is developed. The secretion is scanty and makes itself chiefly apparent by a gluing together of the lids in the morning. The whitish scum often found at the angles of the lids is produced by the lachrymal fluid being beaten up with the secretion of the Meibomian glands into a sort of a foamy emulsion, as a result of the frequent blinking of the lids. The constant moistening of the skin at this spot often leads to the formation of excoriations. In many cases the secretion, instead of being increased seems even to be diminished. In view of the fact that there is little or no increase in the secretion, several authors call many of these cases not by the name of chronic catarrh, but by that of hyperæmia of the conjunctiva.

In proportion to the insignificance of the objective symptoms, the greater is the attention that has to be paid to the complaints made by the patient—in fact, the *subjective* symptoms are generally so characteristic that the diagnosis of chronic conjunctival catarrh can easily be made from them alone. The discomfort of the patient is usually greatest at night. The heaviness of the lids, scarcely noticeable in the daytime, becomes at night so marked that the patient has difficulty in keeping the eyes open; he has the feeling of being sleepy. An annoying sensation of there being a foreign body—like a speck of dust—in the eye, is produced by the scanty secretion which remains in the conjunctival sac in the form of mucous filaments, and if these filaments lie upon the cornea the sight is interfered with, or rainbow colors make their appearance about a candle flame when looked at. Further unpleasant sensations of various kinds are described, as, for example, that the eyes burn and itch; that they are dazzled by the light; that, moreover, they are tired out quickly by working; that they blink often, etc. In the morning the lids are somewhat stuck together, or a little yellowish dried

secretion is found to have collected in the inner angle of the eye. In other cases there is an annoying sensation of dryness, and the eyes can be opened only with difficulty, the patient, at the same time, having the feeling as if the lids were stuck to the eyeball because of the lack of moisture (*catarrhus siccus*). These troubles, so various in their nature, do not always by any means bear any definite relation to the objective conditions. We see the conjunctiva quite intensely reddened in many people without their complaining in the least; while in others, who do nothing but annoy the physician with their expressions of discomfort, there are often scarcely any changes perceptible in the conjunctiva.

130. Course.—Chronic conjunctival catarrh is one of the most frequent of ocular diseases, chiefly affecting adults, and especially persons somewhat advanced in age. In old people it is almost the rule to find a light grade of chronic conjunctival catarrh, which is denominated senile catarrh. The duration of conjunctival catarrh is ordinarily a long one; many people suffer from it for a great part of their lives. The disease can lead to *complications* which in part produce irreparable changes. Among the most frequent complications is inflammation of the edges of the lids (blepharitis), resulting from the frequent wetting of the palpebral margins by the copiously secreted tears. As a further consequence of this wetting with the tears, the skin of the lower lid is attacked with eczema or becomes rigid and contracted, so that the free edge of the lid is no longer in perfect apposition with the eyeball. As a result of this the punctum lacrimale no longer dips into the lacus lacrimalis, so that the transportation of the tears into the lachrymal sac is impeded, the epiphora increased, and thus again a still further injurious reaction upon the character of the skin is produced. In this way there is formed a vicious circle, which leads to a constantly increasing depression of the lower lid (ectropion). This outcome is still further promoted by the circumstance that the patient keeps wiping away the overflowing tears, and thus makes with the handkerchief stroking movements from above downward, by which the lower lid is drawn down. If the contraction of the skin of the lids which have been moistened by the tears is more pronounced in the horizontal direction, blepharophimosis is developed (§ 613). Lastly, small ulcerations of the cornea are among the frequent products of catarrh.

131. Etiology.—The causes which lie at the foundation of chronic catarrh are: 1. A preceding acute catarrh, which, instead of healing completely, passes into the chronic stage. 2. General injurious influences of various kinds. Chief among these is bad air, vitiated by smoke, dust, heat, the presence of many people, etc. Workers in factories where there is a great deal of dust, waiters in inns that are filled with smoke, etc., very frequently suffer from chronic conjunctival catarrh. Going late to bed, staying awake at night, and the immoderate use of alcoholic beverages are additional pre-

disposing factors. Persons who already suffer from chronic conjunctival catarrh find that the latter is made considerably worse after the action of any injurious influence of this sort—for example, after an evening spent at the theater or in a smoky place. So also the constant action of the wind and bad weather frequently causes catarrh in farmers, coachmen, etc. For the same reason, too, eyes which are very prominent (goggle eyes), or whose lids are retracted (lagophthalmus), are attacked by catarrh, because they are too little protected against the air. The effect which constant contact with the air exerts upon the conjunctiva is best shown in ectropion, in which the conjunctiva tarsi, where it lies bare, becomes very much reddened and thickened, and velvety or even covered with large prominences. The conjunctiva bears continued exclusion from the air as little as it does constant contact with it, on which account chronic catarrh sets in when bandaging of the eye is kept up for a long time. 3. Excessive straining of the eyes, especially in hypermetropic or astigmatic persons, can result in chronic catarrh. 4. Local injurious influences. Here belongs irritation of the conjunctiva by foreign bodies lodging in the conjunctival sac, among which, using the term foreign bodies in the wider sense of the word, are to be reckoned cilia which are turned in toward the eye. In most cases the local injurious influence consists of some other disease of the eye, that induces catarrh as a sequela, as, for example, blepharitis or infarction of the Meibomian glands. Accumulation of the tears, as a result of disease of the tear sac, or because the punctum lacrimale does not dip properly into the lacus lacrimalis, is a frequent cause of catarrh, so that we should never forget to look for an infection of the tear passages in unilateral catarrh. We say unilateral, for catarrh produced by local causes is distinguished from that due to general injurious influences in this respect, that the former is very frequently unilateral, while in the latter, from the nature of the case, both eyes are generally affected.

In many cases of chronic catarrh there is found in the scanty secretion as the exciting cause of the inflammation the diplobacillus of Morax-Axenfeld, which, on page 143, was noted as the cause of acute conjunctivitis, but which much more often causes a catarrh that is chronic from the start. These cases are often distinguished by a marked redness of the skin at the angles of the eye [whence the name of *angular conjunctivitis*. The symptoms are persistent itching and burning and sometimes an obstinate asthenopia].

132. Therapy.—It is clear that the treatment must first of all pay regard to the causal factor by regulating in a suitable way, as far as is compatible with the patient's calling, the general conditions under which he lives, and by removing all local causes of catarrh that may be present, etc. For the treatment of the conjunctiva itself we first employ, as we do in acute catarrh, the *nitrate of silver*, which is applied by the brush in 1- or 2-per-cent

solution. We make use of it in those cases only in which the catarrh is accompanied by rather abundant secretion and by relaxation of the conjunctiva—that is, in periods of acute exacerbation, such as frequently occur in the course of every chronic catarrh—and use it, furthermore, when hypertrophy of the conjunctiva has already set in. Otherwise we succeed better with *astringent collyria*, which the patient can instil himself. The most usually employed of these is zinc sulphate, which is instilled in ½-per-cent [or 1-per-cent] solution once or twice a day, and is especially indicated in cases of diplobacillus conjunctivitis [see page 145]. In the chronic catarrh of elderly people with marked reddening of the conjunctiva but no secretion, the most suitable remedy is the collyrium adstringens luteum,[6] which is not ordinarily prescribed undiluted but mixed with an equal quantity of water. Other astringents are lapis divinus[7] (½ per cent), alum[8] or tannin (1 per cent), boric acid (3 per cent).

The order in which these collyria are here arranged about corresponds to their gradation in activity from the strongest to the mildest. They should be instilled once or twice a day, but not at night. So many of them are enumerated, because it is good to have a pretty large number to select from, since as the catarrh is of long duration, a change will have to be made pretty often in the remedies. Every remedy, if too long applied, loses its activity, since the conjunctiva grows accustomed to it. For the sticking together of the lids, as well as for any excoriations that may be present, an ointment of boric acid (2 per cent) or of white precipitate (½ to 1 per cent) may be rubbed upon the closed lids at bedtime.

(c) *Conjunctivitis Follicularis*

133. Follicular catarrh is characterized by the presence of *follicles*. These are small round granules of about the size of a pin's head which lie in the region of transition of the conjunctiva. They are of a pale, translucent aspect and puff up the conjunctiva in the form of small eminences. Either a few follicles only or many are present; in the latter case they are ordinarily arranged in rows like the beads of a rosary. The upper retro-

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[6] This collyrium, called also Horst's eye water, is at present no longer official in most countries, yet it is of the greatest service and in many cases can be replaced by no other. According to the Austrian Pharmacopœia, it is to be prepared in the following way:

Take of ammonium chloride 50 centigrammes and zinc sulphate 125 centigrammes, dissolve in 200 grammes of distilled water and add a solution of 40 centigrammes of camphor in 20 grammes of dilute alcohol and 10 centigrammes of saffron. Digest for twenty-four hours with frequent agitation, and filter.

Rather stronger in action is the tinctura opii crocata, diluted with an equal amount of water and filtered. [The tinctura opii crocata is a solution of opium and saffron, about 9 per cent each in cinnamon water.—D.]

Romershausen's eye water, which is also frequently employed in chronic ophthalmic catarrh, consists of a mixture of aqua fœniculi and tinctura fœniculi.

[7] [Or aluminated copper; a preparation made by fusing together 32 parts each of copper sulphate, potassium nitrate, and alum, and adding a mixture of 2 parts each of camphor and alum.—D.]

[8] Alum is more often applied in the solid form, as a sharpened crystal (*alum stick*) with which the conjunctiva is stroked. *Gallicin* is also employed, being sprinkled in fine powder with a brush upon the conjunctiva. In the sensation of burning in the eyes cool lead-water compresses are serviceable. We prescribe the liquor plumbi acetici [a 14-per-cent solution of lead acetate]; of this 20 to 30 drops are given in a small glass of cold water. Compresses made with this mixture are applied to the closed eyes for 10 to 15 minutes two or three times a day.

tarsal fold usually contains fewer follicles than the lower. Microscopic examination shows that the follicles, as well as the so-called trachoma granules, consist of a circumscribed accumulation of adenoid tissue (Fig. 51, *T*).

Follicles are most frequently observed in youth, and especially among pupils in day schools, boarding schools, etc. In many school children the disease exists in a perfectly latent form, as, in spite of there being a considerable number of follicles, the conjunctiva is not reddened and causes no symptoms of any kind, so that the trouble is discovered only by medical examination. In such cases the follicles often persist for years.

In other cases the disease has an *acute* character; a conjunctivitis sets in with the symptoms and disagreeable accompaniments of a violent catarrhal ophthalmia and with the simultaneous development of numerous follicles. This acute form often occurs epidemically and is more obstinate than a catarrhal ophthalmia that is not complicated with follicles. On the other hand, the follicles do not last as long as in the chronic form, but soon disappear when the conjunctivitis abates. Intermediate between the very chronic and the acute form are the particularly numerous cases in which the presence of follicles is combined with slight irritation of the conjunctiva and with correspondingly mild symptoms such as obtain in a chronic conjunctival catarrh.

The *treatment* of follicular catarrh is directed mainly at the accompanying inflammatory symptoms on the part of the conjunctiva. If these are altogether absent, no treatment at all is needed, as ultimately the follicles disappear of themselves, leaving no trace of their presence. If there are very many follicles present, we had best order the inunction of lead ointment (lead acetate 0.1 gm. to 5.0 gm. of fatty matter) into the conjunctival sac. In doing this it must not be forgotten that the presence of corneal ulcers contraindicates most absolutely the use of the lead ointment. [Ointment of copper sulphate (1–1,000) may also be used.—D.] If the presence of follicles is associated with inflammatory changes in the conjunctiva, the latter are to be treated according to the principles laid down for catarrhal conjunctivitis—that is, by the application of a 2-per-cent nitrate of silver [or a 10- to 25-per-cent solution of protargol or argyrol] if the inflammation is an active one, and in the milder cases by the instillation of a solution of zinc sulphate (½ per cent). As in catarrh of all kinds, so particularly in follicular catarrh, living in fresh, pure air is to be enjoined.

The *prognosis* of follicular catarrh is altogether good, as the disease leads to no complications and leaves no permanent changes in the eye whatever; in this being opposed to trachoma, to which follicular catarrh has some resemblance (see § 148).

The fact that follicles may at one time be associated with intense inflammation of the conjunctiva and at another time may not cause the slightest evidences of irritation

is probably to be explained as follows: The follicles are a circumscribed new formation of lymphoid tissue, this new formation being the way in which the conjunctiva reacts to certain irritants. Lymph follicles occur in many mucous membranes, and even in the conjunctiva they are normally present in many of our domestic animals, though not in man. The injurious influence which induces the formation of follicles in man is not necessarily of a bacterial nature, for in many men we see a conjunctival catarrh with numerous follicles develop after the continued instillation of an atropine solution even when it has been carefully sterilized (*atropine catarrh*) (see §323). In the chronic cases of follicular catarrh the irritation probably originates in the impure air of the school room or in similar injurious influences and causes simply a gradual development of follicles, but not any inflammatory changes in the conjunctiva. In that case, to be sure, the follicles, if they are numerous, may in their turn induce slight evidences of irritation in the conjunctiva. In the cases of acute follicular catarrh, however, there is probably a bacterial infection, such as would under other conditions simply cause a catarrhal ophthalmia but in this case produces in addition a rapid development of follicles.

II. Conjunctivitis Gonorrhoica

134. *Conjunctivitis gonorrhoica*, also called acute blennorrhœa,[9] is an acute inflammation of the conjunctiva, which originates in contagion from gonorrhœal virus, and whose copious purulent secretion is likewise contagious in its action. The carriers of the contagion are gonococci, which are found both in the pus secreted by the conjunctiva and also in the most superficial layers of the conjunctiva itself. They are mostly arranged in pairs, as diplococci, and as a rule lie together in heaps. Fig. 47 shows a specimen taken from the secretion of gonorrhœal conjunctivitis. In it are seen the heaps of gonococci, partly free (*a*), partly upon and within the cells, which are either pus cells (*b*) or cast-off epithelial cells (*c*).

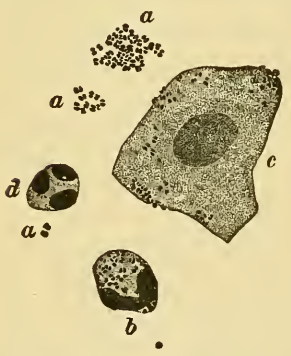

Fig. 47.—Secretion of Acute Blennorrhœa with Gonococci.

Gonorrhœal conjunctivitis occurs both in adults and in new-born infants. The following description refers to the disease in adults; the disease as it occurs in the new-born will be treated of under the head of ophthalmia neonatorum:

[In adults true gonorrhœal conjunctivitis is not a very common affection. According to White, it occurs but once in every seven or eight hundred cases of gonorrhœa.

It must be observed that the micrococcus catarrhalis and the meningococcus, which resemble the gonococcus in morphology and staining, and differ only in biological characters, can also cause an acute purulent conjunctivitis. Since these organisms cannot be differentiated in the smears, but only in cultures, a number of cases of conjunctivitis have doubtless been classed as gonorrhœal, which were not. Cf. remarks on Extragenital Gonorrhœa, page 156.—D.]

[9] From βλέννα, mucus, and ῥέω, I flow.

135. Symptoms and Course.—When infection has taken place, the disease breaks out after a certain period of incubation, the duration of which varies, according to the intensity of the contagious action, from a few hours up to three days. The lids grow red, become hot, and are swollen with œdema, generally to such an extent that the patient can no longer open them, and even the physician often has trouble in separating them far enough from each other to bring the cornea into view. The conjunctiva of the lids and of the retrotarsal fold is intensely reddened and greatly swollen. The swelling is produced by an abundant cellular infiltration of the conjunctiva, which is consequently tense, and has a granular, uneven surface. This feature of acute blennorrhœa serves to distinguish it from catarrh, in which even in the severe cases the swelling is rather of a serous nature, and hence the conjunctiva is yielding and has a smooth surface. The conjunctiva of the eyeball shows a like tense swelling, which stops short at the corneal margin, so that a raised wall is thus formed about the more deeply placed cornea (chemosis). The secretion produced by the conjunctiva is like meat juice—that is, it is a serum which is colored red by admixture with blood, and in which float some flakes of pus. The eye is uncommonly sensitive to contact, the lymphatic gland in front of the ear is swollen, the patient has slight fever.

Ordinarily it takes from two to three days for the disease to mount from its initial point to the pitch just described, and at this pitch it is maintained for two or three days more. This period is designated as the first stage, or *stage of infiltration*. Succeeding this as a second stage is that of *pyorrhœa*. The swelling of the lids gradually diminishes, a fact which we recognize principally by means of the return of the small wrinkles of the skin of the lids, and the tense infiltration of the conjunctiva slowly retrogrades. Simultaneously with this there begins a very profuse secretion of pus, which trickles out continually from the palpebral fissure; hence the name pyorrhœa, or flow of pus. In the further course of the disease the conjunctiva keeps getting less and less swollen all the time, and in many cases returns by degrees to the normal within four or six weeks. In most cases, however, a condition of chronic inflammation of the conjunctiva remains, which is designated as the third stage of the disease, the stage of *chronic blennorrhœa*. In this period the lids are no longer swollen. The conjunctiva is reddened and thickened, especially upon the tarsus, where its surface looks uneven, granular, or velvety. The retrotarsal fold forms an ungainly swelling; the conjunctiva of the eyeball, which shows hyperæmia only, is the least changed. After this state of conjunctival hypertrophy has abated, a process which usually takes months for its accomplishment, there usually remain slight, but permanent cicatrices of the conjunctiva.

The description here given corresponds to the cases of most frequent occurrence, which are those of medium intensity. In addition, both light

and also very severe cases of the disease come under observation which exhibit rather different features. In the light cases, which we are accustomed to call *subacute blennorrhœa*, all the inflammatory changes are less, and the changes are limited chiefly to the conjunctiva of the lids. Frequently such cases are not to be distinguished with certainty by their external aspect from severe catarrh. The diagnosis can be rendered certain by the microscopic examination of the secretion, since by it the presence or absence of gonococci is demonstrated.

In the *severest* cases, the infiltration of the conjunctiva is so great that the latter in places appears no longer red, but grayish-yellow, because, as in diphtheria of the conjunctiva, the vessels are compressed by the bulky exudation, and the conjunctiva is thus rendered anæmic. The conjunctiva forms about the cornea a tense grayish-red wall. Quite often the surface of the conjunctiva is found to be covered with a clotted exudate, or croupous membrane.

136. Complications.—The most dreaded complication of acute blennorrhœa is the *involvement of the cornea*, by which, in many cases, incurable blindness is produced. At first the cornea becomes dull upon its surface and covered with a slight diffused opacity. Then circumscribed infiltrations of grayish color make their appearance, which soon become yellow and break down into ulcers. These infiltrations may be situated at the margin of the cornea, and give rise to speedy perforation of the latter. This is a comparatively favorable result, as, after the perforation has taken place, the purulent infiltration of the cornea not rarely is brought to a standstill, and so a portion of the cornea is preserved. But it can also happen that the marginal infiltrations become rapidly confluent, and unite into a yellow ring surrounding the entire cornea (a so-called annular abscess). In that event the cornea is lost, for this ring soon spreads over the entire cornea and destroys it. In other cases the purulent disintegration occurs first in the middle of the cornea. When, in one way or another, the cornea has gone either entirely or in part to destruction, the outcome is either a formation of cicatrices with incarceration of the iris, or it may even be a panophthalmitis. Since these sequelæ are observed after every destruction of the cornea, even when due to other causes, they will find detailed description under the diseases of the cornea.

Involvement of the cornea is the more certain to take place, the severer the conjunctivitis, and, in particular, the more pronounced the participation of the conjunctiva bulbi in the inflammation. In the severest cases with tense chemosis the cornea is always affected, and is, as a general thing, irretrievably destroyed. In the cases of moderate severity when the chemotic swelling of the conjunctiva is less pronounced and especially is less hard, it is usually possible to preserve the cornea, either entirely or in great part, inasmuch as the ulcers that develop, even if they

are attended with perforation, are of but small size. In the lightest cases, where the process is limited to the palpebral conjunctiva, there is, on the whole, little danger to the cornea.

The severer the course of the inflammation, the earlier the involvement of the cornea sets in; in violent cases, the cornea is already clouded by the second or third day. Sometimes corneal ulcers are not developed until late in the disease, when the conjunctivitis is already well on the retrograde path. These late affections of the cornea are not very dangerous, and it is generally possible to check them readily.

That the lymph gland in front of the ear should swell up in acute blennorrhœa is a fact that accords with the virulent character of the latter; sometimes even suppuration of this gland has been observed (bubo præauricularis).

The *inflammation of the cornea* is to be referred to infection of the latter by the secretion which constantly bathes it and macerates its epithelium. Since the secretion collects chiefly in the gutter lying at the rim of the cornea, between the latter and the steep slope of the chemotic conjunctiva, the purulent infiltration most frequently begins here, too. The tense infiltration present in this chemotic wall of conjunctiva is to be regarded as a second factor in the production of corneal trouble. This leads to obstruction of the circulation in the marginal loops of the cornea, and thus interferes with the nutrition of the latter. Hence, the more pronounced and the more tense the chemosis, the more confidently is an affection of the cornea to be anticipated. It is in harmony with this fact that, in cases where the chemosis is unequally great, we often see the involvement of the cornea take place first at that portion of the corneal rim where the chemosis is the greatest.

Since infection of the cornea is certainly very greatly favored by the existence of gaps in the epithelium of the latter, we must avoid injuring the epithelium of the eye by carelessness in cleansing.

If gonorrhœal conjunctivitis happens to affect an eye which is covered with pannus the latter will afford the cornea a secure protection against suppuration. Nay, more, it is often apparent, after the violence of the inflammation has passed, that the pannus has cleared up considerably, so that in cases of old pannus inoculation with gonorrhœal conjunctivitis has been designedly performed. This procedure has now been abandoned in favor of the treatment with jequirity.

137. Prognosis.—The prognosis of the disease results from what has been said, it being essentially founded upon the condition of the cornea. This is dependent upon the intensity of the inflammation of the conjunctiva bulbi, in accordance with which, therefore, the prognosis must be made.

[The prognosis in all genuine cases must be regarded as serious, although with improved methods of treatment it is better than it used to be. According to Hirschberg, 11 per cent of present-day cases retain fair vision and only 17 per cent become blind (as against 50 per cent formerly).—D.]

138. Etiology.—Acute blennorrhœa is produced simply and solely by infection. The poison can be introduced into the eye from the genitals directly, generally because an individual (whether man or woman) affected with gonorrhœa touches the eyes with unclean fingers after these have

been in contact with the genitals. The infection, however, can also come from an eye affected with blennorrhœa. If, for instance, one eye is already diseased and is affected with profuse suppuration, the other eye can also be infected by a transfer of the secretion to it. An individual with an eye diseased with blennorrhœa can infect the persons who are nursing him or any others who may share his room.

[In Palestine and Egypt a good many cases of acute conjunctivitis of virulent character, and often causing destruction of the cornea, are due to diplococci which in smears appear identical with the gonococcus. As genito-urinary gonorrhœa is extremely rare in these countries, direct infection from the urethra or vagina is unlikely, and these cases have been regarded as due to *gonococcus infection of extragenital origin* (Butler, Ticho, and others). Since, however, the gonococcus cannot be differentiated in smears from other diplococci (micrococcus cartarrhalis, meningococcus), which may cause acute conjunctivitis, this diagnosis is doubtful, and until further proof is adduced we must still hold that true blennorrhœa is derived from the genito-urinary tract. In acute cases, as stated above, this infection is usually direct.—D.] Nevertheless, there are cases in which a conjunctival inflammation of a slighter kind is connected with a gonorrhœa in the way of *metastasis*, just as arthritis and iritis sometimes complicate a gonorrhœa. This metastatic mode of origin is to be understood by supposing that the gonorrhœal poison has got into the circulation, and is exciting inflammation in remote organs which have a predisposition for this poison. A conjunctivitis originating in this way shows the characters of a severe catarrhal conjunctivitis, with a dull injection of the eyeball like that which occurs in scleritis. On the other hand, there is no infiltration of the conjunctiva, such as occurs in acute blennorrhœa, and the secretion also is not so profuse nor so purely purulent as in acute blennorrhœa. As a rule, gonococci are not present in the secretion.

[Metastatic conjunctivitis is said to be from ten to twelve times as frequent as the non-metastatic form. It may be the sole expression of a systemic gonorrhœa, but usually coexists with some other evidence of gonorrhœal metastasis (in the joints or iris). It is probably due rather to the gonococci themselves than to their free toxins. The symptoms are those of an acute bilateral catarrhal conjunctivitis; the discharge is slight and mucoid. The conjunctivitis lasts 1 to 7 (usually 2) weeks, and relapses may occur. It is frequently complicated by a keratitis, usually bilateral and symmetrical, with multiple superficial infiltrates, or by irido-cyclitis (White, Byers).—D.]

As gonorrhœa of the urethra can by metastasis excite conjunctivitis, so also conversely cases have been observed in which a gonorrhœal arthritis, where gonococci have been demonstrated to exist in the pus, has arisen by way of metastasis from a blennorrhœa of the conjunctiva. Cases of this sort have been known both in adults and in new-born children affected with blennorrhœa (Deutschmann and others).

The secretion containing gonococci is usually brought into the eye by means of dirty fingers. Sometimes, however, a *direct transfer* from the diseased mucous membrane to the sound one is observed; for example, when a drop of secretion spurts into the eye of the physician or the attendant while cleansing genitals that are affected with gonorrhœa, or even when cleansing the eye of a patient affected with blennorrhœa.

I have repeatedly seen cases in which a patient, because of a mild conjunctival catarrh, washed his eyes in his own urine (a popular remedy among the laity in many places); as he had gonorrhœa, he thus got a gonorrhœal conjunctivitis. Gonorrhœal conjunctivitis has also been seen to originate from the use of another household remedy —that is, from the practice of laying upon the eye a piece of placenta, which in this case came from a woman affected with gonorrhœa.

If one eye is already infected, the transfer to the other is often brought about by secretion of the diseased eye flowing over the bridge of the nose into the sound eye during sleep. Furthermore, the secretion can be transferred from the eye affected with blennorrhœa to the sound one by the finger, the water used for washing, the sponge, the handkerchief, etc.

The transfer of blennorrhœa from an eye affected with the latter to the eyes of other people is likewise not rare. It occurs most frequently in children who are affected with ophthalmia neonatorum (§ 141), and thus infect their mothers, nurses, etc. In the Vienna Foundling Asylum, during the years 1812 and 1813, there were, for every hundred infants affected with blennorrhœa, more than fifteen nurses so affected, who had caught their eye disease from the infants. I have seen a whole family infected with blennorrhœa by a child having blennorrhœa neonatorum, and thus plunged in the greatest misery. Great caution on our own part, therefore, and, what is more important, careful instruction of the laity are here imperatively required.

We sometimes also observe *acute purulent conjunctivitis in small girls* of the age of two to ten years, who at the same time are troubled with a vaginal discharge (Arlt). Here are we still dealing with contagion from a virulent vaginal catarrh? Or is the vaginal discharge of these girls a benign catarrh caused by scrofula, anæmia, and the like? In some of these cases it has been possible to prove the origin of the vaginal blennorrhœa. The children have acquired the latter from their mothers or from other women about them, who were suffering from virulent vaginal catarrh, and had transmitted the latter by soiled clothes, sponges, baths, etc., to the children (Hirschberg). In other cases, the children had been raped by individuals affected with gonorrhœa. Here, therefore, we are dealing with a pure vaginal gonorrhœa in the children, and, accordingly, it is possible in such cases, too, to demonstrate the presence of the gonococcus both in the secretion of the vagina and in the conjunctiva as well (Widmark). But it would be going much too far to regard the vaginal discharge in little girls as true gonorrhœa in all cases in which infection of the conjunctiva results from the discharge. Here, just as in the case of ophthalmia neonatorum, a non-virulent, simple catarrhal secretion of the vagina is in position to excite an inflammation of the conjunctiva, which in this instance runs a less severe course. The distinction from a true blennorrhœa could be made in this case only by the microscopical examination of the secretion for gonococci.

The interesting researches of Piringer have instructed us in regard to the *relation between the infective material and the ophthalmia produced by it*, as he has made a great number of intentional transfers of virus (generally in the eyes of people already blind, who were paid for the experiment). He found that the more violent the blennorrhœa that serves as the source of the material inoculated, the shorter is the period of incubation. The infective power of the secretion is weakened by various influences, as by dilution with water—by dilution to the 1-tenth strength any secretion can be rendered inert—or by drying. Secretion that has been dried upon a piece of linen loses its activity after thirty-six hours. Preserved like vaccine, it remains infective for sixty hours. In proportion as the virulence of the infecting secretion is weakened, the period of incubation increases in length and the inflammation excited becomes milder. The differences that we observe in the grades of gonorrhœal conjunctivitis can therefore be referred to the fact that the source of infection supplies secretion of different degrees of virulence, and this virulence is, moreover, still further modified by the immediate circumstances attending the process of infection.

139. Prophylaxis.—By proper prophylaxis infection by acute blennorrhœa can be prevented, a matter to be so much the more borne in mind

because, when the disease has once broken out, an unfortunate result can not always be averted. It is the physician's duty to call the attention of every man with gonorrhœa, and also of every woman with a vaginal discharge, to the danger of infecting the eyes, and to urge upon them strenuously the requisite cleanliness. If an eye is already attacked with acute blennorrhœa, care must be taken to keep the other eye from being infected by it and also to keep the disease from being transferred to persons in the vicinity. The protection of the second eye which has not yet been involved in the disease is best effected by a bandage which is applied in the following manner: The palpebral fissure is first closed by means of some narrow strips of sticking plaster applied in a vertical direction. Then the hollow about the eye is filled up with cotton, and the whole is covered by a strip of plaster (zinc adhesive plaster, zinc mull, etc.) which is cut to the proper shape and is carefully attached all round the margins of the orbit. In order to secure it better, the edges of the flap and the adjacent skin may further be coated with collodion. [It is well to have the dressing include a transparent covering (watch crystal, Buller's shield) hermetically fastened over the eye. The eye can then be inspected without removing the dressing. —D.] If there is reason to think that infection of the eye apparently healthy has already taken place, we may try to prevent the outbreak of the disease by instilling a 2-per-cent solution of silver nitrate [or 5-per-cent protargol] before applying the bandage.

To prevent the spread of the disease to those in the neighborhood of the patient, the greatest cleanliness must be inculcated both upon him and upon the persons attending to him; they must always cleanse the hands after touching the affected eye, and must remove, or, best of all, burn, all materials that have been used for cleansing the eye (pieces of linen, cotton, etc.). The physicians and nurses should use protective (large, colorless, coquille-shaped) eye guards. If in spite of this protection secretion spurts into the eye, the latter must be very thoroughly washed out; then a couple of drops of 2-per-cent silver nitrate should be instilled, and subsequently for some hours cold compresses placed on the eye.

140. Treatment.—The treatment of the disease itself consists primarily in careful, repeated *cleansing* of the eye from its profuse secretion (by night as well as day). This is done with a weak lukewarm solution of potassium permanganate (1–5,000), with which the conjunctival sac is washed out thoroughly three or four times a day (Kalt). [Warm boric acid solution, used even more frequently, answers the purpose more pleasantly and apparently quite as well.—D.] The irrigator used had best have a flattened spout so as to be introduced readily between the lids and eyeball. Between each irrigation the eye is cleansed assiduously with cotton pledgets moistened in the same solution; the lids being drawn gently apart when this is done.

If the great swelling of the lids does not permit the palebral fissure to be properly opened, and thus makes cleansing impossible, the palpebral fissure must be fully widened by a section made with the scissors at the external angle of the lids (*canthoplasty;* see § 835). This section has the further beneficial effect of diminishing the pressure which the much-swollen lids exert upon the eyeball.

In the first stage of the disease we combat the inflammation by iced compresses [applied continuously or at short intervals, according to the effect they produce on the swelling of the lids—D.], and also by the application of leeches (six to ten in number) to the temple. In the second stage brushing the conjunctiva with *nitrate of silver* is the best means for making the swelling of the conjunctiva and the profuse secretion rapidly disappear. The application of the brush must not, however, be begun until the tense swelling of the conjunctiva has given place to a soft, succulent condition; there should no longer be any membranous deposit, or any grayish infiltrated spots upon the conjunctiva. The application should be made with a 2-per-cent solution, but quite freely, and must be repeated twice a day as long as the profuse secretion is still present. The presence of ulcers of the cornea furnishes no contraindication to the use of nitrate of silver.

As soon as, in the third stage, the inflammatory symptoms and the secretion also have nearly disappeared and the thickening of the conjunctiva is the only thing that still remains to be removed, we exchange the silver solution for *copper sulphate.* This is applied by whittling a crystal of the substance down to a smooth, rounded extremity (copper pencil or bluestone) and stroking with it once or more the conjunctiva of the everted lids. Then the lachrymal fluid, which is tinged blue by the copper salt dissolved in it, is dipped up from the conjunctiva with a pledget of cotton; otherwise the pretty concentrated copper solution would come into contact with the cornea and irritate it greatly. The application of the bluestone is much more painful than that of the silver solution, but acts more energetically, and hence we get quicker results with it; but this treatment is permissible only if the cornea is either quite sound or has ulcers already in process of cicatrization, and not if there are fresh ulcerations of the cornea, which are still coated with pus.

The treatment of complications involving the *cornea* is conducted according to the rules (§§ 208 et seq.) for purulent keratitis. In very severe cases all treatment proves powerless to preserve the cornea, so that our efforts must be confined to the prevention of the more remote evil consequences of destruction of the cornea, like panophthalmitis or the formation of staphyloma, and to the securing of a flat cicatrix.

The fact that acute blennorrhœa is produced by micro-organisms would lead us to expect that disinfectant substances would be the best remedies in the *treatment* of it. Nevertheless, it has been shown that nitrate of silver far surpasses the disinfectants

proper for this purpose. It is, in fact, specially poisonous to the gonococcus, and, moreover, effects its removal mechanically by reducing to an eschar, and thus leading to the exfoliation of, the superficial layers of epithelium containing this microbe.

In gonorrhœal eye diseases a vaccine has been employed made from dead gonococci. In true gonorrhœal conjunctivitis this has not proved successful, but has been more so in the metastatic gonorrhœal affections (conjunctivitis, iritis).

In the first stage of the disease we may make use of scarifications of the chemotic conjunctiva in severe cases.

III. Ophthalmia Neonatorum

141. We designate by this name the inflammations of the conjunctiva occurring in the new-born. They do not constitute an etiological unit, but are produced by a variety of morbid germs; yet on practical grounds it is advisable to retain the group-name of ophthalmia (or blennorrhœa) neonatorum.

The majority of cases, and particularly almost all the severe cases, owe their origin to the gonococcus, and hence are in the same category as the gonorrhœal conjunctivitis of adults. The infection that gives rise to this *ophthalmia neonatorum gonorrhoica* occurs as a rule during parturition. In the passage of the child's head through the vagina, the eyelids are covered with the secretion contained in the latter, and this either penetrates immediately into the conjunctival sac through the palpebral fissure, or does so as soon as the child first opens his eyes. Under these circumstances the disease breaks out as a rule on the second or third (rarely on the fourth or fifth) day after birth. In those cases in which the disease makes its appearance still later than this, the infection can not any longer be referred to the act of birth. It has then been brought about through subsequent infection by the vaginal secretion of the mother (as is readily possible, particularly if the child sleeps in bed with the mother) or the child has been infected by another child, as, for instance, not rarely happened in former times in lying-in establishments and foundling asylums.

The *symptoms* of the disease are the same as in the gonorrhœal conjunctivitis of adults, except that they are in general less severe. For even when there are great swelling of the lids and very profuse purulent discharge, the part which the bulbar conjunctiva takes in the process is comparatively small, and we rarely find great chemosis. Hence also the danger of suppuration of the cornea is not so great. It does indeed occur, and that often enough too, but only in those cases which are treated badly or not at all. If a case comes under treatment in season—that is, while the cornea is still intact—the latter can almost to a certainty be maintained in a healthy state. The prognosis, therefore, can be stated as correspondingly favorable.

Conjunctivitis of the new-born may be produced by other morbid germs than the gonococcus (pneumococcus, bacterium coli, etc.). In these cases, too, the infection generally occurs during birth; but the in-

flammation usually breaks out rather later and runs a milder course than in the gonorrhœal form. It presents the symptoms of a more or less intense catarrhal ophthalmia. But since gonorrhœal cases are occasionally of a milder character, a sure distinction between the gonorrhœal and the non-gonorrhœal forms can be made only by bacteriological examination of the secretion. [One of the non-gonococcal forms, which is very frequent and widely disseminated, is due to a filtrable virus (i.e., one not containing demonstrable organisms), and is characterized by the presence of cell-inclusions, very like those found in trachoma (see Figs. 50 and 54). The disease itself, which is called *inclusion blennorrhœa,* differs from trachoma in not being followed by pannus and cicatricial changes. The virus inoculated into monkeys or the adult human being reproduces the disease (does not produce trachoma).—D.]

The treatment in the first stage consists in diligent cleansing of the eye (see page 158); when suppuration commences we begin with the application of a 2-per-cent nitrate-of-silver solution to the conjunctiva. In cases with profuse secretion this must be done twice a day. The application should be continued until the cure is complete, as otherwise the process may readily recur to a moderate degree. [It is particularly important that the application should not be made too often or too vigorously (see page 146). In making it, injury to the child's eye should be avoided by fixing the child's head in the manner described on page 76 and by separating the lids with Desmarre's elevators (Fig. 2) instead of with the fingers, which may cause too much pressure on the eyeball.

In ophthalmia neonatorum, as in purulent conjunctivitis in general, both argyrol and protargol are used in place of silver nitrate, and with satisfactory results if applied sufficiently often and in sufficiently strong solution (see page 144).—D.]

In the conjunctivitis of the new-born prophylaxis plays a still greater part than in gonorrhœal conjunctivitis of adults. There is perhaps no other eye disease in which the rigorous carrying out of prophylactic treatment would afford more gratifying results than in the conjunctivitis of the new-born, which might by means of it be made to disappear almost entirely. Credé's method has proved the best. While the first bath is being given the child's eyes should not be wet with the water of the bath but should be cleaned with extreme care with pure water and cotton. Then as soon as the child has been put on the dressing table to be wrapped up, a drop of a 2-per-cent solution of nitrate of silver is instilled into each eye.

Ophthalmia neonatorum is one of the most frequent of diseases; before the introduction of the prophylatic treatment it attacked from 1 to 20 per cent of children in the various lying-in establishments. Since children of the poorer class who were born outside of the institutions and were affected with gonorrhœa often were brought under medical treatment either too late or not at all, many of them went blind. Before the introduction of prophylaxis, ophthalmia neonatorum caused more than a tenth of all

cases of blindness. [In consonance with its usual etiology (gonorrhœa), ophthalmia neonatorum is unknown in Palestine, where also gonorrhœa practically never occurs (Butler, Friedenwald).—D.]

That prophylaxis, as introduced into practice by Credé, is actually efficient, is proved by the following data: Credé formerly had in the Leipsic Lying-in Asylum an average of 10.8 per cent of cases of blennorrhœa neonatorum in the whole number of newborn; after the introduction of his prophylactic method the number sank to 0.1 to 0.2 per cent. Others have similar favorable results to record. Hence, the endeavor should be made to have this form of prophylaxis carried out not only in lying-in establishments and by physicians, but also universally by mid-wives. The majority of pregnant women have catarrh of the vagina with a mucous or purulent discharge. In the greater portion of these cases we have to do with a benign vaginal catarrh, in a smaller portion with a virulent catarrh (gonorrhœa). In individual cases the distinction between benign and virulent is difficult or impossible, for which reason prophylactic treatment ought to be carried out in all cases. Already the effect of such prophylaxis is beginning to make itself felt in civilized countries. In 22 German asylums for the blind in 1876 the number of those who had been rendered blind by ophthalmia neonatorum was on an average 30 per cent, while in 1896 in 45 institutions it was only 19 per cent (Cohn).

Credé's prophylactic treatment acts as a protection against infection during parturition only. Infection can, however, take place even earlier; children have been known to come into the world with a blennorrhœa already fully developed—in fact, with the cornea already destroyed. Furthermore, against subsequent infection by the vaginal secretion of the mother, or by other children, other measures will have to be adopted, among which great cleanliness is to be assigned the first place. In foundling asylums, infants infected with blennorrhœa should be isolated from the rest, as otherwise infection will frequently take place. In the Vienna Foundling Asylum, during the years 1854-'66, no less than fourteen hundred and thirteen children were first attacked by blennorrhœa while in the asylum, and hence caught the infection in the latter.

In infants after the subsidence of the acute inflammation, a chronic hypertrophy of the conjunctiva (chronic blennorrhœa) develops much less frequently than in adults. On the other hand, even after an ophthalmia neonatorum, if severe, a mild cicatricial condition of the conjunctiva, especially of the retrotarsal folds, often remains for life. Perhaps, too, the change described by Schapringer under the name *epitarsus* or "apron of the conjunctiva," and considered by him to be congenital, should in many cases be attributed to a cicatricial formation of this sort; at least, so it appeared to me in the cases which I saw myself. This formation consists in a flat fold of conjunctiva which runs down from the convex border of the tarsus over the conjunctiva of the lid and, growing constantly narrower, finally becomes attached again to the conjunctiva of the lid near the free border of the latter. As in the case of a pseudopterygium (§176), we can generally pass a sound between the fold and the palpebral conjunctiva.

IV. Conjunctivitis Trachomatosa

142. Trachoma, like acute blennorrhœa, is an inflammation of the conjunctiva, which originates by infection, and produces an infectious, purulent secretion. It is distinguished from acute blennorrhœa principally by its chronic course, in which is developed an hypertrophy of the conjunctiva, that forms the most characteristic symptom of trachoma. From the roughness of the conjunctiva, caused by this hypertrophy, the disease has, in fact, received its name.[10]

[10] Trachoma, from τραχύς, rough.

The disease almost always attacks both eyes. Cases exceptionally occur in which the disease, even after it has lasted for years, remains confined to one side, which means that either on account of the patient's carefulness or for unknown reasons the transfer from the eye primarily affected to the other does not take place.

Symptoms.—The patients complain of sensitiveness to light, of lachrymation, and of sticking together of the lids; pain and visual disturbances are also often present. The examination of the eye shows that the latter is less widely opened, partly because of photophobia, partly because the heavy upper lid hangs lower down. After everting the lids, we see the conjunctiva of the tarsus and also that of the fold of transition reddened and thickened; its surface at the same time has become uneven to a varying degree. These changes are to be referred to an hypertrophy of the mucous membrane, which occurs under two different forms.

The *first form* consists in the development of the so-called papillæ. These are elevations newly formed on the surface of the conjunctiva, which consequently appears velvety, or, if the papillæ are large, appears studded with coarse granules, with small nodules, or even with raspberry-like projections, the thickening of the conjunctiva being so great that the subjacent Meibomian glands are no longer visible through it. This kind of hypertrophy, which is called the *papillary form*, is found exclusively in the tarsal conjunctiva (Fig. 48 A). It is always most clearly pronounced on the upper lid, which therefore must be everted in making the diagnosis of the trachoma.

The *second form* of hypertrophy is characterized by the presence of the trachoma granules. These are gray, translucent, roundish granules, showing through the most superficial layers of the conjunctiva, which they push up to form hemispherical swellings. On account of their translucent, seemingly gelatinous character, they have been likened to the eggs of frog-spawn or to grains of boiled sago. They are found principally in the retrotarsal folds (*f*, Fig. 48 A), in which they are imbedded in such numbers that, when the lower lid is drawn down, the fold projects as an elastic swelling, at the summit of which we sometimes see the granules arranged in rows like a string of pearls. To bring into view the granules in the upper retrotarsal fold, we push the fold down. This we accomplish if, after we evert the upper lid, we place a slender stick or glass rod on the skin of the lid above the convex border of the tarsus, and press the skin down. In the conjunctiva tarsi the trachoma granules are less readily visible. They are smaller in this situation, and cannot push the conjunctiva up because the latter is very closely adherent to the tarsus. Here, therefore, they generally appear as small, bright-yellowish points, which are situated deeply in the mucous membrane; quite often, though, they are hidden from sight altogether by the development of the papillæ. Trachoma granules are

often found in the semilunar fold, more rarely in other parts of the conjunctiva of the eyeball. This variety of proliferation of the conjunctiva is called the *granular* form.

The two forms of proliferation of the conjunctiva sometimes occur separately. In the great majority of cases, however, both are found at the same time in the same eye, and so distributed that in the conjunctiva of the lids the most prominent feature is the proliferation of papillæ; in the retrotarsal fold it is the formation of trachoma granules (Fig. 48 A).

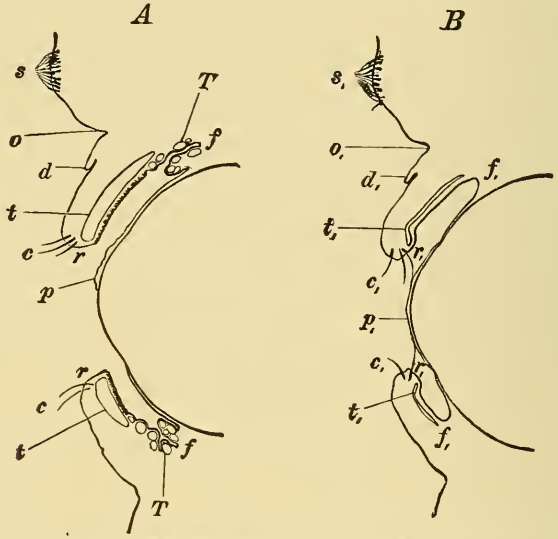

FIG. 48.—SCHEMATIC SECTION THROUGH THE LIDS AND EYEBALL (*A*, IN RECENT, *B*, IN OLD TRACHOMA).

A shows the way in which the two forms of hypertrophy of the conjunctiva are distributed among the separate divisions of the latter; *B*, the stage of sequelæ of trachoma; *s*, s_1, eyebrows; *o*, o_1, furrow between the brow and the lid (sulcus orbito-palpebralis); *d*, d_1, covering fold; *c*, cilia in their proper position; c_1, cilia turned toward the cornea; *r*, free border of the lid, with the borders of the upper and lower lids running parallel and the posterior margins of the lids sharp-edged; r_1, free border of the lid, looking backward, and with its posterior margin rounded; *t*, tarsus thickened by infiltration and covered with the velvety conjunctiva tarsi; t_1, tarsus thinned (atrophic), bent at an angle near its free extremity, and covered with smooth conjunctiva; *f*, fornix with numerous trachoma granulations, *T*, in the folds of the conjunctiva; f_1, fornix smooth, without folds (symblepharon posterius); *p*, thick pannus covering the upper half of the cornea; p_1, a shrunken pannus extending over the whole cornea.

The conjunctiva of the eyeball is, in light cases, unaltered, but when the irritation is more intense shows a coarsely reticulate injection. The conjunctiva discharges a purulent secretion, the quantity of which is more abundant in the fresh cases and in those attended with marked symptoms of irritation. In older cases, on the contrary, and in those which run a more sluggish course, it is very scanty.

Formerly cases marked by papillary hypertrophy and those marked by the formation of granulations were regarded as different diseases, and accordingly a distinction was made between a trachoma papillare (also called chronic blennorrhœa or ophthalmia purulenta chronica) and a trachoma granulosum (trachoma verum, trachoma Arlti,

trachoma folliculare). The cases in which both kinds of changes were present were called trachoma mixtum. At present trachoma is regarded as a single disease, for which the name conjunctivitis granulosa is also employed.

The *papillary* growths which impart to the conjunctiva its velvety or raspberry-like appearance are caused by an increase in size of the surface of the hypertrophic conjunctiva. The latter is thrown into folds, between which correspondingly deep clefts are formed; then on cross section the folds appear under the form of papillæ (Fig. 49, *P* and P_1). The connective tissue forming the papillæ is stuffed full of round cells; the surface of the papillæ is covered with a very much thickened epithelium (*e*, *e*), which, of course, is continued on into the depressions (*t*, *t*) that exist between the papillæ. These depressions hence have in microscopical cross section the appearance of a narrow canal coated with epithelium, and accordingly it was quite possible for them to be regarded as tubular glands. And in fact real, repeatedly branching glandular tubes do grow out from them and extend into the tissue of the conjunctiva.

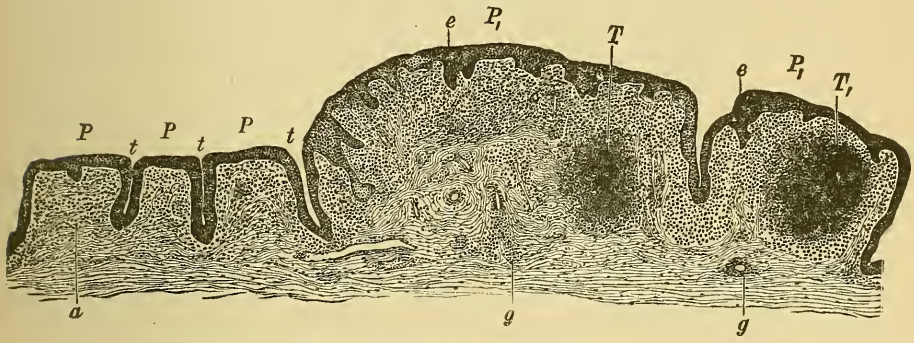

Fig. 49.—Cross Section through the Trachomatous Conjunctiva of the Upper Lid. Magnified 24 ×1.

Both small papillæ, *P*, *P*, *P*, and large ones, P_1, P_1, are found. The former stand side by side like the pickets of a palisade; the depressions, *t*, *t*, *t*, lying between them and coated with epithelium, look like the tubules of glands. The large papillæ contain trachoma granules, *T*, T_1, which are not sharply limited and do not possess a capsule. The epithelium of the conjunctiva is in many places, *e*, *e*, thickened. The mucous coat is in a condition of cellular infiltration, *a*, which is especially marked in the vicinity of the blood-vessels, *g*, *g*.

Papillary hypertrophy of the conjunctiva, however, is by no means a characteristic feature of trachoma, in the sense of being limited to it alone. In a less marked degree it is found in connection with every long-continued irritation of the conjunctiva; as in chronic catarrh, in conjunctivitis eczematosa that has lasted a long time, in ectropion upon the portion of the conjunctiva that is exposed to the air, after acute blennorrhœa when the so-called chronic blennorrhœa has developed from it, etc. Papillæ that are large but compressed and flat are the distinguishing mark of spring catarrh (see Fig. 57).

The *trachoma granulations* appear in microscopical cross sections under the form of rounded accumulations of cells, between which a very delicate connective tissue framework can be made out. The cells in the marginal portions of the granulations are lymphocytes; in its interior parts are predominantly uninuclear leucocytes (epithelioid cells), between which lie a few particularly large cells (phagocytes) which contain in their protoplasm small deeply-staining corpuscles; and in the connective tissue surrounding the granulation are formed plasma cells. The trachomatous granulation either passes without any sharp line of demarcation into the surrounding tissue, which is also very rich in cells (Fig. 49, *T* and T_1), or there is, especially in the case of the older granulations, a sort of incomplete capsule of connective tissue (Fig. 51, *k*). In the later stages of trachoma the granulations as a rule disappear in consequence of the formation in the con-

junctiva of new connective tissue, which by subsequent shrinking compresses the granulations and thus makes them vanish. A few granulations disappear because they are evacuated externally when the epithelium that covers them has been exfoliated (Fig. 50).

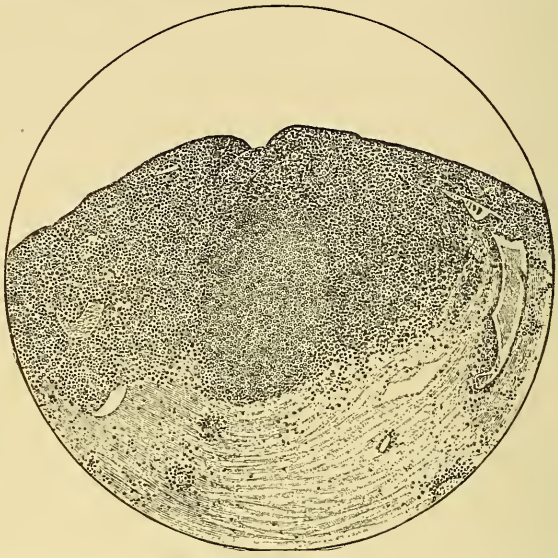

Fig. 50.—Granulation from the Retrotarsal Fold of a Monkey. Magnified 52 × 1.

The monkey 32 days before had received inoculation from a case of ophthalmia neonatorum, the secretion of which contained no gonococci but did contain the inclusions which are like those occurring in trachoma and are probably parasitic [inclusion blennorrhœa, see page 161]. The granulation shows the same histological picture as a trachoma granulation in man. It is still recent and hence is not yet shut off by a capsule from its surroundings. The central lighter portion, which in the section forms a vertical oval, consists almost exclusively of epithelioid cells, while in the peripheral portions of the granulation the lymphocytes predominate. The epithelium of the conjunctiva over the granulation has been exfoliated, and in this spot there has been produced by disintegration of the tissue a depression, through which the granulation would perhaps have been evacuated later on.

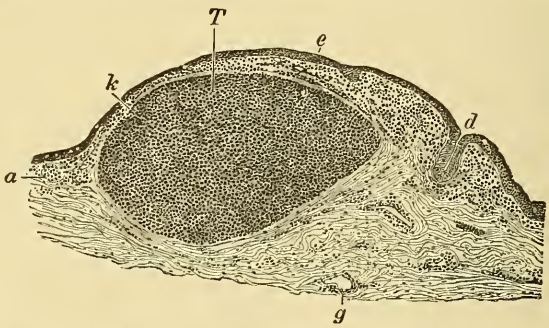

Fig. 51.—Trachomatous Granulation from the Fold of Transition. Magnified 24 × 1.

The trachomatous granulation, *T*, pushes up the conjunctiva in the form of a mound, and is inclosed by a layer of thickened connective tissue, the capsule *k*. The conjunctiva is infiltrated with cells, both in its upper layers, *a*, and along the vessels, *g*; the epithelium, *e*, shows, above the place marked *a*, bright spots which correspond to the goblet cells; at *d* it lines one of Henle's glands.

The so-called *trachoma mixtum*, which clinical observation has already shown to be the most frequent form, is proved by microscopical examination to be almost the only form that occurs. That is, even in those cases in which to the naked eye papillæ alone appear to be present, trachomatous granulations are found in cross sections examined under

the microscope either lying within the papillæ themselves or imbedded in the deeper portions of the mucous membrane. In the former case the papillæ have a particularly broad or even knob-shaped appearance (Fig. 49, P_1). In the second case the trachomatous granulations are concealed by the papillary bodies, beneath which they lie; then we often see them coming into view afterward when, as the result of prolonged treatment, the papillary growths have disappeared.

The *gelatinous trachoma* of Stellwag represents a later stage of mixed trachoma, in which a more uniform lymphoid infiltration exists in conjunction with superficial cicatricial changes. We have in that case a conjunctiva which is thickened, smooth on the surface, yellowish, and of gelatinous translucency.

143. In the *subsequent course* of the disease the hypertrophy of the conjunctiva gradually increases, growing steadily greater, until it has reached a certain height, which is not the same in all cases. Then it disappears again, step by step, while a cicatricial state of the conjunctiva with contraction takes its place. In this way the trachoma is cured in the sense that the specific morbid process has come to an end. Nevertheless, the conjunctiva has not become normal again by any means; on the contrary, it bears upon it lasting marks of the disease that has passed, namely, the signs of a cicatricial contraction which, in many instances entails other, additional, consequences, such as we will group together under the phrase "the stage of sequelæ of trachoma." The more considerable the degree which the hypertrophy of the conjunctiva attains the longer is the duration of the disease, which in most cases is counted by years, and the greater and more striking also is the subsequent contraction. The object of the treatment, therefore, must consist in checking the hypertrophy of the conjunctiva while it is developing, as thus both the duration of the disease is shortened and its evil consequences also are reduced to a smaller amount.

In the *conjunctiva tarsi*, the beginning of the *formation of cicatrices* is betokened by a few narrow, whitish striæ (fine cicatrical bands), which we see emerging in the midst of the reddened and thickened conjunctiva. These striæ gradually become more numerous and unite to form a delicate network, the meshes of which are occupied by red islands—that is, by those portions of the conjunctiva which are still hyperæmic and hypertrophied. Little by little the cicatricial lines grow steadily broader and the islands that they inclose steadily narrower, until at length that condition is produced in which the conjunctiva of the tarsus has become perfectly pale, thin, and smooth. The cicatricial condition of the conjunctiva corresponds in extent and intensity to the amount of hypertrophy that has preceded it. In those cases in which the hypertrophy of the conjunctiva has attained a considerable height in certain spots only, it is also only at these spots that deep cicatrices remain after the trachoma has run its course, while those parts of the conjunctiva which were simply infiltrated, or were hypertrophic to only a very slight degree, return to the normal state.

In the *conjunctiva of the fornix* the same conversion of hypertrophy into cicatricial contraction takes place. Only, the external phenomena are somewhat different, in conformity with the different character of the conjunctiva in this situation. Here we do not see any whitish bands, but we find that the thick swellings which are formed by the hypertrophic retrotarsal fold are becoming gradually thinner and flatter. Associated with this process, and proceeding with it step by step, is a condition of contraction taking place in the conjunctiva, a condition which steadily increases until even the folds that in the normal eye are present in the fornix are smoothed out and disappear (Fig. 48 B, at f_1). The conjunctiva has grown pale, and a delicate bluish-white coating is witness to the cicatricial character of its superficial layers.

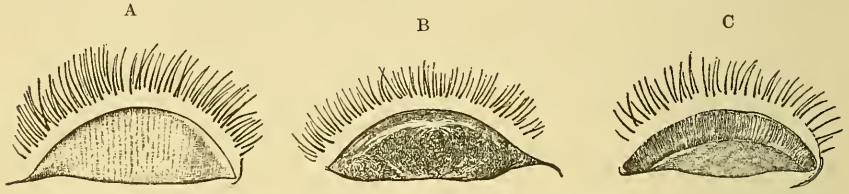

Fig. 52A.—Normal Upper Lid, Everted. The cilia jut out from the anterior edge of the border of the lid in several rows. The posterior edge of the border of the lid is sharp; the shadow directly beneath it represents the shallow groove of the sulcus subtarsalis. The upper convex border of the tarsus now looks downward and is stretched into a straight line, while the straight lower border of the tarsus now has an arched course. Through the conjunctiva are indistinctly seen the Meibomian glands.

Fig. 52 B.—Upper Lid in old Trachoma, Everted. Cicatrix in the sulcus subtarsalis. The tarsal portion of the lid is somewhat lower than in the normal lid. Near the posterior edge of the lid margin and parallel to it runs a longitudinal cicatrix from which minute bands pass upward. At the convex border of the tarsus a cicatrix formation of slighter degree is present.

Fig. 52 C.—Drawing of the Retrotarsal fold up over the Tarsus. As a result of shrinking the lid is not only lower but has also become shorter horizontally, so that it looks as if it were derived from a smaller individual than the normal lid. The posterior edge of the lid margin, owing to the shrinking, has been flattened out from a right to an obtuse angle, and has ceased to be sharp. The tarsal surface of the lid is divided into two parts by the cicatrical line which runs parallel to the lid margin. The lower part adjoining the lid margin is covered by the shrunken tarsal conjunctiva, which shows minute vertically running vessels and folds of similar course. The part lying above the cicatricial line is covered by the retrotarsal fold which has been drawn up upon the tarsus. The convex border of the tarsus, which looks downward, shows in its center a bulge such as frequently occurs in trachoma.

The transformation of the conjunctiva into *cicatricial tissue* proceeds as follows: A part of the numerous cells which are contained in the conjunctiva, and which are either uniformly scattered through it or occur in circumscribed accumulations (trachomatous granulations), disappears by resorption; another part, owing to rupture of the granulations, empties externally; and still another part gradually grows into spindle-shaped cells, and finally into connective-tissue fibers. This new-formed connective tissue shrinks extremely, so that the conjunctiva contracts and becomes thinner and of tendinous character. We have here a process similar to that which occurs in cirrhosis of the liver—i.e., the shrinking of a new connective tissue which has developed out of an inflammatory infiltration. It would be a mistake to suppose that in the trachomatous conjunctiva there are raw spots which become covered with a cicatrix—a mistake into which we might be more apt to fall because of the term granulations. What we call granulations in trachoma have nothing at all in common with the granulations of wounds, except their external appearance.

In the cicatricial trachoma, a linear cicatrix which runs parallel to the lid margin is often apparent on the conjunctiva of the upper lid. This has a varying significance. In the majority of cases it lies quite close to the margin of the lid (Fig. 52 B); it then

corresponds to the sulcus subtarsalis where the infiltration has gone more into the deeper parts (page 175). In the case figured there was also cicatricial formation at another place of predilection, namely, at the convex border of the tarsus. In less frequent cases the cicatricial stria lies higher up (Fig. 52 C). It then represents the boundary line between the conjunctiva of the tarsus and of the fornix. The tarsal conjunctiva is shrunk so as to cover but a small area, and the conjunctiva of the fornix has been drawn up in part upon the tarsus. It of course is not intimately adherent to the tarsus; and thus is explained the fact that in such cases the tarsus can readily be shelled out from the conjunctiva—a thing which is impossible when the conjunctiva is normal. This is an operation that has been recommended by Kuhnt [originally by Heisrath] for many cases of old rebellious trachoma. [See § 833.]

The *tarsus* in recent cases is thickened by the deposition of numerous lymphocytes. Later on it shrinks, its glands (both Meibomian and acinous glands) disappearing and nothing being left but a tough connective tissue which contains very few vessels and which sometimes is converted to a large extent into fatty tissue.

144. Complications.—The disturbance of sight, of which many patients complain, is founded upon a complication affecting the cornea, and appearing under two different forms, pannus and ulceration, which very frequently occur together.

Pannus[11] *trachomatosus* consists in the deposition upon the surface of the cornea of a newly formed, gelatinous, vascular tissue, which pushes its way from the edge toward the center of the cornea. At the spot where the pannus is located, the surface of the cornea is uneven and studded with fine projections, and there is a gray, translucent, superficially situated, cloudy mass, which is traversed by numerous vessels. The latter spring from the vessels of the conjunctiva, which pass over the limbus and out upon the cornea and, after arriving within the pannus, branch in an arborescent fashion (Fig. 84). The pannus ordinarily begins its development at the upper margin of the cornea [although it may also begin below or at one side], and covers first the upper half of the latter (*p*, Fig. 48 A). Quite often it terminates below in a sharp, straight, horizontal border. Afterward pannus develops at other portions of the corneal margin, until at length the entire cornea is covered by it. When pannus is pretty fully developed, the iris likewise participates in the inflammation (iritis). Disturbance of vision sets in as soon as the pannus has advanced into the pupillary area of the cornea. If this region is entirely covered by pannus, vision is reduced until it is limited to the recognition of large objets, or even to the mere ability to distinguish between light and darkness (quantitative vision).

Pannus, provided that further changes, such as will be described below, have not occurred in it, is capable of complete retrogression, so that the cornea can reacquire its normal transparency.

The *ulcers of the cornea* either develop at a spot that is otherwise normal, or they occur in connection with pannus. In the latter case they

[11] *Pannus*, a cloth.

are found principally at the free border of the pannus, more rarely within the latter. Since their character agrees with that of ulcers of the cornea generally, a more detailed account of them will be given under the latter head (§§ 197 et seq.). Ulcers heal, leaving behind them cloudy spots, the influence of which upon vision is dependent upon the degree of their opaqueness and also upon their situation with regard to the pupillary area of the cornea.

[Trachoma is also frequently complicated with infection of the lachrymal sac. Such sacs are very friable and rupture easily (Butler).—D.]

Pannus proves, upon histological examination, to be a layer of new-formed tissue, which, starting from the limbus, spreads over the cornea (Fig. 53, *P*). It is a soft tissue which is extremely rich in cells, and greatly resembles the infiltrated trachomatous conjunctiva. This tissue abounds in vessels, and occurs in alternately thicker and thinner layers, for which reason the pannus looks uneven and nodulated. Pannus, when it is quite superficial, insinuates itself between Bowman's membrane (Fig. 53, *B*) and the

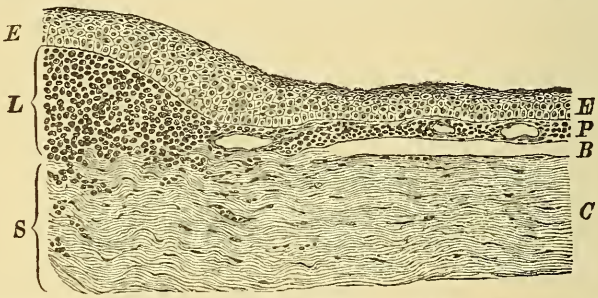

Fig. 53.—Cross Section Through the Margin of a Cornea Affected with Pannus. Magnified 125×1.

Beneath the epithelium, *E*, *E*, is the limbus, *L*, greatly thickened by cellular infiltration; from it the pannus, *P*, in which are perceived the cross sections of several vessels, extends between the epithelium and Bowman's membrane, *B*, over the cornea, *C*. *S*, sclera.

epithelium (Fig. 53, *E*), the latter being thus lifted off from Bowman's membrane and made to cover the pannus. In such a case it is possible for the cornea to regain completely its normal structure and transparency after the resorption of the pannus, since then the epithelium is once more directly applied to Bowman's membrane. But when the pannus lasts for a long time it destroys Bowman's membrane, and this is still more true of the less frequent cases in which the pannus from the very outset penetrates beneath Bowman's membrane. Later on, the most superficial layers of the cornea are infiltrated with a tissue abounding in cells. Then the complete restoration of the transparency of the cornea has become impossible.

For some forms and stages of pannus special names are in use. A recent pannus, which has not yet become thick, is called pannus tenuis, and if it is very vascular, pannus vasculosus. If the pannus has acquired a considerable thickness, it is then known as pannus crassus or pannus carnosus. An old pannus, composed of connective tissue and poor in vessels, is a pannus siccus.

A rare metamorphosis that has been observed in pannus is a process of sclerosis in which there develops from the pannus a dense white or yellowish tissue containing very few vessels. This tissue resembles a dense scar, e.g., such as occurs after deep ulcers of the cornea, but, unlike the latter, replaces only the superficial layers of the cornea—

those extending, for example, from the upper border to the center of the cornea, if the pannus itself had covered the upper half of the latter. Another change in old pannus consists in the development of small, intensely white spots, which frequently form a group in the pupillary region of the cornea. The appearance of the spots, which lie close to the minute blood-vessels of the pannus, reminds one of lead incrustation. The spots are superficial in seat, and may be removed by scraping (§ 264).

For *pseudo-pterygium*, see § 176 .

What are the *causes* of pannus in trachoma? Some see in pannus a direct transfer of the inflammatory process from the conjunctiva to the cornea. Against the occurrence of any such transfer per continuitatem, it has been urged, and with justice, that that portion of the conjunctiva which is interposed between the retrotarsal fold and the rim of the cornea, namely, the conjunctiva bulbi, takes little or no part in the trachomatous process. Another explanation starts from the fact that pannus in trachoma as a rule begins in the upper half of the cornea, and under ordinary circumstances has covered this portion entirely, before the lower half has been attacked at all. This would indicate that the upper lid, by reason of the roughness of its conjunctival surface, causes mechanically an irritation of the upper half of the cornea, and thus gives rise to inflammation in it. It is not to be doubted that this factor does come into play in the production of pannus, but it cannot be the only nor even the most important cause of pannus; for we often find the greatest roughness of the palpebral conjunctiva without pannus, and conversely find pannus in cases in which the palpebral conjunctiva is almost perfectly smooth. At the present time we can merely say that anatomically pannus is analogous to trachoma of the palpebral conjunctiva; that it is a trachomatous affection of that part of the conjunctiva which covers the cornea—i.e., of the conjunctival layer of the cornea. That this part of the conjunctiva becomes diseased in trachoma as readily as the conjunctiva of the lids or of the fold of transition, should not excite our wonder; on the contrary, it is more difficult to understand why the remainder of the conjunctiva, the conjunctiva scleræ, does not take a more active part in the trachomatous process. Perhaps the following explanation is the correct one. Fig. 53 shows that the infiltration of small cells is particularly marked in the limbus of the cornea (*L*), and gradually diminishes as it extends from the latter over the cornea itself. So also, where we make a macroscopical inspection, we find the limbus, at the spot where a pannus is on the point of developing, intensely reddened and so greatly swollen that sometimes it forms quite a thick outgrowth. Hence the impetus to the formation of a pannus seems to be given by a trachomatous affection of the limbus. Now, then, we must propound the following questions: 1. Why is it that the limbus in particular is affected so intensely in trachoma? and, 2. Why does the inflammation pass from the limbus to the cornea and not in the opposite direction—i.e., to the scleral conjunctiva? The first question must find its explanation in the fact that the limbus is by far the most vascular portion of the bulbar conjunctiva, and hence the part that is the most apt to be inflamed. That the inflammation spreads from the limbus in a centripetal direction—that is, upon the cornea—and not in a centrifugal direction upon the conjunctiva scleræ, agrees with what we have been able to observe in other affections of the limbus and of the adjacent portions of the cornea. We are acquainted with many diseases in which inflammatory infiltrations or vessels push their way inward from the limbus into the cornea. Probably this depends upon the centripetal direction of the circulation of the blood in the scleral conjunctiva. The arterial vessels run from the periphery toward the limbus, where they form a dense network of capillary loops. At this point, where the centripetal stream of blood finds its limits, a circulation of lymph begins, which is directed the same way and enters the cornea; and it is in the same direction that the inflammatory products advance, and that the blood-vessels which jut out from the marginal loops of the cornea tend to make their

way. Finally, it still remains to be explained why pannus generally begins at the upper margin of the cornea or why, in other words, the limbus is first affected at this point. If an eye is infected with trachoma, the conjunctiva is not attacked by the infection in its whole extent alike, but the infective matter adheres first to some circumscribed portion of the conjunctiva—generally to the conjunctiva of the tarsus or of the fornix, which is particularly apt to be affected with trachoma. Now, there are two ways in which the affection can spread from the portion of conjunctiva that is first attacked to other parts, namely, by continuity, in which case it extends gradually over the neighboring parts; and, by contiguity, in which case through contact with the diseased conjunctiva tarsi there is an infection of whose portions of the conjunctiva bulbi that lie opposite the former, and especially of the limbus, which is especially predisposed to infection. Now, it is precisely at the upper margin of the cornea that the limbus is in contact with the conjunctiva of the upper lid, and that, too, not only at night, but also all day, while the eye is open, since normally, even when the eye is open, the uppermost part of the cornea is covered by the upper lid. Here, therefore, the constant contact that exists is most favorable to an infection of the limbus by the diseased conjunctiva of the lids. That the rough condition of the latter assists in the production of this infection is likely. Such a condition acts partly as a mechanical irritant, partly by giving an impetus to infection by producing small multiple lesions of the conjunctival and corneal epithelium.

The *ptosis* which almost always accompanies trachoma and which gives trachomatous patients their characteristic appearance is in many cases attributable to the fact that the lid droops because it is heavy. Ptosis, however, is observed even when the thickening of the conjunctiva is inconsiderable, or indeed not present at all, and sometimes patients come to a physician solely on account of the ptosis, without having experienced any other trouble from their trachoma. There must therefore be some other cause for the ptosis beside the thickening of the conjunctiva. I suspect that the organic elevator of the lid (musculus palpebralis superior—see § 581 and Fig. 41, *t*), whose smooth muscular fibers lie directly beneath the retrotarsal conjunctiva, shares in the inflammation of the latter (and in older cases shares in its cicatricial contraction), and consequently becomes paral- zed.

145. Course.—The beginning of the disease shows a clinical picture which varies with the inflammatory symptoms accompanying the fundamental lesion. In the majority of cases the disease sets in with moderate symptoms of irritation—photophobia, lachrymation, pain—which augment with the increase in the objective changes. Not infrequently, however, trachoma develops so *insidiously* that for a long time those whom it has attacked are not aware of it. [In places where trachoma is endemic this seems to be the rule.] Such persons sometimes do not have their attention called to their disease until the pannus as it covers the cornea begins to disturb their sight. These cases belong as a rule to the granular form of trachoma. When the people living in barracks, schools, etc., that are infected with trachoma undergo medical examination, there are always found a number of inmates who do not complain of any troubles whatever and who regard themselves as perfectly healthy, while examination shows in the retrotarsal folds a very considerable development of trachoma granules. [This was eminently the case in New York when systematic inspection of the public schools for eye disease was first taken up; and, because so many of these latent cases were unearthed, the idea got abroad

that there was an alarming increase—a veritable epidemic—of trachoma in the city. The truth is that there were no more cases than before, only that more were discovered.—D.] In contrast with cases running this insidious course are the cases of what is called *acute trachoma*. In these the disease begins with very violent inflammatory accessories; the œdema of the lids, the great swelling of the conjunctiva, the profuse purulent secretion would almost lead us to imagine the case to be an acute blennorrhœa. The correct diagnosis can be made as a rule by our finding the conjunctiva studded with numerous trachoma granules. But if these are absent during the first days of illness, or if, because of the great swelling of the conjunctiva, they are not apparent, the subsequent course of the disease may be the only thing that can clear up the nature of the latter; which it does, since the hypertrophy of the conjunctiva, that is characteristic of trachoma, soon develops. Such acute cases occur chiefly during the prevalence of an epidemic of trachoma, and they are particularly contagious because of the profuse secretion. They are dangerous to sight not so much on account of pannus as of corneal ulcers, that make their appearance during the acute stage.

[These cases are rare.] Trachoma is essentially a chronic disease which only exceptionally begins with great inflammation. The majority of the acute cases, in fact, are not simple trachoma, but trachoma to which is superadded a second infection with bacteria, which like the gonococcus or the germs causing catarrhal ophthalmia, can set up acute conjunctivitis (see pages 142 and 143).

The subsequent case of the disease likewise varies greatly. It depends on (*a*) how far the anatomical changes in the conjunctiva have advanced, and (*b*) to what extent they are combined with inflammatory accidents and complications of a similar character on the part of the cornea.

As regards the first point, in the slightest cases the hypertrophy of the conjunctiva amounts to but little, and the cicatricial contraction that is left by it is correspondingly insignificant; so much so, perhaps, that it may scarcely be possible any longer to make the diagnosis of trachoma, if some time has elapsed since it occurred. In other cases the disease gets well with comparatively slight scars, and this may even occur without any treatment, inasmuch as the hypertrophy of the conjunctiva attains only a moderate pitch and then spontaneously abates. It is not such a very rare occurrence to find in a case the characteristic scars of trachoma in the conjunctiva without the patient's remembering that he ever had any trouble with his eyes. But in other, severe, cases the hypertrophy of the conjunctiva constantly progresses and sometimes reaches an extraordinary pitch, and consequently, also, serious changes are left in the conjunctiva. Serious, too, must be called every case in which the cornea has become implicated. It must be remarked, however: (1) That the symptoms of

irritation do not always by any means bear a fixed proportion in the objective changes; cases with very great hypertrophy of the conjunctiva and thick pannus often running their course without inflammatory accidents, and vice versa. (2) That similarly no fixed relation exists between the changes in the conjunctiva of the lids and those of the cornea. We see cases with very pronounced proliferation of the palpebral conjunctiva without pannus, and, on the other hand, cases with pannus and ulcers associated with a trifling affection of the conjunctiva. (3) In one and the same case the course is often very variable, in that sometimes intermissions or even spontaneous partial recoveries, sometimes relapses and exacerbations, occur. The latter are surely to be expected if, in a case that has been improved by treatment, treatment is too soon discontinued; but it is noticed that they also occur without any known cause under appropriate treatment properly carried out. Thus, a suddenly occurring supplemental attack of pannus can in a short time annihilate the results of months of treatment.

146. Stage of Sequelæ of Trachoma.—It is only the lightest cases, or those that come under treatment early, that are completely cured. In other cases there are left sequelæ, which are accompanied by a permanent impairment of the eye. These affect either the lids and conjunctiva or the cornea. They are as follows:

1. *Distortion of the lids* with faulty disposition of the cilia. The distortion is produced by the cicatricial contraction of the conjunctiva and the tarsus, as a result of which the tarsus bends in such a way as to be convex anteriorly. This distortion is recognizable even from an inspection of the lids while in situ, from the fact that they bulge more than usual. It appears still more clearly on everting the lids, especially in the upper lid, in which the distortion is always more pronounced. We find the conjunctival surface of this lid traversed by cicatrices, among which one that is particularly striking is a cicatricial band which runs in the form of a narrow white line some millimetres above the free edge of the lid and parallel with it (Fig. 52 B). Along this line there is a furrow-like depression produced by the drawing in of the conjunctiva and the tarsus. On everting the lids we feel that corresponding to this spot and lying, therefore, in the neighborhood of the free border of the lid, there is an angular bend of the tarsus (t_1, Fig. 48 B). From this bending of the tarsus the whole lid acquires a sort of boat-like or bowl-like shape.

The cause of the distortion of the tarsus lies partly in the cicatricial contraction of the conjunctiva; for, as the conjunctiva grows shorter upon the posterior surface of the tarsus, it tends to bulge the latter forward. But the distortion is mainly produced by changes in the tarsus itself. The latter is as much the seat of inflammatory infiltration in severe cases of trachoma as is the conjunctiva itself. It is hence increased in size

and, when we evert the lid, we feel that it is thicker, wider, and at the same time less pliable, so that sometimes the eversion of the lids is rendered considerably more difficult. From such a state of things the experienced observer would infer that he has to fear a subsequent distortion of the tarsus with its consequences. The infiltration and thickening of the tarsus are greatest near its lower margin, along the line at which the blood-vessels passing to the conjunctiva from in front perforate the tarsus (see page 133 and Fig. 42, *rp*). There is no doubt but that it is chiefly along these vessels that the inflammatory infiltration makes its way from the conjunctiva to the tarsus. Hence, cicatricial contraction, which succeeds the infiltration and which makes the whole tarsus thinner and narrower, is greatest at this spot and produces there an angular bending of the tarsus, corresponding to which is the cicatricial line that is seen running horizontally upon the conjunctiva tarsi, and the position of which agrees in general with that of the sulcus subtarsalis present in the normal lid (Fig. 52 B).

The immediate consequence of the distortion of the lid is an alteration in the position of its free border and of the cilia springing from it. The free border of the lid no longer looks straight downward (or in the lower lid straight upward), but backward (inward). The internal margin of the lid, which in the healthy state is sharp, becomes rounded off ("worn down") and is no longer to be recognized with distinctness (Fig. 48 B, r_1), this being due partly to the way in which it is drawn by the contracting conjunctiva, partly to the pressure of the eyeball upon it. By the turning inward of the free border of the lid the direction of the cilia (c_1) is changed, so that they now no longer look forward, but backward, and hence touch the surface of the cornea (*trichiasis*). Another factor besides the distortion of the tarsus that contributes to this false position of the cilia, is the tension which the contracting conjunctiva exerts. This tends to draw the skin, and with it the cilia, over the free border of the lid and up upon the posterior surface.

If the distortion of the lid progresses, the entire border of the lid turns backward and *entropion* is produced. In trichiasis and entropion alike there is a permanent condition of irritation, which is excited and maintained in the eye by the constant contact of the cilia with the cornea; if this condition lasts any length of time, diseases of the cornea make their appearance as a consequence of the mechanical injury produced by the cilia (see §§ 205 and 256).

The opposite kind of anomaly of position of the lid—that is, its turning outward, or *ectropion*—also occurs as a result of trachoma. The cause of this is that the conjunctiva, when it is thickened and has undergone great proliferation, crowds the lid away from the eyeball; the contraction of the muscular fibers of the orbicularis then suffices to complete the eversion of the lid. This kind of ectropion is usually found only in the lower lid (see § 611).

2. *Symblepharon Posterius.*—When the cicatricial contraction of the conjunctiva reaches a high degree, the folds of the region of transition flatten out completely; the conjunctiva passes directly from the lid to the eyeball (f_1, Fig. 48 B). If the lower lid is drawn down with the finger, the conjunctiva stretches tightly in the form of a vertical fold between the lid and the eyeball, and if the lid is drawn down still farther, the eyeball, being fastened tightly to it by the conjunctiva, must follow. This condition is characterized as symblepharon posterius (see § 177). In particularly severe cases the lower half of the conjunctival sac is reduced to a shallow groove between the lid and the eyeball.

3. *Xerosis Conjunctivæ.*—This condition develops when the conjunctiva, owing to excessive atrophy, loses its secretory functions. A steady diminution in the lachrymal secretions, which occurs at the same time, contributes to the production of the xerosis. Xerosis manifests itself by the following signs: The secretion, formerly copious, now becomes steadily scantier and assumes a tough, sticky, viscid character. In conjunction with this, a feeling of dryness develops in the eye. Subsequently there appear on the conjunctiva tarsi several dry-looking places, to which the lachrymal fluid cannot adhere any more than if they were smeared with grease. This condition tends to spread, until finally the conjunctiva may be affected by it throughout its whole extent. The cornea, which as a rule has become partly cicatricial from previous pannus and ulcers, likewise suffers from the deficient moistening; its epithelium becomes thicker, epidermoid, dry upon the surface, and hence opaque. Thus is produced that melancholy condition which is called *xerophthalmus* and which forms the worst termination of trachoma; the eye is rendered incurably blind, is disfiguring to its possessor, and in addition keeps torturing him with a constant, very tormenting sense of dryness (see § 180).

4. *Corneal Opacities.*—These are after-effects, both of ulcers of the cornea and of pannus. A recent pannus, it is true, can disappear completely by a process of absorption, so that the cornea reacquires its normal transparency. But often further changes take place in the pannus, which render its complete disappearance impossible. Among them is to be reckoned in the first place (*a*) *the transformation into connective tissue*, which the pannus undergoes if it lasts for a long time. In this the same change takes place in the pannus as in the trachomatous conjunctiva, a portion of the cells of which the pannus is composed growing into spindle-cells and finally into connective-tissue fibers. As a consequence of this the pannus becomes thinner, its surface grows smooth, the vessels with a few exceptions disappear, and at length the pannus is transformed into a thin membrane of connective tissue which covers the surface of the cornea and hardly admits of further resorption. In cases in which the pannus is quite thick and succulent and covers the whole cornea, (*b*) *ectasia of the cornea* sometimes results. That is, as the tissue of the pannus, which is

soft and abounding in cells, penetrates more deeply into the cornea, the tissue of the latter softens and gives way before the intra-ocular pressure (keratectasia e panno). Such a cornea never becomes perfectly clear again. The same thing is true, finally, of those cases in which (*c*) pannus is complicated with *ulcers;* the regions which are occupied by the latter likewise have permanent opacities left upon them.

Trachoma, then, is a disease which is distinguished by its duration, extending over years, and which in many cases renders those who are attacked by it half or wholly blind. If we add to this the fact that because of its infectious nature it is exceedingly apt to spread, we shall understand how, for those regions in which it is endemic, it is a veritable scourge.

147. Etiology.—Trachoma originates exclusively in infection proceeding from another eye affected with trachoma. Infection takes place by transfer of the secretion; contagion by means of the atmosphere, the existence of which was formerly accepted, seems not to occur. In all probability the secretion owes its infectious character to a micro-organism, as to whose nature, however, the investigations have not as yet been concluded. Since it is the secretion alone that transmits the infection, the danger of infection, which any given case carries with it, is in direct proportion to the amount of the secretion; the more profuse the latter, the greater being the danger to those in the immediate neighborhood of the patient. [Per contra, ordinary trachoma, which is attended with very little secretion, is in all probability but slightly contagious. We often see families, some member of which suffers from trachoma and yet fails to infect the others with whom he is all the time brought into intimate contact—and that too, when the commonest hygienic precautions are entirely neglected. It seems probable that something must be added to chronic trachoma to make it actively contagious, and that in most cases this something is an intercurrent conjunctivitis which furnishes the secretion that acts to carry the contagium.—D.] The transfer of the secretion from one eye to another generally takes place indirectly through the medium of the finger or very frequently through the medium of any article which, like sponges, towels, handkerchiefs, etc., is brought into contact with the eyes in making the toilet. A special opportunity for this to occur is afforded when a pretty large number of people have their sleeping apartments in common, and so, too, make common use of the articles above mentioned. Hence, trachoma spreads most extensively in barracks, penal establishments, poorhouses, boarding schools, orphan asylums, day schools of all kinds, etc. Moreover, outside of such institutions the same factor asserts itself, trachoma mainly attacking poor people who live crowded close together and bestow little care upon cleanliness. Furthermore, the fact that in many countries the Jews are special sufferers from trachoma is to be attributed to the same cause. Trachoma, finally, varies in its geographical distribution. It is most frequent in Arabia [and Palestine] and in Egypt, which is regarded as its proper home (ophthalmia

Ægyptiaca, Egyptian ophthalmia). [In these countries scarcely anyone is free from it.] In Europe it is much more wide-spread in the east than in the west. [It was formerly thought that elevated regions were almost entirely free from it. This is true of Switzerland and the Tyrol; but it is very prevalent in the Caucasus and in the mountainous parts of West Virginia and Kentucky.] In Europe it is very frequently found in the low lands (Belgium, Holland, Hungary, and the whole region of the lower Danube).

[In this country it is common in our Eastern cities, especially among the foreign population and most of all among the Russian and Polish Jews and immigrants from Eastern Europe generally and among the Irish and Italians. It occurs frequently, too, among the Chinese and Japanese. It is also common, however, and often in a severe form, among our native Americans, particularly in some parts of the Middle West (Illinois, Kentucky, West Virginia). It is prevalent among the Indians, while negroes, in this country at least, and in Cuba, are almost exempt (Swan Burnett and others). This exemption, however, is probably due rather to conditions of environment than to any racial immunity. In fact, it is doubtful whether any marked racial or climatic immunity or predisposition to the disease exists (Axenfeld).

It has been thought that trachoma is rare in very young children, but in the Near East it is common even in infants, although usually in benign forms, complications being infrequent (Butler, Friedenwald).—D.]

It was at the commencement of the last century that trachoma began to attract the attention of physicians to any great degree. It was then that the disease first showed itself as an *epidemic* among the European armies (ophthalmia militaris). People were of the opinion that it had been introduced into Europe from Egypt (hence ophthalmia Ægyptiaca) by Napoleon I. For when the latter, in July, 1798, landed in Egypt with an army of thirty-two thousand men, most of the soldiers were very soon attacked by a violent ophthalmia, and these were supposed to have brought with them upon their return to Europe the disease which was formerly confined to Egypt. Subsequent historical researches, however, have shown that the disease had already been endemic in Europe since antiquity. It is mentioned in the Ebers papyrus and in a pseudo-Hippocratic manuscript. Celsus gives a good description of the roughness of the lids and the purulent discharge that it occasions. For treatment the ancients employed scarification of the conjunctiva, which is still to-day made use of by some, and which was accomplished both by means of various instruments and also by friction with fig leaves.

From time immemorial, then, trachoma has existed in Europe as an endemic disease. But when by reason of the Napoleonic wars the armies came so repeatedly in contact with each other and with the civil population, the disease became more widely disseminated and occurred in epidemics. In some countries it became frightfully prevalent. In the English army, during the year 1818, there were more than 5,000 on the invalid list, who had been rendered blind as a consequence of trachoma. In the Prussian army, from 1813 to 1817, 20,000 to 30,000 men were attacked with it; in the Russian army, from 1816 to 1839, 76,811 men were subjects of the disease. In Belgium, in 1840, one out of every five soldiers was affected with trachoma. The French army, which was supposed to form the starting-point of the disease, was just the one that, relatively speaking, was least attacked. The armies disseminated trachoma

among the civil population through the discharge of soldiers affected with eye diseases, through the quartering of troops, etc. When they had so many trachomatous soldiers in the Belgian army that they did not know what to do, the Government applied to Jüngken, who had then great repute as an ophthalmologist in Berlin. He recommended them to dismiss the trachomatous soldiers to their homes. By means of this fatal measure trachoma soon became diffused in Belgium to an extent that has been observed in no other European state.

Among the *civil population* trachoma finds a favorable soil for its dissemination in places where many men dwell together, hence among the poorer classes, but particularly in large public asylums. If trachoma has made its way into such an establishment, and no measures are taken against its spreading, soon a great number or even all the inmates will be attacked by it. In a pauper school at Holborn, the whole five hundred children suffered from trachoma (Bader). Hairion, in 1840, found in an orphan asylum at Mecheln sixty-four out of sixty-six orphan girls affected with trachoma; in Mons, seventy-one out of seventy-four orphan girls were suffering from the disease. On board ship, where the crew live so closely crowded together, trachoma can spread very quickly. Mackenzie tells the story of the epidemic which raged upon the French slave ship Rodeur in the year 1819. The disease broke out during the voyage, and first among the negroes who, to the number of 160, were crowded together in the hold. When they took the unfortunate people up on deck, because the fresh air seemed to have a favorable influence on the ophthalmia, many threw themselves overboard, so that they had to desist from doing this. Soon one of the sailors also was attacked, and three days later the captain and almost the whole crew were taken down with the disease, so that it was only with the greatest difficulty that the ship could be brought to its destination.

According to the descriptions of that time, trachoma then ran a very acute course, and was attended with profuse secretion, circumstances which explain the rapidity with which the disease spread and the frequency with which it ended in blindness due to suppuration of the cornea. Now that epidemics have ceased, these acute cases are rare, and the disease has become comparatively benign. At present trachoma exists in many countries as an endemic disease but mostly occurs under that chronic form under which, with scarcely any exceptions, we now see it. At the same time its prevalence has diminished. In 1888 the Prussian army had but 10 trachoma patients for every 1,000 soldiers. In Austria, in whose eastern provinces trachoma is still very prevalent, 8 out of every 1,000 of the military effectives were attacked by trachoma annually during the years 1881 to 1890. [In Roumania in 1900 15.9 per cent of the army and at present 5 to 7 per cent of the school children in Bucharest are affected (Puscariu).] In the Orient there is a very different state of affairs. Thus in Egypt [and Palestine], even at the present time, it is scarcely possible to find a native who has a normal conjunctiva, and innumerable people there are blind. [Blindness, however, in these countries is due rarely to trachoma, but rather to the acute summer epidemics of conjunctivitis with severe corneal complications (Butler and others).—D.]

Trachoma, therefore, not only appears under a varying clinical aspect, sometimes acute and threatening, sometimes chronic and mild, but it also seems to have changed its character in the course of time. The explanation of the way in which this has occurred was afforded by the study of trachoma in its native land, Egypt, where ophthalmia Ægyptiaca still rages as in the olden time. Investigations have shown (Müller, Morax) that in Egypt almost every native suffers from trachoma, with which as a rule he has been infected already in childhood. This runs just as chronic a course as with us and very frequently produces pannus. But, in addition, many of the natives are attacked during the hot season by an acute inflammation of the eyes, in the secretion from which there is found either the gonococcus or one of the germs causing acute

catarrhal ophthalmia, and most often the Koch-Weeks bacillus. It is cases of the former kind that by causing suppuration of the cornea produce the blindness that is so prevalent in Egypt. Now all these different inflammations of the eyes were lumped together under the name of Egyptian ophthalmia and were regarded as identical with trachoma, while, as a matter of fact, only the chronic cases are pure trachoma and most of the acute cases are mixed infections. A knowledge of this fact leads us to suppose that in Europe, too, during the great trachoma epidemics with their cases that ran such an acute and dangerous course, it was generally a question of mixed infections, being so either from the outset or from the subsequent superaddition of a second infection (Morax). However, the existence of an acute inflammation in pure trachoma cannot be altogether excluded. Experiments in the transfer of a pure trachomatous secretion to man have sometimes after an incubation period of 8 to 10 days resulted in the outbreak of trachoma with acute inflammatory symptoms (Addario, Greef). Of animals monkeys alone can be infected with trachoma.

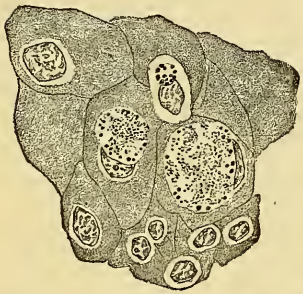

Fig. 54.—Parasites in Trachoma. Magnified 680×1.

A section from the upper retrotarsal fold in a recent trachoma shows the uppermost layers of the epithelium. Three of the epithelial cells contain parasitic inclusions. In the most superficially placed cell some pretty large granules form a small group alongside the nucleus in a ring-shaped cavity which has been produced artificially by shrinking in the hardening fluid. The two cells lying beneath show granules of this same kind, separated from each other and enclosing a cavity in which lie granules still more minute, imbedded in an irregular shrunken ground substance. Thus there is formed a rather large spherical structure, which pushes the cell nucleus to one side and flattens it.

Halberstaedter and Prowazek regard as the *exciting cause* of trachoma a structure which they have found in the epithelial cells of the trachomatous conjunctiva and which they call by the name of chlamydozoon. In the protoplasm of the epithelial cells there first appear granules which are as large as ordinary cocci or larger, and which as they multiply become separated from each other, so that they enclose a cavity destitute of granules (Fig. 54). In this cavity, then, very minute granules make their appearance which are smaller than cocci and which stain differently from the granules that first appeared. [Quite similar structures have been found in new-born infants affected with inclusion blennorrhœa, which, however, seems to be a morbid entity differing from trachoma (see page 161). Although these inclusions are occasionally found in other forms of conjunctivitis and rarely in the normal conjunctiva, their diagnostic significance is considerable and they are to be regarded as really pathogenic. They occur in the conjunctiva only and in all its layers (Axenfeld) and, since they can be cultivated outside of the body, must be regarded as representing definite organisms (Noguchi and Cohen).—D.]

[Some observers, on the other hand, claim that the cell inclusions are due simply to the intracellular growth of gonococci and hæmoglobinophile bacteria and that trachoma itself is not a morbid entity at all, but simply the expression of the reaction of the conjunctiva to various morbid agents. This is undoubtedly true of some of the manifestations of trachoma (see page 181), but the best authorities do not admit that it is true of trachoma itself nor accept this explanation of the cell inclusions.—D.]

[Another disease marked by cell inclusions and clinically closely resembling trachoma is *desquamative epitheliosis*. This condition, which is caused by the Cytozoon atrophicans, and which may occur in conjunction with Samoan conjunctivitis (see page 142), or independently, produces an acute conjunctivitis with œdema of the lids and infiltration of the cornea. The conjunctiva contains follicles, in which are large epithelioid cells containing reddish cell inclusions. Epithelial atrophy and vacuolation of the cell-walls are marked (Leber).—D.]

148. [Differential Diagnosis.—There are several conditions resembling trachoma, particularly in the formation of granulations. Chief among these is (1) follicular catarrh (see § 133).—D.]

The two diseases are very similar, in that lymph follicles occur as characteristic formations in both. In follicular catarrh they are smaller, are more sharply limited, and project farther above the surface of the conjunctiva; in trachoma they are larger, destitute of sharp outlines, and less prominent. Follicles proper are often oblong-oval (cylindrical) and placed side by side, in a row like a string of pearls, while trachomatous granulations are round and more rarely present any such arrangement in rows. But these characteristics are sometimes so obscured that even experts cannot, in many cases, make the diagnosis with certainty, and the subsequent course of the disease alone affords the desired information. Even in the histological structure no thoroughgoing distinction can be found between follicles and trachomatous granulations. A further resemblance between follicular catarrh and trachoma consists in the fact that they both chiefly occur among bodies of men who are confined in a small space. It is therefore easy to understand that these two diseases have repeatedly been confounded with each other; and a number of authors, in fact, explain follicular catarrh as being a kind of trachoma distinguished by its mildness and freedom from danger. That this is not correct is obvious from the fact that in follicular catarrh the exciting agent characteristic of trachoma is not found. To the same effect are the observations on the origin of follicular catarrh. It is not, indeed, yet certain whether follicular catarrh occurring among confined bodies of men is propagated by contagion, like trachoma, or is merely a result of the contamination of the air by dust, exhalations, etc. On the other hand, it is quite satisfactorily established that, under certain circumstances, follicular catarrh *may* arise without any contagion whatever. This is the case with the catarrh produced by atropine (see § 323), in which the irritation is a purely chemical one. But trachoma can never arise without contagion. A further and more important distinction between the two diseases is the course. Follicular catarrh is not associated, or is associated to only an inconsiderable degree, with papillary hypertrophy of the conjunctiva; it never leads to shrinking of the conjunctiva, to pannus, or to any of the other sequelæ; it is a disease perfectly devoid of danger, one which, even without any treatment, finally gets well and leaves no trace behind; so that on this account alone the differentiation of the two diseases is not only theoretically, but also practically, of great importance.

If a physician in any case is doubtful whether he is dealing with a trachoma or a follicular catarrh—a distinction which often cannot be made in the first examination, especially in cases of acute onset—he must denote the case as suspected trachoma, must take the steps necessary to avoid the spread of the disease, and must begin the treatment. If this treatment in a few days leads to rapid decline of the inflammation and diminution in size of the granulations, the case is one of follicular catarrh, since a trachoma would be much more obstinate. The right diagnosis can also be made if it is possible to demonstrate the presence of the exciting organisms of trachoma, but the preparation of properly stained specimens of these organisms requires special practice.

Probably we must regard the formation of granulations composed of adenoid tissue not as anything at all specific, but only as a particular form of reaction, which the conjunctiva of the eye, like other mucous membranes, exhibits toward many different sorts of irritants. Such a formation of granulations occurs in its most pronounced form in trachoma and in follicular catarrh. It may also occur in the following conditions:

(2) Certain cases of tuberculosis of the conjunctiva (page 208), which begin with an abundant development of granulations, quite as in trachoma.

(3) Certain conjunctival affections, regarded as syphilitic, in which there is an abundant formation of granulations (Goldzieher and Sattler).

(4) Cases in which nodules resembling follicles have formed round small foreign bodies, as when plant hairs or caterpillar hairs have gotten into the eye and penetrated into the tissue of the conjunctiva.

(5) Parinaud's conjunctivitis (see page 209).

(6) Sporotrichosis of the conjunctiva (see page 210).

[(7) Desquamative epitheliosis (see page 180).]

149. Therapy.—The treatment of the trachomatous *conjunctiva* has a twofold object in view; on the one hand, it seeks to do away with the inflammatory complications and the increase of secretion, which is associated with them; on the other hand, to further the disappearance of the conjunctival hypertrophy. In this way it is most likely that the process of shrinking in the conjunctiva will be restricted as much as possible, so as to avert the evil consequences resulting from its cicatricial contraction. We attain both objects by the proper employment of caustics, of which two are almost exclusively in use: nitrate of silver in 2-per-cent solution and sulphate of copper in the form of a stick. The silver has the feebler action, and is therefore borne better; copper, being applied in substance, has a correspondingly stronger cauterant action, but also causes more irritation (see page 159). These remedies are, as a rule, applied once a day, it being only in severe cases that they are used twice a day. The indications for the two remedies are as follows: Nitrate of silver is employed in all recent cases with violent inflammatory symptoms and great secretion. It can also be used when there are ulcers upon the cornea that are still in the progressive stage, if we take care that none of the solution comes into contact with the cornea itself. Copper sulphate is suitable for those cases in which the inflammatory symptoms are small, and its chief use is in removing the hypertrophy of the conjunctiva. For this purpose it acts much more energetically than the silver solution, and should therefore be used in preference to it in all those cases in which its application is allowable at all. Great inflammatory irritation, and particularly the presence of ulcers of the cornea in a state of active progress, contraindicate the use of the bluestone.

From these indications it follows that, as a rule, we treat a recent case with the silver solution until the inflammatory symptoms have disappeared and the secretion has diminished. As soon as this has occurred—and several weeks are generally sufficient for the purpose—we replace the silver solution by bluestone. At any rate, we must avoid using the silver solution for too long a time on account of the argyrosis which may develop as a result of it. The copper is now to be used, the application of it being made stronger or weaker according to the degree of hypertrophy, and is to be kept up for months and even years, until every trace of hypertrophy has vanished and the conjunctiva has become free from congestion and smooth throughout. At first the application is made every day; but when only slight remains of

the hypertrophy exist, it is sufficient to make the application every other day, and subsequently every third day; and at this stage, the milder acting alum pencil (a sliver of alum whittled down to a fine edge) may be substituted for the bluestone. Moreover, the application should be made less and less energetically all the time, until finally, when the cure of the trachoma is complete, the application is entirely suspended. In these later stages of the disease we can instruct the patient how to evert the lid himself and touch it with the bluestone, so that he need not come so often to the physician. Or, we may prescribe for him an ointment of copper sulphate (½ to 1 per cent) or of copper citrate (10 per cent; Arlt, Jr.), which he himself can rub into the conjunctival sac. [An effectual substitute for the copper stick is a 10-per-cent solution of copper sulphate in glycerin. This is diluted with from 15 to 60 parts of water, and the mixture is dropped into the eye three or four times a day. The aqueous mixture must be made up fresh daily, and its strength increased as fast as the patient can stand it (Prince). —D.] When there is great cicatricial contraction of the conjunctiva the bluestone is not applicable at all, and must be replaced by ointments. A 1- or 2-per-cent ointment of white or yellow mercurial precipitate (the latter acts more energetically) may be rubbed into the conjunctival sac. In relapses with great inflammatory irritation, such as often occur in the course of the treatment, the copper is always to be replaced for a short time by the silver solution. If, however, the symptoms of irritation are very violent, the silver solution itself cannot always be borne, and must then be replaced for some time by milder remedies, such as instillations or compresses made with weak solutions of corrosive sublimate or boric acid.

The *operative treatment* of trachoma is indicated in cases in which very numerous granulations are present in the retrotarsal folds. [In recent cases with abundant, succulent granulations, we remove the latter by curetting, or the galvano-cautery, or, better and more thoroughly, by expression or brossage (see § 819). Cauterization with carbon dioxide snow has also been used with success (Butler).—D.]

Keining's method of daily repeated friction of the conjunctiva with a 1: 2,000 sublimate solution or the massage of the conjunctiva with a rounded glass rod is also efficient, mainly owing to its mechanical effect—i.e., to its causing expression of the granules. [A sublimate solution of 1: 500 may be used, rubbed well into the retrotarsal folds. It causes no more irritation than the copper stick and by many is held to be at least equally effective. Phototherapy and actinotherapy have also been used with more or less success for removing the granulations.—D.]

None of the mechanical measures should be applied, if there is any evidence of considerable inflammatory irritation of the eye, such as marked redness and swelling of the conjunctiva and secretion in corresponding amount. If these are present, they are to be allayed by a sufficiently prolonged treatment with the silver solution before any operation is done.

Neither an immediate nor a radical cure of trachoma is effected by these mechanical means, since along with the larger granulations small ones in process of development are always present, which cannot be removed, and which grow bigger afterward. Hence it is necessary after the reaction produced by the operation has subsided to apply caustics in the usual way. But it must be conceded that in suitable cases the duration of treatment is considerably shortened by expression and similar procedures.

The treatment of trachoma must be kept up until the hypertrophy of the conjunctiva is completely done away with, as otherwise relapses are to be looked for sooner or later. The chief difficulty in the treatment lies in its great length, it often requiring many months for a complete cure. Those patients who have not the endurance nor the means necessary for such a course, give up treatment as soon as their subjective troubles have disappeared, without, however, being completely cured. Then we commonly see them returning after some time with a relapse, which is often more severe than the disease for which we originally treated them. This lack of completeness in the treatment is the reason why the disease with many men drags on through their whole life.

[To avoid this protracted and incomplete treatment and particularly to prevent complications, many advocate the early performance of a radical operation (excision of the tarsus and infected conjunctiva—see §§ 824,833). This seems indicated, at all events, whenever the disease shows no prospect of yielding to the milder measures already outlined or when pannus or other threatening complications are setting in. In children, however, the radical operation should not be done, unless absolutely necessary.—D.]

150. The treatment of *complications affecting the cornea* is conducted on the principle that the affections of the cornea, caused by a conjunctival trouble, are best cured by the treatment of the conjunctival trouble itself. Hence, *ulcers of the cornea* when occurring in connection with trachoma are not combated directly, but have their cure brought about by means of applications made to the conjunctiva. The only limitation to this is that, where there are corneal ulcers in active progress, the silver solution is demanded and the bluestone, on the other hand, is contraindicated, and further, that contact of the caustic with the cornea should be avoided as far as possible. For the iritis, which not rarely is associated with ulcers of the cornea, atropine in 1-per-cent solution is instilled. In other respects, ulcers of the cornea are to be treated according to the rules which are in general applicable to them (see §§ 208 et seq.). Only it must be noted that bandaging, which is generally indicated in the case of ulcers of the cornea, should be avoided as far as possible when trachoma is present, because by the closure of the eye the secretion is retained in the conjunctival sac, and thus both the conjunctival and the corneal troubles are aggravated.

Pannus, in recent cases, disappears of itself, simply from applications being made to the conjunctiva. If the pannus is unusually dense, it is allow-

able to make careful applications of the caustic to the pannus itself. Since pannus is often associated with slight iritis, atropine should be instilled from time to time, in order to keep the pupil dilated and prevent the formation of posterior synechiæ. [In recent pannus we may try subconjunctival injections (page 65) and in thick pannus, peritomy (§824).]

Very old pannus, which already is partly made up of connective tissue and has lost all but a few of its vessels, requires special treatment. Experience has shown that further resorption can be obtained in such a pannus by exciting a violent inflammation in it, and so producing an increased succulence and a greater vascularity. For this purpose we make use of the *jequirity* treatment (De Wecker). We here employ a 3- to 5-per-cent infusion of jequirity, which is prepared by extracting the ground jequirity beans for twenty-four hours with cold water. With this infusion, which is to be prepared fresh every day, the conjunctiva of the everted lids is painted very thoroughly two or three times a day. The inflammation that is thus produced reaches the desired height on the second or third day, when the lids are reddened and become swollen with œdema, the conjunctiva is strongly injected and covered with a croupous membrane, and slight chemosis is often present. This inflammation we call jequirity ophthalmia. As soon as it has attained the height just described, the further application of the remedy is discontinued, as otherwise we should make the inflammation increase to the point where it would cause a necrotic disintegration of the conjunctiva and cornea. We now allow the inflammation to run its course, simply keeping the eye clean; when the inflammation has completely subsided, the cornea is found to have gained in transparency as compared with its former state, and sometimes to a very considerable extent. This very energetic treatment is adapted only to those old cases of trachoma in which the more pronounced symptoms of inflammation are wanting, the conjunctiva is in great part cicatricial, and the cornea is entirely covered by old pannus.

Jequirity (the seeds of the Abrus præcatorius) has for a long time been in use in Brazil, where trachoma is endemic, as a popular remedy for this disease. Its action does not depend upon the presence of micro-organisms in the infusion, as was originally believed, but upon an unorganized ferment (abrin) which is excessively poisonous (Hippel, Neisser, Salomonsen, Venneman). A pure product of this sort, prepared by Römer under the name of jequiritol, may be used instead of the infusion of jequirity.

151. Of the *sequelæ* of trachoma, [the distortion of the lids and its consequences,] trichiasis, entropion, [and ectropion], demand operative treatment (see §§827–839). The symblepharon posterius, which is produced by the shrinking of the conjunctiva, is not amenable to treatment. Xerosis of the conjunctiva is also incurable, so that treatment must be limited to the amelioration of the patient's sufferings. To diminish the sense of dryness, frequent instillations of milk, glycerin, or mucilaginous substances (e.g., the mucilago seminum cydoniorum) may be made. For bad cases, Rudin, in order to preserve the eyeball from desiccation, has advised refreshing the edges of the lids and stitching them together, so as to unite them throughout except for a small space in their middle.

152. Hygienic Treatment.—In addition to the foregoing measures for the treatment of trachoma, it is self-evident that care must be taken to keep the eye clean, for which purpose we may prescribe weak antiseptic solutions. The patient should have a nourishing diet; he ought not to be kept in his

room, but, on the contrary, should be made to go out as much as possible in the open air and take exercise, and if necessary may be directed to engage in some light out-of-door work.

With a disease of this infectious character, its dissemination should be checked by suitable *prophylaxis*. The physician must set a good example, and must cleanse his hands carefully after touching a trachomatous eye. He must call the attention of the patient affected with trachoma to the contagious nature of this disease. He must teach him how to protect from infection the other eye, which may be still healthy, and how to avoid spreading the disease among those in his immediate neighborhood, his family, his fellow-workmen, etc. For securing the latter object, the prime requisite is that the patient should have his own washing materials, linen, bed, etc., and should keep them exclusively for his own use.

The prevention of epidemics of trachoma in public establishments, such as barracks and institutions and schools of every sort, constitutes an important duty of the officials in charge of such places. These officials should take care that the members of their community have separate washing materials, linen, etc., for their use. They should be kept apprised of the presence of any trachomatous patients by means of frequent medical inspection, and, as soon as such a patient is found, he should be immediately removed from the community; for, where no trachomatous patient is found, no extension of the disease is possible.

[In this country a great deal has been done in the way of prophylaxis by excluding trachomatous immigrants, by the systematic inspection and treatment of children in the public schools and in some places by making trachoma a reportable disease. It is important that the public should be educated with regard to the grave significance of the disease and the way in which it can be avoided.—D.]

V. Conjunctivitis Diphtherica

153. Conjunctivitis diphtherica,[12] like gonorrhœal conjunctivitis and trachoma, is a purulent inflammation of the conjunctiva which spreads by contagion, and the secretion of which is infectious. But the contagium is different from that of the other two diseases, being in this case the diphtheria bacillus of Löffler. The inflammation produced by this is generally violent, and in the severe cases is among the most intense of any that are observed in the conjunctiva. The lids are much swollen and reddened, hot, and painful to the touch. In particularly severe cases the lids are actually of board-like hardness, so that it is impossible to evert them, and scarcely possible even to open the palpebral fissure. The lymphatic glands in front of the ear or in the neck are swollen. The appearance of the conjunctiva is characteristic of the property that the diphtheria bacillus possesses of producing

[12] From διφθέρα, a membrane.

a profuse exudation which has a great tendency to coagulate. This coagulation either affects the exudate that is poured out upon the surface of the conjunctiva, and which consequently clots to form membranes, or it takes place within the tissue of the conjunctiva. Consequently we may distinguish two forms of diphtheria of the conjunctiva, the clinical aspect of which is depicted below. This, however, does not suffice for a sure diagnosis, since the same picture may be produced by other bacteria beside the Löffler bacillus (see page 191). Hence, a bacteriological examination is also necessary, and the more so because upon it will depend in part the treatment (e.g., as to employment of antitoxin, and regulations to prevent the transmission of the disease). [The two forms are:—]

(*a*) The *superficial* or *croupous* form. This is characterized by the presence of a grayish-white membrane, which adheres pretty closely to the surface of the conjunctiva, but still can be usually removed from it with a forceps. When this is done we find the conjunctiva beneath to be greatly reddened and swollen and in some places bleeding, but we notice no great losses of substance in it. The membrane which we have detached consists of a minute fibrous meshwork of clotted fibrin, in which pus corpuscles and a few epithelial cells from the conjunctiva are imbedded. The croupous membrane covers the tarsal conjunctiva; more rarely, the retrotarsal fold and even the conjunctiva of the eyeball. In most cases after from one to two weeks the membrane gradually disappears. The eye now merely presents the condition of an intense catarrhal inflammation which gets well without leaving any permanent changes in the conjunctiva. In severe cases the disease is complicated with corneal ulcers, which, however, but rarely lead to extensive destruction of the cornea.

(*b*) The *deep* form (*diphtheria* of the conjunctiva in the narrower sense). This runs a much more serious course than does the superficial form, as in order for it to occur the exudate must coagulate while still within the tissues of the conjunctiva, so that the vessels are compressed by it, and the mucous membrane consequently is rendered rigid and bloodless and falls a prey to necrosis. Hence, on everting the lids we find beside the marked swelling and redness of the conjunctiva spots in which the conjunctiva is somewhat depressed, smooth, and of a grayish-yellow color, and often contains a few dirty-red speckled markings (ecchymoses). In the severest cases, either a very large portion of the conjunctiva, or even its entire surface, acquires this character and is uniformly gray and hard, just as it is after being intensely cauterized, e.g., by the action of lime. The lymph glands in front of the ear and often also those in the neck are swollen and painful.

The condition just described, which develops rapidly after a short period of incubation, is called the first stage of the disease, or stage of *infiltration*. It keeps up for from five to ten days, according to the extent of the diphtheritic process upon the conjunctiva. Then the spots of diphtheritic

infiltration begin gradually to disappear. Where the infiltration is not so very dense, resorption of the exudate occurs, but in those spots from which the circulation has been altogether cut off by the infiltration and the tissue has consequently mortified the necrotic portions slough away. Thus are produced in the conjunctiva losses of substance, which soon become covered with granulations such as cover a raw surface. Meanwhile the secretion has become more abundant and more purulent, for which reason this second stage is characterized as the stage of *blennorrhœa*. The third stage is that of *cicatrization*, in which the granulating surfaces, that are produced by the sloughing off of the gangrenous portions of the conjunctiva, gradually grow smaller and are covered over with a new epithelial lining. Since the latter change is effected by a drawing in of the neighboring conjunctiva, the conjunctival sac as a whole is contracted; frequently, too, in single spots adhesions are produced between the conjunctiva of the lid and that of the eyeball (symblepharon). The more extended the diphtherial process the more striking is the subsequent cicatricial contraction of the conjunctiva. As a result of it trichiasis, cicatricial entropion, or even xerophthalmus may subsequently develop.

The deep form of diphtheria is much more severe than the croupous, not only in regard to its effect upon the conjunctiva, but also in other respects. Hence the cornea is much more frequently and much more seriously affected. The greater the extension of the diphtherial process upon the conjunctiva the more certain is corneal suppuration to occur. If the entire area of the conjunctiva is infiltrated and rigid the cornea is probably always irretrievably lost.

The general condition of the little patients is very much disturbed. They have high fever and are greatly prostrated. Weakly children not infrequently succumb to the severity of the general disease. The prognosis, therefore, in the severe cases is very serious, not only as regards the eye, but also with respect to life itself.

We owe the first exact description of conjunctival diphtheria to von Graefe, who, in Berlin, had an opportunity of seeing many cases of this disease. His description relates to the deep variety, of which he distinguishes two groups of cases. In the first group—that of diphtheria en plaques—constituted by the lighter cases, the diphtherial spots are found under the form of large or small islands, which occur especially on the conjunctiva of the lids and between which lie areas of tissue that is not so much diseased. In the severer cases, on the other hand, the diphtherial foci rapidly coalesce, so that the entire conjunctiva becomes rigid and bloodless (confluent diphtheria).

154. Etiology.—That the two forms just described, which differ so much in their appearance and course, are, nevertheless, the same disease, namely diphtheria, is proved from the fact that Löffler's bacilli are found in the conjunctival secretion in both. Often, too, the patients present other important and undoubted diphtherial affections. Small-sized diphtherial patches are frequently found at the edges or angles of the lids, the

nostrils, or the angles of the mouth; sometimes even there is a fully developed nasal or pharyngeal diphtheria.

Diphtheria of the conjunctiva is mainly observed in those countries where diphtheria of all sorts is a frequent occurrence,[13] and occurs especially at times when an epidemic of pharyngeal diphtheria is prevailing. It can often be proved that children affected with diphtheria of the conjunctiva had previously been thrown with others, who soon afterward developed pharyngeal diphtheria; and such children may themselves in their turn spread the disease to others still. The predisposition to diphtheria diminishes with the age. Consequently, diphtheria of the conjunctiva usually attacks children, and most frequently those between the second and eighth year of life. Adults are only exceptionally attacked, and then generally by one of the lighter forms.

After Löffler had discovered in the membranes of pharyngeal diphtheria the bacillus that bears his name it was soon after shown to be present in diphtheria of the conjunctiva also (Babes, Kolisko and Paltauf, and others). On the other hand, no one supposed that the cases in which a membrane forms upon the conjunctiva must also be regarded as diphtheria until the presence of the Löffler bacillus was demonstrated in them too (first by Gallemaerts). The same thing occurred in this case as in that of pharyngeal diphtheria and laryngeal croup, whose etiological identity was long unrecognized. It is supposed that membranous inflammation of a mucous membrane implies a feebler action of the diphtheria bacilli than does diphtheritic inflammation proper, the comparatively slight effect of the bacilli in the former case being due either to their having lost their virulence or to the patient's being more refractory to the influence. But apart from this, the severity of the inflammation is influenced by the fact that beside the Löffler bacillus other germs occur on the inflamed conjunctiva, such as the staphylococcus and streptococcus. In fact, the streptococcus by itself is competent to produce a disease of the clinical aspect of conjunctival diphtheria; and in my clinic actually the severest cases were those in which the streptococcus alone was present, the slighter or croupous cases being associated with the Löffler bacilli. Such cases of severe streptococcus conjunctivitis are found, particularly in children, as a result of the acute exanthemata (small-pox, measles, and scarlet fever), which may thus cause blindness.

155. Treatment.—In the severe cases of diphtheria of the conjunctiva the injection of antitoxin is indicated. The injection is best made beneath the skin of the lids. In addition the serum is often instilled into the conjunctival sac. In the lighter (croupous) cases we may content ourselves with employing local treatment alone. In the first stage of the disease this is chiefly limited to careful cleansing of the eye, for which purpose the best thing for us to employ is a weak antiseptic liquid (beside solutions of corrosive sublimate and potassium permanganate, a solution of quinine is particularly recommended). Cold compresses, which would seem to be indicated by the great swelling and redness of the lids, must be applied only when the conjunctival circulation is not too seriously em-

[13] [In America severe cases of conjunctival diphtheria are of rare occurrence.—D.]

barrassed by the diphtherial infiltration. Otherwise, it is better to employ warm compresses which by dilating the blood-vessels increase the circulation. As regards the conjunctiva itself, Fieuzal has recommended painting it with lemon juice; and painting it with strong sublimate solution (1:1,000), either directly or after the removal of the membrane, if present, is highly spoken of. Except for this purpose there is no object in removing the membranes in the croupous variety, since these at once re-form. When, after separation of the membranes or the slough, the conjunctiva has become strongly congested, soft, and succulent, and the secretion begins to be abundant, we may commence the application of a nitrate-of-silver solution, by means of which we bring the swollen conjunctiva more rapidly back to its normal state. In so doing we must first proceed with great caution, use a pretty weak solution (one per cent) and discontinue the application at once if membranes or deep infiltrations once more develop.

We keep on making the applications to the conjunctiva as long as the latter is red and swollen and discharges a copious secretion. If in the deep form, after the subsidence of the disease, partial necrosis and sloughing of the conjunctiva have set in, we try during the subsequent period of cicatrization to oppose, as far as possible, the formation of adhesions between the lids and the eyeball (doing this by frequently drawing the lid away from the eyeball or by laying a pledget soaked in oil between the two), for adhesions once formed can be removed only by an operation. Complications affecting the cornea are to be treated according to the rules that will be given further on for purulent inflammation of the cornea in general.

All operative procedures, whether upon the cornea or upon the lids, should be avoided in the first stage, as the wounds thus produced generally become diphtherial too.

In consideration of the very infectious character of diphtheria, our special aim must be *prophylaxis*. Accordingly, we remove from the vicinity of the patient all persons who are not indispensably necessary for purposes of nursing; but, most of all, we insist upon the removal of children, as these are particularly susceptible to infection. If diphtheria has attacked only one of the patient's eyes, the other must be protected against infection by a carefully applied occluding bandage, just as in gonorrhœal conjunctivitis.

The persons who have charge of the patient must be particularly enjoined to cleanse the hands carefully after touching the diseased eye, to destroy at once the materials employed in cleansing, etc.

156. **Croupous Membranes on the Conjunctiva.**—Croup and diphtheria are primarily anatomical terms denoting definite forms of inflammation. Croupous inflammation is characterized by the deposition of an exudate upon the surface of a tissue, where, by coagulation, it hardens into a membrane. The essence of the diphtheritic inflammation, on the contrary, consists in the exudation of a great mass of material within the tissue itself, with consecutive necrosis of the latter. Diphtheritic inflammation may be regarded as a croupous inflammation carried to a higher pitch, in so far as the same

injurious cause may, when acting to a slight extent, produce a croupous, when acting to a greater extent, a diphtheritic inflammation of the mucous membrane. Sourdille has demonstrated experimentally that by painting the conjunctiva with ammonia one can at will produce either the croupous or the diphtheritic form of inflammation, according to the intensity, greater or less, with which the agent is applied. We meet with the same experience in our medical practice, when, by making too strong or too frequent applications of the silver solution to an inflamed conjunctiva, we produce a croupous coating upon it, and then, in spite of this result, keep on with the application. In this case a diphtheritic inflammation with partial necrosis of the tissue will ensue. Chemical irritants of an organic nature may bring about the same result. Thus the repeated application of the jequirity infusion produces first a croupous, afterward a diphtheritic inflammation. And, furthermore, the same thing holds good for many of those inflammations of the conjunctiva that are caused by micro-organisms. Thus in a gonorrhœal conjunctivitis, when the inflammation attains a high degree of severity, either a croupous coating or a diphtheritic infiltration of isolated portions of the conjunctiva may be observed, and such cases are often regarded as genuine diphtheria.

Accordingly, the same clinical picture—e. g., that of a diphtheritic conjunctivitis—may be produced by the most various kinds of pathogenic agents, both of a chemical and a parasitic nature; and, on the other hand, the same pathogenic agent—e. g., the Löffler bacillus—may give rise to a variety of clinical pictures—i. e., to both croupous and diphtheritic inflammation and in exceptional cases to nothing more than a very slight conjunctivitis having the appearances of a catarrhal inflammation.

It is not tenable, therefore, as has hitherto been done, to employ the expressions croup and diphtheria of the conjunctiva both to characterize certain anatomical changes, and also to denote definite types of disease, each of single etiology. With regard to the expression diphtheritis, I have adhered to Roser's proposition, using the word diphtheritis as an anatomical term for that variety of inflammation in which the exudate undergoes coagulation within the tissue itself. On the other hand, diphtheria and diphtherial are used in an etiological sense to denote those affections which, whatever appearance they may present, are caused by the Löffler bacillus. The expression croupous conjunctivitis should be used simply as an anatomical term.

Formerly most of the spontaneously developing conjunctivitides that were associated with the formation of a membrane were comprehended under the terms *conjunctivitis cruposa* or *membranacea*, and thought to constitute a single independent disease. But recent bacteriological investigations have shown that the most various pathogenic agents may give rise to the formation of a membrane upon the conjunctiva. As far as is at present known, a croupous conjunctivitis may originate from the following causes:

(*a*) *Spontaneously* developing croupous conjunctivitis. This usually runs an *acute* course, and is the kind that was formerly described under the name of conjunctivitis cruposa as a distinct disease. We have seen above that a part of these cases, being caused by the Löffler bacillus, are to be attributed to diphtheria. This knowledge is practically important, because we know that even these apparently light cases of conjunctivitis may by transfer to others produce severe diphtheria of the conjunctiva or of the pharynx, and that we must consequently apply to them all customary precautionary measures.

Croupous inflammations, sometimes of a serious character, are caused by the streptococcus, and less severe kinds by the pneumococcus, meningococcus, etc. Among the cases that run a light course belong also those of acute catarrh, that are produced by the bacillus of Weeks, and are associated with the formation of membrane (Morax); and that the conjunctivitis caused by the gonococcus not infrequently shows membrane formation has already been stated above. A severe form of conjunctivitis with membrane is caused by the thrush fungus (Pichler).

To the cases of membrane formation upon the conjunctiva that pursue a more *chronic* course belong the very rare instances of *herpes iris* of the conjunctiva. These latter can readily be diagnosticated, provided the characteristic exanthem of herpes iris (a central reddened or pigmented area of skin surrounded by a wall of vesicles) is also to be found upon the skin. This, however, is not always present. Sometimes, too, a formation of membrane like that upon the conjunctiva occurs upon the mucous membrane of the mouth. In some cases the disease recurs frequently.

Furthermore, there have been described cases of membrane formation upon the conjunctiva which were peculiarly chronic cases—lasting for months, or even years—the nature of which is still doubtful (Arlt, Hulme, Morton, and others).

(*b*) By the application to it of external irritants of a *chemical nature* the conjunctiva may take on an inflammation with membrane formation. As already mentioned, such irritant substances include bodies both inorganic and organic, like ammonia, nitrate-of-silver solution, and jequirity infusion.

(*c*) *Losses of substance* on the conjunctiva (and the same thing is seen in other mucous membranes) very soon become covered with a membrane of coagulated fibrin, under which the healing of the wounds proceeds. This process is observed after operations (e.g., tenotomy), injuries, and also spontaneously developing wounds—as, for example, those occurring after rupture of pemphigus vesicles (see page 203).

VI. Conjunctivitis Eczematosa[14]

157. Symptoms.—In its simplest, typical form, conjunctivitis eczematosa presents the following picture: A little red eminence, of about

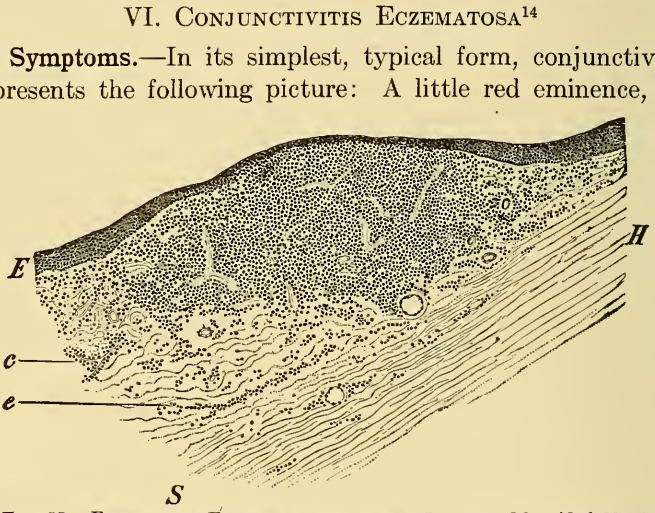

Fig. 55.—Eczematous Efflorescence in the Limbus. Magnified 62×1.

The sclera, *S*, is distinguished by its more delicate fibrillation and its blood-vessels from the more homogeneous, non-vascular cornea, *H*. The nodule is situated at a point corresponding to the boundary between the two membranes but more over the sclera than over the cornea. It consists of densely packed round cells, between which the blood-vessels are recognizable under the form of lighter-colored striæ. In the vicinity of the nodule the vessels of the conjunctiva (*c*) and episclera (*e*) are bordered by extravasated leucocytes. The epithelium (*E*) of the conjunctiva is bulged forward by the nodule, and at the apex of the latter is thinned, and, owing to the penetration of the round cells into the epithelial layer itself, has lost the sharp border ordinarily existing between it and the connective tissue.

[14] Synonyms: Conjunctivitis lymphatica (scrofulosa, phlyctænulosa, pustulosa, exanthematica), herpes conjunctivæ (Stellwag), [phlyctænulosis]. Several of these synonyms are derived from the view that the efflorescence on the conjunctiva or cornea is a hollow vesicle filled with fluid (φλύκταινα [bladder], pustula, herpes vesicle). But the efflorescence is in reality never a vesicle, but a solid, though soft projection, which is formed chiefly by an accumulation of leucocytes (Figs. 55 and 56). The softening and liquefaction of this cellular mass do not begin in the interior of the projection, but at its apex, so that no cavity is formed (vesicle or pustule), but a loss of substance (ulcer) occurs, lying upon the free surface at the apex. The name herpes corneæ, moreover, can give rise to a confusion with true herpes corneæ (herpes febrilis and herpes zoster, see §§ 227 and 228). [The term phlyctænular conjunctivitis, though anatomically incorrect, is so firmly fixed by usage that it would seem desirable to retain it, to the exclusion of other more logically formed synonyms.—D.]

the size of a millet seed, develops at some point upon the limbus of the conjunctiva. This is the *efflorescence* (Fig. 55). In the beginning it is conical, its apex being covered by the epithelium of the conjunctiva. In a short time the epithelium at the summit of the efflorescence separates, and the tissue that lay beneath it breaks down, so that the apex of the cone, so to speak, melts away; and the cone itself bears on its top a minute gray ulcer, which thus lies above the level of the neighboring, healthy conjunctiva. By a continuation of the breaking-down process the cone at length disappears entirely, the ulcer sinks to the level of the conjunctiva, and speedily becomes clean and then covered with epithelium. Thus the ulcer heals, without a visible mark being left upon the conjunctiva.

As the efflorescence springs up, the adjacent part of the conjunctiva becomes hyperæmic, the injected vessels being directed from all sides toward the little nodule. Hence, the reddened portion of the conjunctiva shows the form of a triangular sector, the apex of which lies in the limbus and corresponds to the nodule. The remainder of the conjunctiva is perfectly free from congestion.

The simplest type of conjunctivitis eczematosa, therefore, consists in the formation of a sharply circumscribed, nodular exudate, to which there corresponds an injected district of the conjunctiva. Conjunctivitis eczematosa is hence a *focal* affection of the conjunctiva of the eyeball, and is thus distinguished from all the varieties of conjunctival inflammation hitherto described, which are diffuse inflammations, in that they extend in a uniform fashion over large sections of the conjunctiva.

158. The clinical pictures which conjunctivitis eczematosa actually exhibits present modifications of the simplest type above described which differ most widely from each other. These modifications concern—

(*a*) The *number* of the efflorescences. It is rare that we find but one of these; generally there are several, and not infrequently a good many, present at the same time. The fewer they are, the larger they generally grow; in rare cases they attain almost the size of a lentil. Where there are many nodules present they are small; often we find the entire limbus, or even the cornea itself, covered with very minute eminences, so that the surface of the eyeball looks as if fine sand had been strewn over it. Such very small nodules commonly disappear in a few days by resorption, without any preliminary disintegration. When multiple efflorescences are present, the injected portions of the bulbar conjunctiva belonging to the separate nodules become confluent, and the conjunctiva then appears reddened all over, so that the focal character of the diseases is obscured, and declares itself only by the presence of separate nodular exudates. So, too, when the inflammation is great, the palpebral conjunctiva also participates in the injection, so that, in that case, conjunctivitis eczematosa is no longer an affection limited to the bulbar conjunctiva.

(*b*) The *site* of the efflorescences may be not only in the limbus itself but also exterior to the latter, in the anterior segment of the bulbar conjunctiva, and likewise interior to the limbus, in the cornea itself. In the latter the small gray nodules are situated in the most superficial layers of the cornea (Fig. 56). By the breaking down of the nodule there is produced in the cornea a very shallow loss of substance, which leaves scarcely any opacity. Often, however, the affection assumes a more serious form from the fact that the exudations have a tendency to spread farther in the cornea, extending either into the depth of the latter or along its surface. If the infiltration extends through Bowman's membrane into the parenchyma proper of the cornea, an ulcer is produced by its disintegration which penetrates more deeply and can even perforate the cornea. In that case, after the ulcer heals, a permanent opacity remains.

(*c*) The corneal ulcers which result from the efflorescences may assume a serpiginous character—that is, they may spread by a sort of creeping process along the surface of the cornea. In this way is produced the *vascular fasciculus* (Fischer), which is also called keratitis fascicularis. This affection begins by the development of a small ulcer out of an efflorescence at the rim of the cornea. After some days this ulcer becomes clean in its peripheral half—that is, in the part turned toward the corneal rim. At the same time, in accordance with the ordinary behavior of regressive corneal ulcers, blood-vessels develop, which run from the limbus to that edge of the ulcer which is healing. These serve to keep up the process of cicatrization. But in the meantime, that margin of the ulcer which is toward the center has remained infiltrated and gray. Inasmuch as at this place the infiltration and the subsequent purulent disintegration keep on extending, the ulcer constantly advances toward the center of the cornea, while on its peripheral side it heals equally fast, and trails the blood-vessels after it. The vascular fasciculus accordingly appears under the guise of a narrow red band formed of blood-vessels (hence the name), and extending from the corneal margin some distance into the cornea. At its apex it bears a small gray crescent, the infiltrated, advancing margin of the ulcer. The arrest and recession of the process first occur when the ulcer is completely healed. Until this has taken place the vascular fasciculus

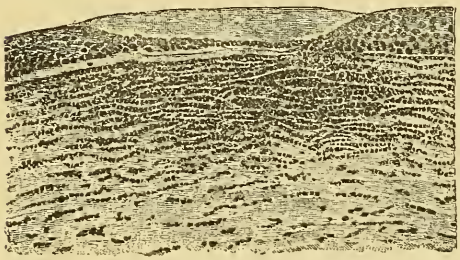

FIG. 56.—EFFLORESCENCE IN THE CORNEA. (After Hayashi.)

The efflorescence which is two days old consists of a deposition of leucocytes in a series of rows between the superficial lamellæ of the cornea, which in attenuated form are still to be recognized between the cells. Over the right side of the accumulation Bowman's membrane has a gap, and the spot in the infiltrate lying just beneath this point no longer shows any nuclear staining. Still more widespread is the necrosis in the epithelium which for almost the entire extent of the accumulation is transformed into a mass destitute of recognizable cell nuclei. Thus the disintegration of the efflorescence is preparing the way for the formation of an ulcer.

can advance far into the cornea, to its center or even beyond it. The small ulcer, however, always remains superficial, and a perforation of the cornea due to it is never observed. When the vascular fasciculus has at length come to a standstill, the vessels gradually disappear from it, and there only remains a superficial opacity of the cornea which corresponds in shape to the long-drawn-out form of the vascular fasciculus. This opacity never clears up again completely, and hence, when found at any time during the whole subsequent life of the patient, enables us to diagnosticate the previous existence of a vascular fasciculus.

(*d*) The severest cases of conjunctivitis eczematosa are those in which the exudation from the start makes its appearance in the deep layers of the cornea as a widely diffused *deep-lying* infiltration. We then find the cornea occupied to a considerable extent by an opacity of a uniform gray or yellowish color, with hazy outline and seated in the deeper layers of the cornea; the surface of the cornea over it is stippled. In the bad cases the infiltrate, originally gray, becomes more and more yellow, and finally breaks down into pus, so that an extensive loss of substance is produced in the cornea. In the benign cases, on the contrary, the infiltrate gradually disappears again by resorption, and the cornea regains its transparency either wholly or in part. It is astonishing to what extent even extensive infiltrates can undergo resolution.

(*e*) Instead of appearing as separate circumscribed foci, the exudate may occur under the form of a continuous new formation of tissue upon the surface of the cornea—that is, under the form of *pannus*. This is called pannus eczematosus, to distinguish it from trachomatous pannus. It does not, like the latter, show a predilection for the upper part of the cornea, but develops from any spot whatever upon the corneal margin. It is ordinarily thin and not very vascular, and is quite apt to undergo complete resolution.

The authors separate the eczematous affections of the conjunctiva from those of the cornea; they speak of conjunctivitis and of keratitis eczematosa or phlyctænulosa according as the efflorescence is located upon the conjunctiva or the cornea. In this way, for mere love of system, a picture of disease that, clinically speaking, is a perfect unit, is torn in two. In fact, we have here really but one single disease, which is localized sometimes in one place, sometimes in another. Frequently enough we find in the same eye, at the same time, one efflorescence in the conjunctiva of the eyeball, a second in the limbus, half in the conjunctiva and half in the cornea, and a third upon the cornea itself (very exceptionally efflorescences occur even on the conjunctiva of the lids). Hence, in the foregoing description of the disease the expression conjunctivitis eczematosa is employed for the disease in general, no matter upon what part of the surface of the eyeball it is localized. This can be done without doing violence to anatomy, inasmuch as the outermost layer of the cornea must be looked upon as the continuation of the conjunctiva over the cornea. Accordingly, we can regard the involvement of the cornea in conjunctivitis eczematosa as an involvement of the "conjunctival layer" of the cornea. For the same reason we regard pannus trachomatosus as one of the symptoms of conjunctivitis trachomatosa, and not as an independent affection of the cornea.

The efflorescences begin with an accumulation of leucocytes beneath Bowman's membrane (Fig. 56). This is so adherent that it cannot be lifted up by the accumulated cells like the delicate conjunctiva scleræ, and for this reason the efflorescences on the cornea, in contrast with those on the limbus, are not at all or but little prominent. When the infiltration of the uppermost lamellæ of the cornea has reached a certain point, the epithelium over it and then also ultimately Bowman's membrane break down. When this occurs the true loss of substance begins, and the efflorescence is converted into an ulcer. Pannus scrofulosus, too, usually begins beneath Bowman's membrane: contrasting in this regard with pannus trachomatosus.

159. Diagnosis.—The differential diagnosis between conjunctivitis eczematosa and the other affections of the conjunctiva and the cornea is, as a rule, easily made. The characteristic mark of the former lies in the focal character of the affection as well as in its localization upon and immediately about the cornea.

Only one other variety of conjunctivitis, the conjunctivitis ex acne, shares this peculiarity with conjunctivitis eczematosa; but that disease is readily distinguished from the latter by the accompanying acne rosacea upon the face (see page 202). In spring catarrh also (page 204) little nodules occur upon the limbus, but never break down into ulcers. Of the diffuse inflammations of the conjunctiva, catarrh might be confounded with conjunctivitis eczematosa. For in intense and obstinate cases of conjunctivitis eczematosa, the affection spreads to the palpebral conjunctiva, which may become very much reddened, swollen, or even velvety; in that case, too, a mucous or muco-purulent secretion forms upon the conjunctiva. It is often difficult to distinguish between such cases and catarrh, especially if just at the moment of examination there are no characteristic efflorescences present upon the conjunctiva of the eyeball. Fortunately, a mistake in diagnosis does no harm, since, with such a condition of the conjunctiva existing, gentle cauterization with the nitrate-of-silver solution is always indicated, no matter what the origin of the disease. The pustular form of acute conjunctival catarrh forms a sort of intermediate stage between catarrhal conjunctivitis and conjunctivitis eczematosa (see page 141).

Eczematous pannus is to be chiefly distinguished from pannus trachomatosus by the fact that an exact examination of the conjunctiva of the lids and of the retrotarsal fold either discloses the changes of trachoma or establishes the fact of their absence.

Corneal ulcers which have been preceded by the efflorescences of a conjunctivitis eczematosa sometimes are located so very near the margin of the cornea that they extend into the limbus conjunctivæ; and, since corneal ulcers as peripherally situated as this occur only in connection with conjunctivitis eczematosa, the diagnosis of an antecedent conjunctivitis eczematosa can be made even years after it has elapsed from scars having such a situation (Fig. 92).

The *vascular fasciculus* can readily be confounded with an ordinary corneal ulcer, to which, in the course of healing, vessels have made their way from the limbus so as to form a red bridge between the limbus and the ulcer. In such a case there is no fear of the ulcer's extending into the pupillary area of the cornea, and, after the ulcer heals, only a small, rounded macula is left, and not a long, opaque stria, as in the case of the vascular fasciculus. The distinction between these two affections can be made as follows: In the vascular fasciculus, the advancing margin of the ulcer, infiltrated with gray, is readily visible; the blood-vessels as they run up to it lie in the furrow which the ulcer has channeled in the course of its progress—lie, therefore, at or below the level of the corneal surface. In the case of a simple ulcer with development of vessels, this furrow is wanting, and the vessels have a quite superficial situation.

160. Symptoms.—Conjunctivitis eczematosa is generally accompanied by profuse lachrymation. Mucous or muco-purulent secretion, on the contrary, such as occurs in catarrh, is not present as a rule; hence the lids do not ordinarily stick together in the mornings. The only exception to this is formed by those old cases in which the inflammatory process has passed over to the palpebral conjunctiva, and has, we may say, thrown it into a state of concomitant catarrhal inflammation.

The *subjective symptoms* consist of photophobia combined with spasm of the lids (blepharospasm). Slight in some cases, in others they reach an extraordinary pitch; children creep into a dark corner of the room, bury their faces in their hands, and struggle so violently against any attempt at opening their eyes that the examination on the part of the physician is made with great difficulty. The intensity of these symptoms bears no definite relation to the severity of the disease; in fact, it is precisely in that form of corneal affection which spreads more extensively and penetrates more deeply that the evidences of irritation are often pretty slight. The annoyance suffered, contrary to what takes place in conjunctival catarrh, is, generally speaking, greater in the morning than in the afternoon and evening.

The photophobia in many cases persists obstinately for months. The parents then bring the children to the physician, with the statement that they have been "blind" for such or such a number of weeks. The children offer the greatest resistance to the forcible opening of the eyes, especially when there are excoriations at the external commissure, which give pain and are prone to bleed when the lids are separated. Hence, in such cases the palpebral fissure is to be opened cautiously and not too wide, so as to avoid making the struggles of the children still greater. The lids, and especially the upper lid, are rendered œdematous by the constant blepharospasm, because the veins of the lids, which pass between the fibers of the orbicularis, are compressed by persistent contraction of this muscle. Furthermore, a state of inversion of the lids (entropion spasticum) may be induced by this forcible squeezing together of the eyelids. Finally, cases have been described in which children who have suffered for a long time from blepharospasm were perfectly blind after the disappearance of this symptom (von Graefe, Schirmer, Leber, and others). Such blindness is transient. As in the most cases no objective changes were demonstrable as the cause of the blindness, the latter is probably to be regarded as a central, purely functional affair, like hysterical amaurosis. In other words, the light stimulus is conveyed to the cortex of the brain, but does not there pass the threshold, i.e., does not reach the consciousness.

161. Course and Prognosis.—A single typical efflorescence upon the limbus passes through all its phases up to complete subsidence in eight to fourteen days. If several efflorescences are present, the process of cure requires a proportionately longer time. Nevertheless, the disease would not last so very long if it limited itself to a single attack. This, however, is but rarely the case. Usually, after a period of quiescence, or even before the first attack of inflammation has quite run its course, the eye becomes red again, and new nodules shoot up in or near the limbus.

Thus the disease may, with longer or shorter intermissions, last on for months or years. Its beginning occurs in childhood; the separate attacks, however, are often protracted until the time of puberty, or sometimes even later; the affection being situated now in one eye, now in the other, then in both at the same time. Finally, the attacks become less and less frequent, and at length cease altogether.

The constant wetting of the lids by the tears frequently leads to blepharitis, to eczema of the skin covering the lids, and, as a consequence, to ectropion of the lower lid. Excoriations are frequently present at the external angles of the lids, and blepharophimosis often develops later on.

The *prognosis* of any single attack of conjunctivitis eczematosa is favorable, in so far as the eye is but rarely rendered entirely blind by it. Superficial efflorescences disappear without leaving a trace behind; ulcers which penetrate into the parenchyma proper of the cornea leave permanent opacities, which, however, are in most cases thin and superficial (maculæ of the cornea). On the other hand, the prognosis of the disease as a whole is rendered unfavorable by the frequent recurrences. In persons who have gone through many recurrences of conjunctivitis eczematosa the corneæ often bear quite a number of maculæ as signs of past attacks. Thus the sight is impaired, squint or myopia often develops, and the persons thus affected often become incapable of doing fine work. In addition to this, children, in consequence of the frequently repeated inflammation of the eyes, fall behind in their physical and mental development. Finally a not infrequent outcome is secondary blindness, often occurring many years after the inflammation has passed, and representing the after-results of the corneal scars with inclusion of the iris which remain after the corneal ulcers.

162. Etiology.—Conjunctivitis eczematosa is one of the most frequent of eye diseases,[15] and has its origin in the scrofulous diathesis. Like the latter, it is a disease of childhood and youth. In very young children —those under the age of one year—it occurs but seldom, and it generally ceases at the time of puberty. Adults are attacked by it only in case they have carried the disease along with them from their childhood. The enormous majority of children affected with conjunctivitis eczematosa belong to the poorer classes. Such children receive insufficient and unsuitable nourishment, live in damp, poorly ventilated dwellings, and are kept constantly dirty. Other children affected are those who, though once healthy, have become run down as a result of other diseases (scarlet fever, measles, typhoid fever, whooping cough, etc.). Such children as these look either pale and thin or bloated and spongy-looking. The glands at the lower jaw, in the neck, and in front of the ear, are swollen. Partly as a result of the suppuration of these glands, and partly as a result of the breaking down of the scrofulous infiltrations in the skin, ulcers and fistu-

[15] [Less frequent in America than in Europe.—D.]

lous passages are produced, which require months and years for their cure, and leave characteristic and disfiguring scars behind. Patches of moist eczema occur at various spots upon the body, most frequently upon the face; and the constant coryza from which many of these children suffer is to be attributed to an eczematous affection of the nasal mucous membrane. Adenoid vegetations, too, are not rare. In the lids we find blepharitis. The nose and the upper lip are rendered thick by frequently recurring attacks of inflammation. More profound affections that occur are caries of bones (caries of the petrous bones appearing under the form of an otorrhœa being frequent), tuberculosis, and in girls, delayed and irregular menstruation.

Some of the above-mentioned symptoms of scrofula, and often several of them at once, accompany most cases of conjunctivitis eczematosa. Sometimes, though rarely in comparison with other cases, the disease is observed in an individual who otherwise is quite healthy, just in the same way that other indications of scrofula occur at times as altogether isolated phenomena.

In accord with the precedent set by the older authors, I formerly called conjunctivitis eczematosa by the name of conjunctivitis lymphatica (or scrofulosa) on account of its undeniable connection with scrofulosis. Later, following many recent authors, I replaced this etiological term by that of conjunctivitis eczematosa. A conjunctivitis which is undoubtedly one of the symptoms of eczema I have observed on a few occasions in adults who had been attacked by a very extensive eczema and at the same time by a conjunctivitis that was associated with marginal infiltrates of the cornea which actually led to perforation. Although the clinical picture was in some respects like that of severe conjunctivitis eczematosa, yet it was a different disease that was in question. Whether the conjunctivitis of scrofulous children can properly be characterized as eczematous we shall first be able to tell when the true cause of the conjunctivitis, on the one hand, and of the scrofulous eczema, on the other, has been determined. If we question ourselves as to the nature of the efflorescences of conjunctivitis eczematosa we must keep two facts in mind.

(1) The efflorescences when quite recent are sterile, containing neither the ordinary pyogenic germs nor tubercle bacilli. Inoculation from them into a test animal does not lead to tuberculosis. Hence it cannot be assumed that, like other inflammations of the conjunctiva, they are referable to ectogenous infection, and as little are they to be regarded in the light of true tuberculous nodules.

(2) In a great number of patients with conjunctivitis eczematosa there are changes which are certainly tuberculous, most frequently appearing under the form of scrofulous lesions of the glands and bones, and not infrequently also of pulmonary tuberculosis. But even in those patients who otherwise show no clinically demonstrable evidence of scrofula or tuberculosis, the examination with tuberculin (by subcutaneous injection or cutaneous inoculation) proves with rare exceptions the presence of latent tuberculosis. If then the efflorescences are not actual tuberculosis nodules and yet, on the other hand, they occur with such preponderating frequency in tuberculous men, we should not be far out in explaining them as being due to the action of toxic substances, in the same way as, for example, nodules develop in tuberculous individuals after the rubbing of a tuberculin ointment into the skin (Moro). In harmony with such a connection is the observation that after the inoculation of tuberculin in children whose eyes were hitherto healthy efflorescences sometimes appear on the conjunctiva after an incubation period of about 14 days. [It has been suggested that the development of efflorescences in tuberculous subjects is an example of anaphylaxis (Verhoeff). In any

event it seems clear that the relationship of this disease to tuberculosis is at most an indirect one. The results of treatment point to some other contributory or perhaps essential cause (disturbance of metabolism due to gastro-intestinal toxæmia, pharyngeal and tonsillar infection, etc.).—D.]

163. Therapy.—In the lighter cases, the *local treatment* consists in the application of irritants, of which calomel and the yellow-precipitate (or Pagenstecher's) ointment (page 60) are most in use. The calomel in a finely powdered state is sprinkled in a thin layer upon the conjunctiva of the lower lid [or directly on the cornea] by means of a camel's-hair brush; the precipitate ointment (0.05 to 0.15 of yellow precipitate to 5 of fat), on the other hand, is introduced into the conjunctival sac by means of a glass rod, and is then rubbed about with the lids, so as to be distributed over the whole conjunctiva.

Both remedies are irritant in their action; the precipitate ointment more so than the calomel. Hence, in the beginning of the disease, where the eyes are in a marked state of irritation, it is best to employ calomel; and subsequently, when the inflammatory symptoms are diminishing, to replace this by the yellow ointment. The remedies mentioned are to be applied only once a day, but it is advisable to keep on with their application for quite a long time, in order to prevent relapses. Both find a contraindication in the presence of recent infiltrates or progressive ulcers in the cornea. In such cases, before having recourse to irritant remedies, we must wait, meanwhile employing atropine, until the process of infiltration has subsided or the ulcer has become clean. Pannus and vascular fasciculi do not contraindicate calomel or the yellow ointment.

In the cases of ulcers of larger size covered with exudation, as well as in the case of deep infiltration of the cornea, hot moist compresses applied several times a day for one or two hours at a time over the closed eyes, prove most efficient. With regard to the treatment of deep ulcers and of the cicatrices that follow ulcers, the general rules set forth under the head of affections of the cornea are applicable. A bandage should be applied only in case of imperative necessity (e. g., when there are ulcers penetrating deeply into the cornea); otherwise its use had better be avoided. It hinders the ready escape of the tears which are so copiously secreted, and, as it very soon becomes wet through with the secretion, it is likely to set up eczema of the skin of the lids.

In conjunctivitis eczematosa, *general treatment*, conducted with due regard to the etiology, is of especial importance. The child's nourishment should be strengthening and administered at regular hours. [Sweets, especially candy, cakes, pies and pastry of all kinds should be forbidden, and only very plain, but substantial food allowed. It would seem that regulation of the diet alone may do a great deal toward effecting a cure in these cases.—D.] Care must be taken that the dwelling place be dry and well

ventilated, and the child should be sedulously kept out in the open air, irrespective of any photophobia that may exist. Indeed, in any case, we must not yield too much to this symptom of photophobia, and it would be quite a mistake to keep children in a dark room just because they shun the light. As invigorating measures, sponging with cold water are of service; also a sojourn in the country, especially at the mountains or the seashore. After the acute inflammation has run its course, the use of brine baths or of sea baths is of great service in preventing relapses. Unfortunately, the carrying out of all these regulations is only too often frustrated by the force of circumstances and by the poverty of the patients.

The medicinal treatment of scrofula consists in the administration of cod-liver oil and of the preparations of iodine, iron, arsenic and quinine. [Calomel in broken doses (0.003–0.006 gm. three or four times a day) is also used with good results. It may be given in these doses for weeks at a time with advantage.—D.]

The sort of remedy employed and its dose must be adapted to each individual case. In case of a clinically demonstrable tuberculosis methodically continued tuberculin injections may be tried. [These are quite helpful, especially in preventing relapses (Derby, Wilder, Norman).—D.] Furthermore, the cure of the eye disease is favorably influenced by treating any coexisting scrofulous affections, especially blepharitis, and also eczema of the face and of the nasal mucous membrane, [diseased tonsils] and adenoid vegetations. For the affection first named the application of white-precipitate ointment (1 to 2 per cent) or of ichthyol ointment (5 per cent) does good service. In blepharitis this ointment is smeared at night over the closed palpebral fissure. Eczematous spots upon the face are covered with a pledget of linen smeared with the ointment, and, to relieve eczematous coryza, the ointment is introduced from the anterior nares as far into the nose as possible and rubbed in. For relieving moist eczema of the face, we can also employ with great advantage a 5- to 10-per-cent nitrate-of-silver solution applied with the brush, after removal of the crusts, to the raw cutaneous surface, which thus becomes covered with a thin eschar, under which the raw spots heal rapidly. This application must be repeated at first daily, afterwards at intervals of several days, and be kept up as long as crusts continue to form.

Many of the patients, who generally belong to the poorest classes, suffer from head lice, the removal of which often has a strikingly favorable effect on the eye trouble.

If we undertake the inspersion of *calomel* in patients to whom at the same time iodine is being administered internally, we not infrequently observe a strong corrosive action from the calomel; for the latter forms with the iodine excreted in the tears the very corrosive mercuric iodide (Schläfke). These two remedies, therefore, are incompatible.

In most cases the *blepharospasm* soon yields if the conjunctival trouble, which forms the basis of it, has been ameliorated by appropriate treatment. In case the spasm of the lid is particularly obstinate, we often obtain rapid improvement if we succeed in getting the child to open the eyes for some time at least once a day. We may secure this if we drop in cocaine a number of times in quick succession until the surface of the eyeball has become insensible. Other effective measures are to douche the child all over with cool water or to dust dionin powder into the conjunctival sac. These procedures must be repeated daily until the spasm of the lids has yielded. The œdematous thickening of the lids produced by their spasm is combated by massaging them.

Exanthematous Conjunctivitis

164. Besides eczema, the following exanthemata, both acute and chronic, are associated with diseases of the conjunctiva.

Acute Exanthemata.—*Measles* is regularly associated with a conjunctivitis. This appears under the form of an acute conjunctival catarrh, develops early (before the eruption of the exanthem upon the skin), and generally disappears of itself after two or three weeks without leaving any bad consequences behind.

Only in exceptional instances does the conjunctivitis of measles take on a blennorrhœal or even a diphtheritic aspect (without actually turning into true blennorrhœa or diphtheria). In such cases the cornea is endangered. During convalescence in some cases of measles, when the conjunctival inflammation had already become pretty slight, I have observed numerous Meibomian glands, both on the upper and the lower lids, becoming inflamed and ultimately suppurating (hordeola meibomiana, § 604). The purulent contents were evacuated partly through the orifices of the glands, partly upon the inner surface of the lid after breaking through the tarsus and the conjunctiva.

In *variola*, smallpox pustules not infrequently develop upon the conjunctiva, generally upon the tarsal conjunctiva near the intermarginal line. Smallpox pustules which develop upon the conjunctiva of the eyeball near the limbus are dangerous from their setting up a purulent keratitis in the adjacent part of the cornea—a condition which should not be confounded with the ulcus serpens that develops in smallpox (see § 217; for vaccine pustules, see page 210).

165. Chronic Exanthemata.—(1) Acne Rosacea Conjunctivæ.—This disease of the conjunctiva, described by Arlt, begins as follows: A minute nodule forms, with moderate symptoms of irritation, upon the limbus [or at some distance from it in the cornea (Verhoeff)]. This efflorescence breaks down after some days, and the ulcer thus produced heals without leaving any visible cicatrix behind. This affection bears the greatest resemblance to the simple typical picture of conjunctivitis eczematosa, and shares with the latter its peculiar tendency to frequent recurrence. On this account it is very tormenting to the patient. It is possible to make the differential diagnosis chiefly from the fact that conjunctivitis ex acne attacks only adults and those who are at the same time affected with acne rosacea. It is important to make the correct diagnosis, since otherwise we might labor in vain to prevent the recurrences—a thing which can be done only by a suitable and long-continued treatment of the acne rosacea. The conjunctivitis itself is most speedily cured by dusting in calomel [and the corneal lesions may be cured by peritomy (Verhoeff)].

(2) Pemphigus Conjunctivæ.—In this rare disease the conjunctiva, although reddened as a whole, displays one or two spots that are deprived of their epithelium and covered with a gray coating. While these spots are slowly undergoing cicatrization—a

process attended with shrinking of the subjacent conjunctiva—spots of the same nature appear in other places. Thus there is produced a constantly increasing cicatricial contraction of the conjunctiva, whose progress, it is true, is very slow (extending over months and years), but is irresistible. The conjunctiva becomes whitish, cloudy, and tense. First, the retrotarsal folds vanish, then folds make their appearance, stretching from the lids across to the eyeball, and finally the lids are drawn in so that trichiasis results. The conjunctiva at the same time grows continually drier, and the lachrymal secretion dries up, owing to the fact that the excretory ducts of the lachrymal gland become occluded by the shrinking of the conjunctiva. Ulcers form upon the cornea, which later gets to be clouded all over, and likewise dry upon its surface. In the bad cases the lids at length become completely adherent to the eyeball, so that the cornea is permanently covered by the lids and the eye is incurably blind (symblepharon totale). Hence, the prognosis of pemphigus is very unfavorable—the more so as both eyes are always attacked. [The cornea may remain intact for a very long time (even twenty years), and the disease itself may remain stationary for as long as sixteen years (Buck). One disagreeable symptom is the overpowering fetor that often accompanies pemphigus (Stieren).—D.]

In pemphigus of the conjunctiva, contrary to what happens in pemphigus of the skin, bullæ are only exceptionally found, their place being taken by denuded areas in the conjunctiva. This is explainable from the anatomical character of the latter. Its epithelium is so soft and delicate that it cannot, like the epidermis, be lifted up in broad layers by serous exudation, but ruptures and is thrown off in the form of shreds, so that only as an exception do we chance to see vesicles, and these very small. The raw spots upon the conjunctiva produced by the rupture of the vesicles soon become covered with a gray coating, as is so frequently the case in wounds of mucous membranes.

A further distinction between pemphigus of the conjunctiva and, indeed, of the mucous membranes generally, on the one hand, and pemphigus of the skin, on the other, consists in the fact that the process in the mucous membrane, in correspondence with the more delicate structure of the tissue, goes deeper in and hence leads to scar formation, while the pemphigus vesicles of the skin heal without leaving scars behind.

Pemphigus of the conjunctiva is rarely found in conjunction with eruptions of pemphigus upon the skin. More frequently there exists with the pemphigus of the conjunctiva an analogous affection of the mucous membrane of the mouth, throat, or nose. In these localities the pemphigus runs a course like that in the conjunctiva and may, particularly in the buccal cavity, lead to shrinking of the mucous membrane, and thus to stenosis of the mouth. But it may also happen that a lesion of the kind just described exists in the conjunctiva without pemphigus being present elsewhere in the body. That such cases as these, which were first described by von Graefe as essential phthisis of the conjunctiva, are also to be ascribed to pemphigus, is not certain, though probable.

Treatment has usually no power to restrain the process. Arsenic is administered internally for the pemphigus; and to make the patient easier, mucilaginous remedies are instilled into the eyes as in xerophthalmus (see page 185). [A number of these cases are associated with syphilis, and in these antiluetic treatment may be given, although, as a rule, with little prospect of success.—D.] Transplantation of pieces from another mucous membrane into the conjunctival sac may be tried in order to replace the conjunctiva that has been destroyed.

(3) Lupus Conjunctivæ.—Lupus of the skin sometimes is continued over the edges of the lids upon the conjunctiva. In this situation it appears as an ulcer, the bottom of which is covered with granulations in which tubercle bacilli can be made out. Lupus of the conjunctiva must therefore be regarded as a form of tuberculosis of the conjunctiva (see page 209).

In the case of *other exanthemata*, too, for instance in macular and papular syphilides, in pityriasis, psoriasis, ichthyosis, herpes iris, dermatitis herpetiformis, lepra, etc., the conjunctiva is sometimes characteristically implicated. In *lepra*, yellowish, translucent, non-vascular nodules generally develop near the margin of the cornea. These lie both in the conjunctiva and in the subjacent sclera and sometimes grow out upon the cornea. When upon the latter they not infrequently have the appearance of a new growth. There is an associated iritis sometimes combined with the formation of lepra nodules in the iris; also cyclitis. The nodules in the different parts of the eye finally break down, and the eye is lost.

VII. Conjunctivitis Vernalis[16] (Spring Catarrh)

166. Symptoms and Course.—Spring catarrh (Saemisch) is a chronic disease, persisting for years and setting up very characteristic changes in the conjunctiva, both of the tarsus and of the eyeball. The conjunctiva of the tarsus is covered with papillæ, which are broad and flattened, so as to make the conjunctiva appear like a pavement of cobble-stones (Fig. 57).

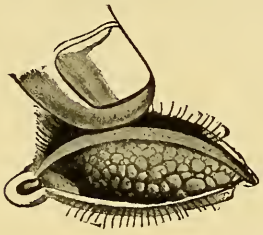

Fig. 57.

Fig. 58.

Fig. 57.—Spring Catarrh. Outgrowth on the Conjunctiva of the Lids. Taken from a man eighteen years of age who had suffered from the disease for two years. The everted lid shows the pavement-like papillæ which are largest at the upper margin of the tarsus; from this point downward they become smaller and disappear completely at a short distance from the free border of the lid. The limbus was normal.

Fig. 58.—Spring Catarrh. Outgrowth at the Limbus. Taken from a boy thirteen years of age who had had the disease for two years. The outgrowth is of unusual extent, being particularly broad and going all the way around the cornea. From it dilated veins run backward in the conjunctiva. In the conjunctiva of the upper lid nothing wrong could be made out except that it had a slightly satiny texture.

Over the whole lies a delicate, bluish-white film, as if a thin layer of milk had been poured over the conjunctiva. The changes in the conjunctiva of the eyeball are still more striking. Growths arise from the limbus at the outer and inner side of the cornea, under the form of brownish, uneven, hard nodules of gelatinous appearance (Fig. 58). These extend partly into the transparent cornea for a short distance, and still farther in the opposite direction into the conjunctiva. In contradistinction to the nodules of conjunctivitis eczematosa, which break down so speedily, these nodules never ulcerate; on the contrary, they are very stable bodies, often lasting for years with but slight variations in size.

Quite as characteristic as the changes, objectively perceptible, are the statements given by the patients. They say that during the winter they

[16] Synonym: Conjunctivitis æstivalis.

experience little or no annoyance from their eyes, but as soon as the first warm days come in spring the eyes begin to grow red and to water; the patients are greatly tormented by photophobia, and especially by a constant itching in the eyes. The warmer the weather, the greater the intensity of the subjective troubles; conversely, the patients feel easier if, for instance, there occur in summer a series of cool, rainy days. In autumn the troubles once more abate, and during the cold season they disappear completely, only to begin anew in the following spring. The difference in the objective condition at different seasons of the year is considerably less than one would suppose from the great change in the patient's subjective state, and consists principally in the eyes' being free from discoloration in winter and injected in summer, while the growths upon the conjunctiva appear but slightly smaller in winter than in summer.

Spring catarrh is not a catarrh, as the not altogether suitable name chosen for it would indicate, but is a disease sui generis. It was first described by Arlt (1846), who regarded it as a peculiar variety of conjunctivitis eczematosa. Subsequently Desmarres mentioned it under the title "hypertrophie périkératique," von Graefe as gelatinous thickening of the limbus, Hirschberg as phlyctæna pallida. Saemisch first brought into prominence the characteristic exacerbation of the disease during the warm season, and therefore called it spring catarrh, by which name it is at present commonly designated. Horner discovered the peculiar character of the tarsal conjunctiva, and thus completed the picture of the disease.

It is only in a few cases of spring catarrh that the changes in the conjunctiva of the lids and in the limbus are equally pronounced. Generally we find changes of one sort only, while those of the other sort are merely indicated or are absent altogether. The cases with proliferations of the palpebral conjunctiva without proliferation of the limbus are much more frequent than the contrary kind. Furthermore, we must not expect always to find the changes so striking as the pictures, which purposely are taken from quite far advanced cases, show. The papillæ are often quite small or altogether absent, so that the bluish-white veil which covers the conjunctiva of the tarsus is the only evidence of the disease. Sometimes a peculiar palish, persistent reddening of the eyeball, consisting of a coarse reticulate conjunctival injection combined with a distinct ciliary injection, is the only thing to indicate the trouble. In such doubtful cases the diagnosis can be made sure only by the history, of which the specially characteristic features are the dependence of the symptoms upon the external temperature, and the itching.

The proliferations in the limbus are sharply demarcated from the healthy cornea. Sometimes we can see in the cornea, parallel with the margin of the nodule, a narrow gray stria like the arcus senilis, separated from the margin of the nodule by a narrow strip of transparent cornea. This opacity usually remains permanently even after the disappearance of the proliferations. Very rarely the proliferations advance a greater distance into the cornea under the guise of a tissue which is like a pannus but which contains very few vessels and hence is pale and gelatinous-looking. This may actually cover the whole cornea.

The papillary growths in spring catarrh are the reason for this disease's being commonly regarded as trachoma, and being treated with nitrate of silver or with bluestone. The result of this treatment is that the growths do not recede and the irritation of the eye is heightened. The papillæ in vernal catarrh are much larger (broader), harder, and also paler than in trachoma, and above all are distinguished by the bluish-white lining

which is absent in papillary trachoma. Particularly important in the differential diagnosis is the history. The extremely characteristic statements in regard to the return of the disease every year in the spring often establish the correct diagnosis before we have even looked at the eye. In hay fever, to be sure, there is also generally a return of the conjunctivitis every year in the spring (see page 143). But this recurrence is an acute one, and runs its course within a few weeks, while, on the contrary, the symptoms of spring catarrh last during the whole of the warm season. [Spring catarrh and trachoma sometimes occur simultaneously, especially in Palestine, where spring catarrh is fairly frequent (Butler).—D.]

The papillæ on the tarsal conjunctiva are hard—sometimes as hard as cartilage. They are produced by overgrowth of the subconjunctival connective tissue which later undergoes hyaline degeneration. The presence of this hyaline layer is probably the reason for the bluish-white coloration of the conjunctiva. In the deeper layers there is present a somewhat more marked cell infiltration consisting mainly of plasma cells. The epithelium itself is diseased primarily as a result of the changes in the underlying tissue; it proliferates and sends offshoots into the deeper parts. The proliferations in the limbus show anatomical changes like those which the papillæ on the lid present. The secretion, which is usually scanty, is marked by the presence of numerous eosinophile cells [Herbert, Pusey], and their demonstration can be utilized for diagnosis in doubtful cases. The distinction between spring catarrh and trachoma so far as the anatomical changes are considered consists in the fact that in the former the principal thing is the connective-tissue proliferation, in the latter the infiltration with cells. This infiltration in spring catarrh consists predominately of plasma cells but in trachoma of lymphocytes. These latter become aggregated to form granulations, which again is not the case in spring catarrh.

167. Spring catarrh is a pretty rare disease, chiefly affecting the male sex and the ages of boyhood and youth. Many patients seem to be in other respects quite healthy, while others, without being scrofulous, display besides pallor of the complexion multiple swellings of the lymphatic glands, especially on the neck and lower jaw. Almost always both eyes are attacked. The disease generally keeps on making its return annually for three or four years, and often longer still, for ten or even twenty years, until finally it becomes extinct, without leaving any marked trace of its presence behind. The prognosis is therefore good as regards the ultimate outcome, but bad as regards the duration, as up to the present time we know of no remedy for curing the disease or for preventing its annual return. The cause of the disease is unknown.

Inasmuch as we are unable to cure the disease, the *treatment* must be limited to the amelioration of the subjective symptoms. We combat the inflammation by instilling mild astringents, e.g., solutions of zinc (½ per cent), boric acid (3 per cent), ichthyol (1 to 2 per cent). For the itching the frequent instillation of a weak solution of acetic acid (5 drops of acidum aceticum dilutum to 10 gm. of water) does good service. [Dionin is also said to afford much relief (Swift), and anæsthesin has helped in some cases.—D.] In many cases the dusting in of xeroform or the instillation of suprarenal extract has a good effect. To shield them from the light we direct the patients to wear protective glasses (of grayish-smoke, euphos, [or Crookes] glass). If

we have patients of pale aspect and with swollen glands we administer arsenic and iron internally. Furthermore, in summer the advice must be given to keep cool, use cold baths and showers, or reside in the mountains.

If the growths are of a considerable size they may be removed [either by ablation or by means of electrolysis or carbon dioxide snow. Diathermy (page 57) has also been employed and actinotherapy has been used with success.—D.].

168. Amyloid Degeneration of the Conjunctiva.—This rare disease has been observed mainly in Russia and the countries adjacent, and was first described by Oettingen in Dorpat. It consists in a peculiar degeneration of the conjunctiva, by reason of which the latter becomes yellowish, translucent like wax, non-vascular, and very friable. With this there is associated a considerable thickening of the membrane, so that it forms large swellings which look like new growths. The affection begins in the retrotarsal fold, and from this passes over to the conjunctiva of the eyeball and of the lids; in the lids the tarsus also is subsequently implicated in the degeneration. In a case that has lasted a long time the following clinical picture is found: The patient cannot open the eye because the two lids, transformed into large, misshapen swellings, cover it up. If the lids are drawn as far apart as possible, the wax-like conjunctiva is seen rising up under the form of a rigid prominence all about the cornea, which latter is either clear or is covered by pannus. Thick swellings, seated in the retrotarsal fold, protrude between the lids and the eyeball; the plica semilunaris also is enlarged until it forms a misshapen mass. These various swellings are so friable that they often tear when an attempt is made simply to separate the lids for examination, although when they tear they bleed very little. The disease runs a very chronic course, dragging on for years without any real inflammatory symptoms, until at length the patient is deprived of the use of his eyes by inability to open the misshapen lids.

Microscopic examination has shown that the degeneration of the conjunctiva originates from the subconjunctival cellular tissue. This at first is found to be very abundantly infiltrated with cells (adenoid proliferation). This is followed by the formation in the tissue of dully lustrous, homogeneous bodies, which are called by the name either of amyloid or of hyalin, according to the reaction that they give with stains. Hyalin may occur as a preliminary stage in the development of amyloid; but generally it represents a variety of tissue degeneration (*hyaline degeneration of the conjunctiva*) distinct from the amyloid, and showing no tendency to pass over into the latter. Hyaline and amyloid degenerations present almost precisely the same clinical picture, so that a positive distinction between the two can be made only by examining excised pieces of conjunctiva. Finally, calcification or ossification may take place in the degenerated mucous membrane.

The disease attacks people in middle life, and ordinarily both eyes are affected. Very frequently amyloid degeneration is preceded by trachoma of the conjunctiva, which, however, should not be regarded as the cause of the affection, inasmuch as the latter can develop in eyes that previously were healthy. The actual cause of the disease is not known. In every instance it is a purely local process, for the individuals attacked by it are sound as far as the rest of the body is concerned, and do not suffer from amyloid degeneration of the internal organs, with which, therefore, amyloid degeneration of the conjunctiva has nothing at all to do.

Medical treatment is powerless against this disease. We must confine ourselves to removing the growths upon the conjunctiva to such an extent that the lids can be opened

and vision thus rendered possible. It is by no means necessary—indeed it is not at all advisable—to remove by a radical operation all the diseased parts, since the portion of the growth that is left behind generally atrophies of itself afterward.

Conjunctivitis Petrificans.—Leber describes under this name a disease in which white spots appear in the conjunctiva which are produced by the deposition of lime and from which ulcers develop whose floor is rendered white and hard by lime deposits. This disease drags on for years with frequent remissions, and produces adhesion of the lids to the eyeball.

VIII. Tuberculosis of the Conjunctiva

169. In the conjunctiva tuberculosis ordinarily appears under the guise of ulcers. Tuberculous ulcers are located as a rule in the tarsal conjunctiva. The diseased lid even on external inspection looks thickened. On everting the lid there appears upon its conjunctival surface an ulcer which is either covered by grayish-red granulations (Fig. 59), or has a yellowish-red, lardaceous-looking base. In its vicinity small gray nodules (tubercle nodules) or outgrowths resembling a cock's comb are often found in the conjunctiva. The ulcer shows no disposition to heal; on the contrary, it spreads, although it does so very slowly. It may pass over to the conjunctiva of the eyeball; and even the cornea is sometimes covered over by a sort of pannus. In especially severe cases the ulcer does not remain confined to the conjunctiva, but eats through the entire thickness of the lid, so that even on external examination a deficiency of tissue is observable in the lid. Quite early in the disease the lymphatic gland in front of the ear becomes swollen; afterward the lymph glands about the lower jaw and in the neck also become enlarged. The clinical picture afforded by the disease is sufficiently characteristic to make the diagnosis from it alone; but to be quite sure upon this point, we should remove bits of tissue from the ulcer and either demonstrate the presence of tubercle bacilli in them by the ordinary methods, or else, by inoculating them into rabbits' eyes, set up tuberculosis in the latter.

Fig. 59.—Tuberculosis of the Conjunctiva.

Taken from a girl eighteen years of age who looked healthy but had a clearly demonstrable affection of the apices of both lungs. The conjunctiva of the lower lid is to a large extent occupied by a grayish-red outgrowth demarcated from the rest of the conjunctiva by a border which rises abruptly and in places is actually overhanging. On its surface the outgrowth bears numerous grayish-coated ulcerated areas. On the conjunctiva of the upper lid there is merely a little papillary hypertrophy. The gland in front of the ear appears on palpation to be the size of a pea.

Tuberculosis of the conjunctiva generally attacks only one eye. The patient does not suffer pain; it is only by the swelling of the lid, the purulent secretion, and subsequently by the diminution of visual power, that he is annoyed and made aware of the existence of his trouble. The disease occurs, almost without exception, in young people, and runs an uncommonly chronic course, being often protracted over many years. Even after

an apparently radical cure it shows a great tendency to recur, and it can, by infecting the rest of the organism, finally lead to death from tuberculosis. Treatment consists in radically excising or curetting the ulcer and then cauterizing the raw surface; using here the galvano-cautery loop or lactic acid (pure or 50 per cent), and in the latter case taking care that none gets on the cornea. If the tuberculous foci are so extensive that complete removal would be followed by great deformity of the lids or marked symblepharon, we may try to dissipate the tuberculous tissue by tuberculin injections or phototherapy. For after treatment it is best to apply iodoform for a long time to the conjunctival sac in powder or 10- to 20-per-cent ointment—iodoform being specially efficacious in tuberculous processes.

Tuberculosis and *lupus* of the conjunctiva are to be regarded as essentially identical affections, in that both represent ulcerative processes produced and maintained by the presence of tubercle bacilli. In fact, the first cases of tuberculous conjunctival ulcers were described as primary lupus of the conjunctiva (i.e., lupus without coincident lupus of the skin) (Arlt). The two processes are distinguished only by external differences relating to their aspect and course. Thus, as a rule, lupous are distinguished from tuberculous ulcers of the conjunctiva by the fact that they have migrated from skin to conjunctiva, and that, like lupus of the skin, they show spontaneous cicatrization on one side while on the other the ulcer keeps advancing (see also pages 181 and 203).

Tuberculosis of the conjunctiva may originate either in ectogenous or in endogenous infection. In individual cases, to be sure, one often cannot determine which sort of infection is present. We should, e.g., think the infection *ectogenous* if a quite circumscribed tuberculous ulcer were situated on the conjunctiva of a man in whom clinically demonstrable evidences of tuberculosis were not present elsewhere. For example, a particle of dust carrying bacilli might get into the conjunctival sac and by its sharp angles produce a small superficial lesion of the conjunctiva, which is thus infected (tubercle bacilli, according to the researches of Valude, not penetrating into the conjunctiva when the epithelium is intact). In favor of an infection of this sort is the fact that we so often see tuberculous ulcers begin in the region of the sulcus subtarsalis, where small foreign bodies are so readily retained. In such cases, the conjunctival tuberculosis may represent the only focus of the disease in the body—*primary* tuberculosis of the conjunctiva. This may remain for a long time confined to the conjunctiva; indeed, in exceptional cases, it may even heal spontaneously. The rule, however, is for tuberculosis to spread from here to other parts of the organism. This extension may be by way of the lymphatic circulation, the neighboring lymph glands being first attacked by the tuberculosis. Or the disease may extend by continuity, the tear passages first and then the nasal mucous membrane being infected by the tears containing the bacilli. Those cases of conjunctival tuberculosis are regarded as *secondary* in which there is at the same time tuberculosis of the internal organs (especially the lungs). The eye may then be infected by the patient's getting some of his own tuberculous sputum into it. Or the tuberculosis extends by continuity, a tuberculous or lupous affection of the nasal mucous membrane passing to the conjunctiva through the tear passages. (Not infrequently, therefore, we find conjunctiva, lachrymal sac, and nasal mucous membrane attacked at the same time by tuberculosis, and careful study of the history of the case and exact examination generally render it possible to ascertain whether the affection has passed from conjunctiva to nose or vice versa.)

An *endogenous* (hæmatogenous) infection is present when the tubercle bacilli have been carried into the conjunctiva through the circulation. This sort of infection may

be assumed to exist especially in those cases in which the disease occurs under the form of discrete miliary foci in the conjunctiva (see page 181).

170. Parinaud's Conjunctivitis.—In this disease, which develops acutely with fever and other evidences of constitutional disturbance, reddish or yellowish granulations form in the highly inflamed conjunctiva, and these not only in the retrotarsal folds but also in the conjunctiva of the lids and even in the conjunctiva of the eyeball. The granulations sometimes grow so as to form quite large swellings; in many cases also there are very small superficial gray-coated ulcers in the conjunctiva. A characteristic sign of the disease is the swelling which affects the pre-auricular lymph gland and indeed the whole parotid region, sometimes even down to the neck. The swollen glands not infrequently suppurate. The disease ends in recovery within from a few weeks to a few months without producing any further bad results. Usually but one eye is affected. Parinaud ascribed the disease to infection derived from animals, [but this cannot always be traced, nor is tuberculosis, which is present in some cases, uniformly present. Verhoeff in a number of cases found a leptothrix forming filamentous masses walled off by an area of cell necrosis.—D.].

Sporotrichosis Conjunctivæ.—This rare disease is caused by various species of sporotrichon and produces small, light-yellow, soft nodules in the conjunctiva with [points of ulceration and with purulent discharge. There is] swelling of the neighboring lymph glands. Nodules also occur in the lid-margin. [The canaliculi may contain concretions enclosing the sporotrichon. The disease resembles Parinaud's conjunctivitis, from which it can be differentiated by the discovery of the sporotrichon (Wilder, Bedell). —D.] A cure usually results from the internal use of potassium iodide.

171. Ulcers of the Conjunctiva.—Ulcers of the conjunctiva, besides occurring as a result of tuberculosis, are also observed in the following conjunctival affections:

(*a*) As one of the symptoms of a conjunctivitis, an example being the minute ulcers originating in the efflorescences of conjunctivitis eczematosa or those which have given its name to the pustular form of catarrh (see page 141).

(*b*) [In squirrel-plague conjunctivitis (see page 141).]

(*c*) In sporotrichosis of the conjunctiva, and Parinaud's conjunctivitis (see § 170).

(*d*) After the separation of necrotic portions of the conjunctiva, as in diphtheria, or after burning of the conjunctiva with heat or caustics. Here belong also the eschars produced artificially by the use of too strong applications.

(*e*) As a result of exanthemata; ulcers, for instance, which are derived from a variolous pustule or from the rupture of a bulla of pemphigus upon the conjunctiva.

(*f*) Upon the tarsal conjunctiva there is quite often found a small raw spot, from which rises a little mass of granulations. Here we have to do with a chalazion which has broken through on the inner side of the lid. As a rule, a slender sound can be introduced through the granulations into the cavity of the chalazion.

(*g*) Vaccine ulcers due to transfer of the vaccine poison from a vaccine pustule (Purtscher). They are heavily coated ulcers and are attended with considerable swelling of the pre-auricular lymph gland (cf. § 219, Keratitis Disciformis and § 591, Vaccine Ulcers of the Margin of the Lids).

(*h*) Ulcers in glanders.

(*i*) Ulcers which have developed from the breaking down of an epithelioma of the conjunctiva.

(*j*) Syphilitic ulcers. Generally we have here to do with those losses of substance which have arisen from the breaking down of an initial sclerosis. These, as a rule, are situated near the free border of the lids, but are also observed in the retrotarsal fold and even in the conjunctiva of the eyeball. The transmission of syphilis appears to take place most frequently by kissing, and in small children also by the practice which many

nurses have of moistening the agglutinated edges of the lids with saliva in order to open them. In some countries it is the custom to remove foreign bodies from the conjunctival sac by licking them out with the tongue. Occasionally, also, syphilitic ulcers have been observed, which were produced by the breaking down of gummata of the conjunctiva (Hirschberg). Syphilitic ulcers of the conjunctiva are among the greatest of rarities. Still rarer is soft chancre of the conjunctiva.

IX. Injuries of the Conjunctiva

172. The following varieties of injuries of the conjunctiva, which are of such frequent occurrence, are observed:

(*a*) *Foreign bodies* in the conjunctival sac. Small-sized foreign bodies, like grains of dust, particles of coal or of ashes, which so often get into the eye during a railroad journey, the wing cases of small beetles, etc., fall first upon the surface of the eyeball, are brushed away from this spot by the movement of the upper lid, and then generally stick to the inner surface of the latter at a spot not far from its free border, where a shallow furrow, the sulcus subtarsalis, that runs parallel to the edge of the lid, catches the foreign body. The pain which such a foreign body causes, and which is often quite considerable, does not originate in the conjunctiva itself, which has very little sensitiveness, but in the cornea, inasmuch as with every movement of the lid the foreign body is carried over the cornea and scrapes it. Hence the pain is absent as long as the eye is kept quietly closed. It is easy to remove the foreign body after the lid is everted.

In other cases, small, sharp-pointed foreign bodies penetrate into the conjunctiva, and may remain there a long time. Grains of powder remain fixed in the conjunctiva of the eyeball without giving rise to any further irritation, and may therefore be left in the conjunctiva. Larger-sized foreign bodies are retained in the conjunctival sac only when they get into the upper retrotarsal fold. In this spot they stay, remaining still even during the act of winking, cause no irritation of the cornea, and therefore produce but little trouble. After some time has elapsed they begin to excite the symptoms of chronic conjunctival catarrh.

(*b*) *Solutions of continuity* of the conjunctiva are not rare, and are often associated with extensive infiltration of blood (ecchymosis). If the edges of the wound are not too greatly lacerated, the conjunctival wound can be closed with a stitch.

(*c*) *Burns* of the conjunctiva and *corrosive injuries* are pretty frequent. Burns are the result of hot water or steam, hot ashes (especially cigar ashes), exploding powder, flames striking against the eye, molten metals, etc. Of corrosive injuries, which may be produced both by acids and by alkalies, those that arise from the action of lime are the most frequent, the lime getting into the eye usually under the form of mortar. As a rule the injury is greater in the lower part of the conjunctival sac than in the upper, because the eyeball turns up the moment the injury is inflicted, so that its lower

part is exposed in the palpebral fissure, and also because the hot or corrosive liquid flows down at once into the lower part of the conjunctival sac.

The action of burns is the same as that of caustics; the conjunctiva at the affected spots is destroyed and converted into an eschar. These spots stand out as gray or white patches in the midst of the reddened and swollen portions of the conjunctiva that are not escharotic. The eschars separate in consequence of a delimiting suppuration, and the resultant granulating losses of substance heal by a drawing in over them of the neighboring healthy conjunctiva. The final result is therefore always the formation of a cicatrix. This may lead to a diminution in size of the conjunctival sac, or, if of great extent, to adhesion of the lids to the eyeball (symblepharon).

[Corrosive injuries, especially those affecting the limbus, quite frequently cause a rise of tension, which develops several days after the injury, and last a week or more (Kuemell).—D.]

The *prognosis* of an injury by burns or caustics with regard to the preservation of sight depends primarily upon the condition of the cornea, which, indeed, is always implicated in any extensive lesion of the conjunctiva. Next, the losses of substance in the conjunctiva itself must be considered, since the adhesions that develop from them may more or less harm the function of the eye. [The amount of injury done often cannot be determined in the first two or three days, so that the prognosis must be guarded.—D.]

The prime *therapeutic* requisite, when we get an eye under treatment soon after injury by caustics, is the complete removal of any corrosive substance still present. We remove solid particles with a linen pledget or a forceps, and then wash the conjunctival sac out thoroughly with a gentle stream of water. In the further course of an injury by burns or caustics, we must restrain subsequent inflammation by cold compresses, atropine, a bandage, etc. [If the tension rises we may have to use miotics.] After separation of the eschars, our aim must be to confine the resulting adhesions within the smallest limits. For this purpose we repeatedly draw the lids away from the eyeball to prevent adhesion of the two opposed raw surfaces. If the defect extends so far as to implicate the retrotarsal fold, we try by transplanting a flap of skin or mucous membrane to prevent the formation of an adhesion between the lid and eyeball, starting from the fornix (symblepharon posterius). If, nevertheless, this does develop, it must be removed, as far as can be, by operation later.

Sometimes foreign bodies are introduced into the eye purposely. Chief among these are what are called crab's eyes—*lapides cancrorum*. These are flat, calcareous concretions derived from the stomach of the crab, which are in great favor among the laity for removing foreign bodies from the eye. The crab's eye is introduced between lid and eyeball, and then pushed across the cornea, carrying with it, it may be, mechanically any foreign body there present.[17] Sometimes it happens in performing this manipula-

[17] [Eyestones (the flat opercula of certain molluscs) are also used for the same purpose.—D.]

tion that the crab's eye slips into the superior fornix and remains there unnoticed. We may then find it there months or even years afterwards, entirely imbedded in the outgrowths of the conjunctiva, which has undergone chronic inflammation. Foreign bodies, like sand, ashes, scrapings from a wall, etc., are also intentionally introduced, in order to simulate eye disease, into the eye, where they set up a conjunctivitis.

[That the effect of burns, e.g., from particles of molten metal flying into the eyes, is often quite superficial is due partly to the cooling action of the tears, partly to the fact that the vaporized moisture forms a layer between the hot body and the eye (Eales).—D.]

By the entrance of caterpillar hairs (see § 395, Ophthalmia Nodosa) or of plant hairs into the conjunctival sac, acute inflammations of the conjunctiva develop often with the formation of small nodules in the conjunctiva inclosing the hairs.

After the action of irritants on the eye an acute *traumatic conjunctivitis* is produced, evidenced by intense reddening of the conjunctiva with great photophobia, lachrymation, and pain, with which is associated in violent cases œdematous swelling of the lids. Such irritants are acrid vapors, liquids, or dust-like particles which get into the eye either by accident or as part of the day's work in certain industries. [Examples of this *occupational* conjunctivitis are seen in cigar-makers, men whose work exposes them to metal, stone or emery dust, workers on certain kinds of wood, those engaged in preparing aniline dyes and other chemicals, workers in pitch or those employed on tarred roads, hop-pickers, oyster-shuckers, etc. A very violent, but transient conjunctivitis is produced by eel's blood spurting into the eyes. Some of the irritants mentioned, e.g., the aniline dyes, also cause discoloration of the conjunctiva. Discoloration without marked irritation occurs from the impact of silver dust in silver polishers (argyrosis).—D.] For the physician it is important to know that chrysarobin, which used as a remedy (especially for psoriasis), may cause acute conjunctivitis, and must then be discontinued. Under the same guise of an acute traumatic conjunctivitis occurs the conjunctival inflammation which follows the action of intense light, e. g., after dazzling by the reflection from snow (*snow blindness*), or by the electric arc light (*ophthalmia electrica*). (See page 24.)

X. Pterygium

173. Symptoms and Course.—A pterygium is a triangular fold of mucous membrane which extends from the conjunctiva of the eyeball to the cornea, either at the inner or the outer side of the latter (Fig. 60). The blunt apex of the triangle lies in the transparent portion of the cornea, and is solidly and immovably united to it. The base of the triangle spreads out in the conjunctiva of the eyeball, and passes into it without there being any sharp line of division between the two. The apex is called the head, the base the body, of the pterygium. The part that lies between the two and corresponds to the margin of the cornea is the neck of the pterygium. Here the limits of the fold of conjunctiva are the most sharply defined, its borders being inverted in such a way that a small sound (*S*, Fig. 60) can be pushed beneath them for a short distance. A pterygium of recent origin is succulent and abounds in vessels which run converging from the base to the apex and impart to the pterygium its red color. In fact, from its similarity in form and vascularity (venation) to the wings of many insects (Hymenoptera), the name of pterygium is derived.[18] The fold of conjunctiva forming the

[18] From [πτερύγιον, the diminutive of] πτέρυξ, a wing.

pterygium is tightly stretched, so that there are produced a number of radially disposed furrows or flutings; furthermore, in pterygia which are situated at the inner side of the eye the plica semilunaris is often quite obliterated and is included in the body of the pterygium (Fig. 60).

. In the course pursued by a pterygium two stages must be distinguished. In the first the pterygium keeps on gradually growing, for years it may be, toward the center of the cornea, which it may finally reach or even pass (*progressive pterygium*). Finally, the pterygium comes to a standstill so as to remain permanently attached to the same point upon the cornea (*stationary pterygium*). Whether in any given case we are dealing with a progressive or a stationary pterygium is determined mainly by the character of the apex of the growth. In the progressive stage this appears surrounded by a gray non-vascular zone which looks thick and gelatinous. In a stationary pterygium this marginal zone is found to be thin and cicatricial looking and the whole pterygium is thin, pale, nearly destitute of vessels, and tendinous.

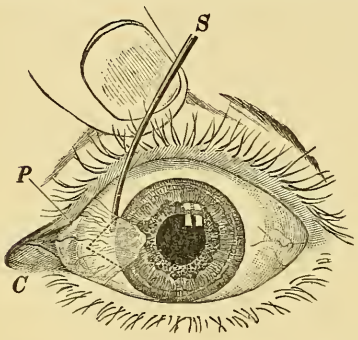

Fig. 60.—Pterygium.

A sound, *S*, is carried beneath the edge of the pterygium, *P*. The dotted line shows the way in which the section is made in removing the pterygium. *C*, caruncle. The adjacent plica semilunaris has been flattened out by the tensive force of the pterygium, and is hence invisible. *P*, upper punctum lacrimale.

Pterygium occurs only in that part of the cornea corresponding to the interpalpebral fissure. It is found most frequently on the inner side of the cornea; when there is already one in this spot another may form on the outer side also. The two pterygia may even meet in the center of the cornea. A genuine pterygium is practically never observed at the upper or the lower margin of the cornea; on the other hand, it is not infrequently the case that both eyes are attacked simultaneously by pterygium, so that we sometimes see patients who have four pterygia, one on the outer and one on the inner side of either cornea.

Among the injurious *results* which a pterygium entails, the worst is the damage done to the eyesight. This begins to be produced as soon as the point of the pterygium projects into the pupillary area of the cornea, and it increases in proportion as the point approaches the center of the latter. Moreover, pterygium by the tension to which it gives rise often causes a condition of irritation in the eye, as is shown by the marked injection and succulence (catarrhal inflammation) of the pterygium itself. In addition, the membrane, particularly if very much reddened, forms a striking disfigurement and can also cause a restriction of the mobility of the eye. If, for example, a pterygium is situated on the inner side of the cornea and the eye is meant to be turned strongly outward, the eye may be restrained in its

movement by the tension of the pterygium, and therefore fail to move laterally as far as the other or healthy eye, so that binocular diplopia may be produced in consequence of this faulty placing.

The older writers distinguished a pterygium crassum (vasculosum, carnosum, sarcomatosum) and a pterygium tenue (membranaceum). The former corresponds to a catarrhally inflamed, and hence therefore red and thick pterygium, the latter to a stationary pterygium, which has become thin and tendinous.

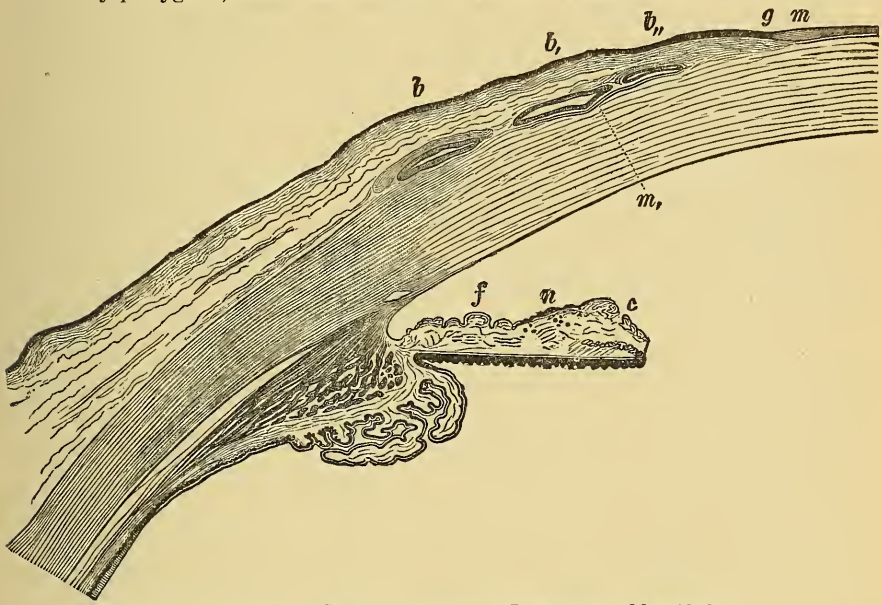

Fig. 61.—Longitudinal Section through a Pterygium. Magnified 12 × 1.

The apex of the pterygium is marked by the termination of Bowman's membrane at *m*. At *g* is the boundary between the epithelium of the conjunctiva and of the cornea. At *b*, b_1, b_{11}, lie cavities lined with epithelium. These are the cross sections of spaces which jut in from the lateral margin of the pterygium, between the latter and the surface of the cornea, and which are divided by the section close to their blind extremity. Beneath them at m_1 are to be seen remains of Bowman's membrane. The eye had a wide pupil, consequently the iris in the section looks short and thick; its pupillary portion rises abruptly from the anterior capsule of the lens and indeed actually is overhanging so as to conceal the retinal pigment layer of the iris (compare *p*, Fig. 142). Conspicuously visible on the anterior surface of the iris are the very protuberant contraction folds, *f*, also the orifice of a crypt, *c*, and lastly an accumulation of pigmented cells, *n*, in the superficial layers which appears as a brown spot in the otherwise blue iris (nævus iridis).

174. Etiology.—A pterygium is nothing but a fold of conjunctiva drawn up over the cornea and fastened to it. It originates from the pinguecula, the degenerative process which exists there making its way into the limbus, and then gradually upon the cornea itself. The pinguecula, therefore, grows up, as it were, over the cornea, and in so doing draws the conjunctiva after it. Since the pterygium originates in the pinguecula, it is evident why, like the latter, it should occur only at the inner and outer margins of the cornea. And as the pinguecula develops in consequence of the injurious influences which the conjunctiva in the region of the palpebral fissure is exposed to in the course of years, the like is true of pterygium and in a still greater degree. Hence it is found particularly [but by no means

exclusively] in elderly people and especially in those who are much exposed to dry heat, bright sunlight,] wind or dust, as in the case of farmers, coachmen, masons, stonecutters, [cooks, seamen,] etc., while among the well-to-do classes pterygium belongs among the rarities. The fact that the conjunctiva is stretched tightly over the cornea accounts for the triangular form of the fold, its incurved margin, and its great tension in a horizontal direction.

A pterygium consists mainly of fibrillar connective tissue, which is covered with the epithelium of the conjunctiva. The apex of the pterygium, however, is often covered by the corneal epithelium, so that the pterygium may be said to force its way underneath the latter (Fig. 61, *g*). In the tissue of the pterygium are found new-formed tubular glands and also larger spaces lined with epithelium (*b*, b_1, b_{11},); from both of these small cysts may develop. Bowman's membrane, where it lies beneath the pterygium, is destroyed; indeed, the uppermost lamellæ of the cornea itself are replaced here and there by the tissue of the pterygium. This explains why, even after the ablation of the pterygium, the cornea does not regain its normal transparency.

175. Treatment.—This consists in an operation (see § 820); which is indicated in every case of progressive pterygium. Even though the pterygium is still small, we cannot be sure that it will not grow into the pupillary area of the cornea. Hence we prefer by performing operation to guard in time against injury to the eyesight. If the pterygium has approached sufficiently near to the center of the cornea to cause disturbance of vision, this disturbance is indeed rendered less by the operation of ablation, but is not completely done away with, since those spots of the cornea, which were occupied by the pterygium, never again become perfectly transparent. In any case, the associated symptoms of irritation, the restriction of mobility, and the disfigurement are removed by the ablation. A stationary pterygium does not necessarily require operation; in this case we shall be guided mainly by the wishes of the patient with reference to the removal of the disfigurement, etc.

176. Pseudo-Pterygium (Cicatricial Pterygium).—Sometimes we observe as a result of inflammatory processes fixation of a fold of conjunctiva upon the cornea, giving a picture similar to that of true pterygium. For example, there may be a gonorrhœal conjunctivitis, with great chemosis and a pretty large marginal ulcer of the cornea. The chemotic protuberance of the conjunctiva becomes applied to the surface of the ulcer and adheres to it. After the inflammation has abated, the swelling of the conjunctiva goes down, and the chemotic protuberance disappears; but, at the spot where union with the cornea has taken place, the conjunctiva remains permanently fixed to the latter. We then see a triangular fold formed of conjunctiva extending over the limbus and upon the cornea, and attaching itself there. Ordinarily, at that part of the fold corresponding to the limbus a small sound can be passed all the way beneath the fold, a sign that the latter is adherent to its bed at its apex only and not throughout its whole extent. This is the most important distinguishing mark between a true and a false pterygium; another consists in the fact that a pseudo-pterygium does not keep on growing over the cornea, as a true pterygium does, but remains fixed forever to the spot at which it first became adherent. In its origin and its behavior a pseudo-pterygium is more like a symblepharon than a genuine pterygium.

Pseudo-pterygia are observed not only after gonorrhœal conjunctivitis, but also after diphtheria, burning by heat and by caustics, prolapses of the iris, the removal of

new growths, etc. It is evident that they may develop not only at the outer and inner sides, but at any side whatever of the cornea. The pseudo-pterygia remaining after gonorrhœal conjunctivitis are usually found above, those produced by burns, etc., most frequently at the lower part of the cornea (in the area corresponding to the palpebral fissure).

Another kind of pseudo-pterygium is that which develops after a chronic superficial ulceration of the marginal portions of the cornea (keratitis marginalis superficialis, Fig. 95). Owing to the cicatrization which follows the ulcerative process the conjunctiva is drawn up steadily over the cornea. These pseudo-pterygia are very similar to true pterygia, for like the latter they keep on growing slowly over the cornea, and unlike other pseudo-pterygia are not completely perforate at the limbus. The differential diagnosis between these and the true pterygia can be made only in case we find the ulcerative process or its sequelæ (a superficial corneal opacity) upon the marginal portions of the cornea not implicated in the pterygium.

Small pseudo-pterygia may without disadvantage be left undisturbed; larger ones we generally remove in the same way as genuine pterygia, and unite with stitches the wounds left in the conjunctiva. In those cases in which the pseudo-pterygium is not adherent to the surface of the eyeball at a spot corresponding to the limbus, ablation and the use of the suture may be dispensed with; it is sufficient simply to free the point of pseudo-pterygium from the cornea, upon doing which the former retracts of itself and disappears by a process of atrophy.

It sometimes happens that an old pannus which has already been transformed into connective tissue is united to the subjacent cornea only by loose cellular tissue, and thus acquires a certain freedom of movement, so that it can be shifted this way and that upon its bed along with the conjunctiva of the eyeball. So in this way, too, a picture similar to that of a pterygium may be produced.

XI. Symblepharon

177. Characteristics.—By symblepharon[19] we understand a cicatricial adhesion between the conjunctiva of the lids and the conjunctiva of the eyeball. In that case, when we attempt to draw the lid away from the eyeball

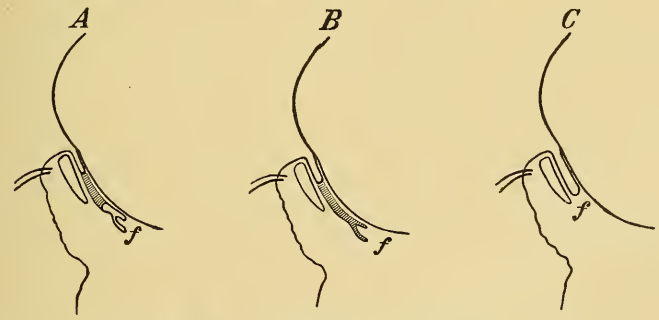

Fig. 62.—Symblepharon (Schematic).
A, symblepharon anterius; *B*, symblepharon posterius by adhesion; *C*, symblepharon posterius by cicatricial shrinking; *f*, fornix.

we observe that in one or more places bands extend from the inner surface of the lid to the surface of the eyeball, and that these become tightly stretched and prevent us from drawing the lid away completely. These

[19] From σύν, together, and βλέφαρον, eyelid.

bands generally look tendinous, more rarely fleshy, and may be attached not only to the conjunctiva scleræ, but also to the surface of the cornea itself. If the adhesion between the two conjunctival surfaces extends so far peripherally as to reach into the fornix, we call this *symblepharon posterius* (Fig. 62 B). If the adhesion does not extend as far as this, so that the cicatricial bands stretch like a bridge between the lid and the eyeball, and we can introduce a sound beneath them and carry it along the fornix between the lid and the globe, it is then called *symblepharon anterius* (Fig. 62 A). This distinction has been made for practical reasons, inasmuch as symblepharon anterius can be readily cured by an operation, while symblepharon posterius can be cured with difficulty or not at all. *Symblepharon totale* is a total adhesion between the lids and the eyeball—a condition which occurs but rarely.

178. Etiology.—Symblepharon develops whenever two opposed spots of the conjunctiva of the lid and of the eyeball have raw surfaces which come into contact with each other, and in consequence become adherent. Such a process of adhesion results of necessity when the two raw surfaces extend up into the fornix and there become continuous, since two raw surfaces which meet at an acute angle always begin to unite at this angle of junction. Causes which may give rise to the formation of raw surfaces upon the conjunctiva are burns by the action of the heat, burns from caustic substances, diphtheria, operations, ulcers of all kinds, etc.

The expression symblepharon is also employed in a somewhat different sense to denote the contraction of the conjunctiva which occurs as a result of its gradual *shrinking*. In this case it is not a question of an adhesion between two raw conjunctival surfaces, but of a gradual diminution in size of the conjunctival sac. The folds of the retrotarsal region are first smoothed out; the conjunctiva of the lid passes to the eyeball directly (Fig. 62 C) and whenever the lid is drawn away is pulled out into tense perpendicular folds. In advanced cases the conjunctival sac is converted into quite a shallow groove between the eyeball and the lid. Since contraction of the conjunctival surface due to shrinking always makes itself first apparent by disappearance of the retrotarsal fold, all these cases belong to symblepharon posterius. This kind of symblepharon is observed principally after trachoma; furthermore, in the rare cases of pemphigus of the conjunctiva.

179. Symptoms and Treatment.—Very slight cases of symblepharon produced no bad *results* worth mentioning. When there are more pronounced adhesions, the excursions of the eye are hindered, and thus possibly diplopia may be produced just as in the case of pterygium. Inasmuch as with the movements of the eye traction is made at the site of the adhesions, the eye gets into an irritated condition. If the adhesions extend into the region of the palpebral fissure they become disfiguring, and if they extend

as far as the cornea they may injuriously affect the sight. Sometimes the lids are so fastened down by extensive adhesions that their complete closure is impossible, and lagophthalmus is produced with its consequences that are so baneful to the cornea. Total symblepharon is obviously associated with complete blindness (or at most with quantitative perception of light).

Treatment is by operation (see §§ 821 and 822).

For *epitarsus*, see page 162.

XII. Xerosis

180. Symptoms.—Under the name of xerosis[20] conjunctivæ we designate an alteration of the conjunctiva, generally occurring in patches and consisting in a dryness of the membrane. The surface of the conjunctiva at the xerotic spots glistens like fat and is of a whitish color, and looks either like epidermis or as if it were covered with dried foam. If the change affects the scleral conjunctiva the latter is thicker, less pliable, and lies in stiff folds. The tears flow over the diseased spots without moistening them. An analogous change is also observed in the cornea, the surface of which looks dull, lusterless, and dry, while the parenchyma of the cornea at the same time is found to have lost its transparency (xerosis corneæ).

Etiology.—Xerosis occurs in two groups of cases. *In one it results from a local affection of the eye.* This occurs—

(*a*) In *cicatricial degeneration* of the conjunctiva. It is observed most frequently as the final outcome of trachoma, and also, but more rarely, after diphtheria, pemphigus, burns, etc. It begins in isolated spots, but may ultimately spread over the entire conjunctiva, and over the cornea also. In the latter case, since the xerotic cornea grows opaque, the eye becomes blind. This form of xerosis is incurable.

(*b*) *Deficient sheltering* of the conjunctiva, so that the latter is constantly in contact with the air, may likewise lead to xerosis. This occurs in ectropion and in lagophthalmus (incomplete closure of the lids). In the former case the exposed portion of the tarsal conjunctiva, in the second case that part of the scleral conjunctiva and of the cornea lying in the course of the papebral fissure, becomes covered with a thickened, dry, epidermoid epithelium, by means of which these parts protect, so to speak, their deeper layers against desiccation. In such cases assistance can be rendered only when it is possible (by operative interference) to provide the exposed conjunctiva or cornea with its normal shelter again.

In the second group of cases xerosis appears as a symptom of a *general disease* of a peculiar character, the real nature of which is as yet unknown. It occurs pre-eminently in persons who are depressed in nutrition. The xerosis in these cases begins at the outer and inner parts of the conjunctiva

[20] From ξηρός, dry.

of the eyeball under the form of small triangular areas which are covered by what looks like a fine dried foam and are not wet by the tears (Bitot). As a rule there is present at the same time a peculiar disturbance of sight, namely *hemeralopia* or night blindness (see § 569).

If it is adults that are affected, the xerosis and hemeralopia pass off within a few weeks. In small children, on the other hand, the disease often takes a malignant course. The xerosis spreads from the area occupied by the palpebral fissure over the whole conjunctiva of the eyeball and then over the cornea too. The latter becomes cloudy and suppurates under the guise of *keratomalacia* (see § 221), and the little patients die with the symptoms of a severe general disease. For the treatment see §§ 151, 220, 222.

FIG. 63.—XEROSIS CONJUNCTIVÆ. Magnified 820 ×1.

Epithelial cell scraped from the xerotic conjunctiva and stained by Gram's method. The nucleus of the cell is marked by the fact that numerous granules of keratohyalin lie immediately about it, although still in the protoplasm of the cell. Upon the cell are seen xerosis bacilli.

The separation, first made by Cohn, of xerosis into a variety dependent upon local causes and one caused by a general disease corresponds nearly to the ordinary division into xerosis parenchymatosa and xerosis epithelialis. In local xerosis the mucous membrane is diseased in its deeper as well as in the superficial layers (xerosis parenchymatosa); while in xerosis produced by a general disturbance of nutrition the change affects the epithelium only (xerosis epithelialis). Many also make a distinction between xerosis partialis (sive glabra) and xerosis totalis (sive squamosa).

The anatomical changes which underlie xerosis mainly affect the epithelium. The uppermost layers of epithelial cells undergo keratosis, the protoplasm of the succeeding layers containing abundant granules of keratohyalin (Fig. 63). In consequence the epithelium appears thickened, whitish, cloudy, and epidermoid. Moreover, it is covered with the sebum-like secretion of the Meibomian glands, and hence assumes a fatty character, in consequence of which the lachrymal fluid does not adhere to it. To this circumstance the peculiar dry appearance is chiefly owing, for if the diseased spots are freed from their fat by pencilling with soap they become capable of being wetted by the tears (Leber).

Reymond and Colomiatti and shortly afterward Kuschbert and Neisser described as occurring in xerosis a special micro-organism, the bacillus of xerosis, which is extremely like the diphtheria bacillus. This is found under the form of short rods adhering in great quantities to the surface of the epithelial cells (Fig. 63). It is, however, neither the cause of xerosis, nor indeed characteristic of the latter. It occurs frequently in the conjunctival sac anyhow, and simply seems to find in the dying epithelium of the xerotic spots a specially favorable soil for growth, so that it develops there in great quantities.

What part does the *lachrymal secretion* play in xerosis? The real cause of the dryness of the conjunctiva is not, as has been believed, the deficiency of tears. In the beginning of the disease, as long as only small isolated spots of the conjunctiva are xerotic, we often find actually an increase in the lachrymal secretion. Per contra, xerosis of the conjunctiva has never been known to occur after extirpation of the lachrymal gland. The real cause of the dryness of the conjunctiva is rather the fact that the tears do not adhere to it. Nevertheless it is true that in advanced xerosis the lachrymal

secretion diminishes, and may even disappear altogether, for, owing to the marked shrinking of the conjunctiva, the secretory ducts of the lachrymal gland, which empty along the upper retrotarsal fold, become closed, and as a result of this, atrophy of the gland itself ensues. The patients, when led to weep by their melancholy state, feel only a sensation of fulness in the eyes; they cannot shed tears. Arlt found in a case of xerosis obliteration of the efferent ducts of the lachrymal gland produced by the great shrinking of the conjunctiva, the lachrymal gland itself being diminished to one third of its normal volume and transformed into a tissue resembling fat. In that form of xerosis also which occurs in connection with keratomalacia there is a striking deficiency in the lachrymal secretion; but here probably we have to do with a nervous disturbance, namely, with an absence of the reflex lachrymal secretion, due to depression of the general nutrition and particularly of the nervous functions. In a case of this sort Cirincione has found on autopsy an inflammation of the ciliary ganglion and ganglion Gasseri.

XIII. Extravasation of Serum and Blood beneath the Conjunctiva

181. Œdema and also extravasation of blood, if of any great extent, are observed only in the conjunctiva of the eyeball and in the retrotarsal fold, as these, on account of the laxity with which they are fixed to the subjacent parts, can be readily lifted up over quite large areas by fluid. The same thing does not occur in the conjunctiva tarsi, because it is too intimately adherent to the underlying cartilage.

In the conjunctiva of the eyeball we meet with both inflammatory œdema (œdema calidum), occurring in inflammations of the eyeball or the organs near it, and with non-inflammatory œdema (œdema frigidum), resulting from simple transudation of fluid. Since œdema, as a rule, is of importance merely as a symptom of some other affection, treatment will have to be directed to the latter. Should it be desirable to take any special measures against the œdema itself, the most suitable means for this purpose would be a pressure bandage, or, in the case of a tense œdema, scarification of the conjunctiva.

Inflammatory œdema is associated with the most various inflammatory affections, such as inflammations of the lids (erysipelas, hordeolum), of the margin of the orbit (periostitis), of the lachrymal sac (dacryocystitis), of the conjunctiva (particularly gonorrhœal conjunctivitis), of the eyeball itself (purulent keratitis, irido-cyclitis, and panophthalmitis), and, furthermore, of structures posterior to the eyeball (tenonitis, phlegmon of the orbit, cerebrospinal meningitis). Inflammatory œdema is especially apt to occur in old people, whose conjunctiva is particularly extensible and so loosely attached that we observe œdema in this situation sometimes with trifling conjunctival catarrhs. We commonly find the œdema most pronounced in the region of the palpebral fissure, because here the counter-pressure exerted by the lids is wanting; and not infrequently an œdematous fold of conjunctiva is found in the palpebral fissure, pinched off, so to speak, from the rest by the lids.

An inflammatory œdema of the conjunctiva is produced artificially for therapeutic purposes by *dionin* (see page 64).

Non-inflammatory œdema is caused by hydræmia or by stasis. In the former case it occurs as a symptom of albuminuria, and sometimes then recurs a number of times,

but each time disappears again rapidly (œdema fugax). A peculiar sort of œdema is the *filtration œdema* of the conjunctiva of the eyeball. It is observed after operations or injuries which have produced a perforation in the most anterior section of the sclera, and arises from the fact that the aqueous from the anterior chamber oozes between the incompletely closed edges of the wound and lodges under the conjunctiva. Accordingly, when cicatrization occurs, the œdema commonly disappears. But if a minute opening remains in the sclera, the œdema, too, persists—a condition which is designated as cystoid cicatrization (see Fig. 135). Œdema from filtration is found either in the region of the fistulous opening or in the lower part of the bulbar conjunctiva, the fluid sinking in obedience to gravity.

Not infrequently we see in the scleral conjunctiva small bright vesicles, arranged in a row like a string of pearls, or even joined so as to form longer sausage-like swellings. Here we have to do with dilated lymph-vessels that are filled with a clear liquid (*lymphangiectasiæ*). These occur both in inflammation of the conjunctiva and also when the conjunctiva is perfectly healthy.

182. The exudation of blood beneath the conjunctiva of the eyeball is known as *ecchymoma subconjunctivale.* We then see a spot of varying extent and of a vivid-red or dark-red color; sometimes the whole conjunctiva scleræ is suffused with blood. Ecchymosis is easily distinguished from an inflammatory redness of the conjunctiva by its uniformly red coloration, in which no network of vessels can be recognized, and also by its sharp limitation from the unsuffused portions of the conjunctiva, which are ordinarily quite normal and uncolored.

Ecchymoses of the conjunctiva develop after injuries and operations upon the conjunctiva (particularly after squint operations), and, furthermore, in violent inflammations, especially in catarrhal ophthalmia. Spontaneous suffusions in an otherwise healthy conjunctiva are frequent in old people, whose blood-vessels have brittle walls, the rupture of the vessels being often occasioned by great bodily exertion, or by coughing, sneezing, vomiting, straining, etc. In children also spontaneous ecchymoses of the conjunctiva are observed, chiefly after whooping cough. A special symptomatic significance belongs to those ecchymoses which develop, to all appearance spontaneously, after an injury to the skull. Here we have to do with cases in which a fracture of the base of the skull has occurred, and the blood, as it escapes, gradually oozes forward through the orbit until it lodges beneath the conjunctiva (see § 619).

Subconjunctival ecchymoses become absorbed in from a few days to a few weeks without producing any ulterior serious consequences, and in themselves require no treatment whatever. We generally prescribe compresses of lead water [or hot water], more for the purpose of appeasing the patient than of obtaining a more speedy resorption.

Ecchymoses of the conjunctiva, free from danger as they are, terrify the patient by their conspicuous look, especially if, as is frequently the case, they spread still further in the next few days after they first develop. In these cases the pinguecula stands out with peculiar distinctness, as a light, white or yellowish spot upon the red substratum. From the transparent cornea the ecchymosis is delimited by a narrow gray border.

This is the inner margin of the limbus, which is too closely adherent to the cornea to be lifted up by the blood. In eyes with blue iris, the latter often has its color apparently changed to green in the spot corresponding to the ecchymosis. This is caused by the extension of the blood in a very thin layer (in which it appears green) between the lamellæ of the cornea, so as to make the iris which lies behind it seem green.

183. Emphysema of the Conjunctiva.—The entrance of air beneath the conjunctiva is sometimes observed simultaneously with the escape of air beneath the skin of the lids or into the orbital tissue (see §§ 618 and 723).

[**Hæmorrhage from the Conjunctiva.**—Hæmorrhage from the conjunctiva ("bloody weeping") is due usually to traumatism (incised or contused wounds, rough or too frequent applications to the conjunctiva—especially too vigorous application of silver nitrate in ophthalmia neonatorum, etc.). In other cases, ulceration or abrasion of polypi or other vascular tumors is the cause (see page 226). Very rarely hæmorrhages occur spontaneously in typhoid fever, in connection with menstruation, and conjointly with retinal hæmorrhages (Wallis and others). The bleeding may be profuse enough, at least in infants, to endanger life.—D.]

Pigmentation of the Conjunctiva.—Pigment is often present in the normal conjunctiva and most of all at the limbus, where, especially in dark-skinned persons, we not infrequently observe discrete dark brown spots or a more uniform brown coloration. [Another instance of what may be called normal pigmentation is the pinguecula (page 136). Pathological types of pigmentation are afforded by the pigment moles and melanosarcomata (see pages 226 and 228), and the staining produced in argyrosis (page 146) and by various chemicals (page 213). Circumscribed, often extensive, sharply defined black spots (*melanosis of the conjunctiva*) occasionally occur in chromidrosis, or as the result of an unabsorbed hæmorrhage, or from other causes (Blanchard, Wirtz, Randolph). These are not in themselves malignant.—D.] Yet, in most cases, melanosarcomata develop from them later.

XIV. Tumors of the Conjunctiva

184. Benign Tumors.—Both malignant and benign tumors occur in the conjunctiva. The most important form of *benign* tumors is the

Dermoid Tumor.—This is a flat growth of solid consistence which, so to speak, straddles the margin of the cornea, being situated partly in the conjunctiva and partly in the cornea, with which latter it is immovably connected. It most frequently occurs on the external (temporal) side of the cornea (Fig. 64). Its color is white or reddish, its surface epidermoid and often dry. It is sometimes covered with fine down, or even with longer hair. Histological examination shows that the growth possesses the composition of the external skin; it consists of a stroma of connective tissue, covered with epidermis, and contains hair follicles and various glands. It is, so to speak, an island of skin upon the surface of the eyeball.

Dermoid tumors are always congenital, and are frequently found along with other congenital anomalies, like congenital harelip or wartlike appendages of skin in front of the ears. Sometimes they grow still larger after birth.

The chief harm that dermoids do is to produce a considerable disfigurement. If they are large, and especially if they are covered with hair,

they cause mechanically an irritation of the eye, and also interfere with vision, in so far as they encroach upon the pupillary area of the cornea. They are removed by a simple process of ablation, which consists in detaching the tumor as accurately as possible from the subjacent cornea and sclera. The resulting wound in the conjunctiva should, as far as possible, be covered by drawing the adjacent conjunctiva over it (see § 820). That portion of the cornea upon which the tumor was formerly situated remains clouded permanently. If remnants of the tumor have been left, the latter may in part form again.

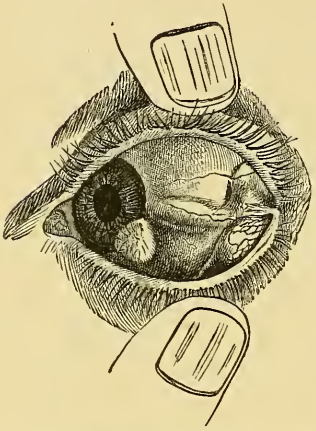

FIG. 64.—DERMOID TUMOR OF THE CORNEA AND SUBCONJUNCTIVAL LIPOMA IN A THIRTEEN-YEAR-OLD GIRL.

The hairy dermoid tumor is situated on the outer and lower margin of the cornea, lying to a small extent in the cornea, and more largely in the sclera. The eye has to be turned strongly inward in order to bring the lipoma well into view. The lipoma has a form differing from the ordinary in that it consists of two lobules, one, more bulging, lying beneath the lower lid, the other, flatter and lying under the upper lid. In addition it sends out a process which runs in the horizontal meridian of the eyeball as far as the outer margin of the cornea. It is everywhere covered by compact conjunctiva which resembles skin, but which nevertheless allows the yellow color of the underlying fat to show through.

Dermoid tumors are solid, not hollow, and hence are not to be confounded with dermoid cysts (§ 730). They occupy a position midway between that of skin and mucous membrane. Their thick epithelium is like epidermis but does not become corneous. The tough connective tissue lying beneath the epithelium resembles the cutis and, like the latter, contains, besides hair follicles, sebaceous glands and often also sweat glands. On the other hand, acinous glands also occur such as are present in mucous membranes (like Krause's glands of the conjunctiva). In rare cases dermoids contain hyaline cartilage or bone (the latter cases being described as *osteomata*), so that complicated structures develop which may be called teratomata.

Remak supposed that dermoids, like dermoid cysts, originated in a fetal invagination of the outer germ-layer. On the other hand, Van Duyse believed that they owe their origin to a circumscribed adhesion between the amnion and the surface of the eyeball, which before the fourth month is not covered by the lids. The adhesion between the amnion and the eyeball afterward is drawn out into a cord and finally breaks in two, its point of attachment to the eye remaining behind as a dermoid tumor. But this hypothesis does not explain why this point of attachment shows the structure of the external skin with glands, since the amniotic bands are simple connective tissue. It is hence more probable that the amnion contributes to the development of dermoids only to this extent, that when there is a smaller amount of amniotic fluid, the amnion is in closer contact with the eye and may press the lid against the surface of the eyeball. The same thing might be done by amniotic bands. As a result of the pressure there might develop a circumscribed adhesion between the lid and the eyeball, which afterward becomes detached but not without rudimentary portions of skin tissue being left on the surface of the eyeball, which rudimentary portions afterwards develop into a dermoid.

Lipoma subconjunctivale forms a tumor, situated upon the upper and outer circumference of the eyeball, between the rectus externus and rectus superior muscle, and appearing of a yellowish color as seen through the conjunctiva. It is of triangular

form, the sharply defined base of the triangle looking toward the cornea, while the two sides of the triangle, which are directed outward pass gradually into the orbital fat. If the tumor is small it ordinarily remains concealed beneath the outer canthus, and can be brought into view only by turning the eye strongly inward (Fig. 64). Larger lipomata are visible in the palpebral fissure even when the gaze is directed straight forward, and hence cause disfigurement; but they do no other harm. Microscopical examination shows that the tumor consists of fat lobules. The conjunctiva that coats it is thickened and of a character resembling skin, and in this regard a lipoma is akin to the dermoids (lipo-dermoid). Like the latter, it is congenital, but sometimes grows to a considerable size at the time of puberty. If it is desired to remove the tumor on account of the disfigurement it produces, we divide the thickened conjunctiva as far as may be, and then remove as much of the mass of fat as is visible in the palpebral fissure; a radical removal of all the adipose tissue is unnecessary.

Cysts in the conjunctiva are commonly represented by small vesicles filled with a limpid liquid. Most of these vesicles, and particularly those situated upon the conjunctiva of the eyeball, originate from dilated lymph vessels (see page 222). In the retrotarsal fold cysts occur which take their starting point from Krause's glands or from new-formed glands in the conjunctiva (page 134); and there are also congenital cysts and cysts that are produced in the conjunctiva by traumatism. Larger cysts situated ene a th the conjunctiva are formed by the cysticercus cellulosæ. The latter is observed for the most part in children or in adolescents. In such a case the conjunctiva at one particular spot is found traversed by dilated vessels, and forms a nodular protuberance (Fig. 65). Underneath may be felt the cyst, which, as a rule, can be readily pushed about upon the subjacent parts; in individual cases, however, it is intimately adherent to the subjacent sclera or to one of the ocular muscles. If the cyst has very thin walls, the head of the worm can be recognized as a whitish spot in its interior. It is easy to remove the cysticercus by splitting up the conjunctiva and dissecting out the cyst, which consists of the cysticercus vesicle inclosed in a capsule of connective tissue that has been formed about the animal.

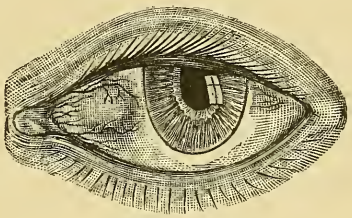

Fig. 65.—Cysticercus Subconjunctivalis. Magnified 1½×1.

In a girl ten years of age a small blood-red spot was observed on the nasal side of the cornea two months before she came under treatment. This spot became constantly more elevated and at the same time paler until it assumed its present form, that of a freely movable cyst, lying beneath the conjunctiva.

Under the name of *polypi* of the conjunctiva we understand soft, rarely rather hard, outgrowths which are attached to the conjunctiva by a pedicle, and whose smooth surface is covered with mucous membrane. These originate most frequently in the retrotarsal fold or in the conjunctiva of the lids. As a rule, they are so small as not to be disclosed until the lids are everted; sometimes, however, they are large enough to project out between the lids. Larger polypi are frequently ulcerated upon their surface, as a result of the mechanical injuries to which they are necessarily subjected. In their nature polypi are small fibromata, which push out the conjunctiva in a sac-like protrusion before them. Their treatment consists in ablation and a subsequent cauterization of their base with the silver-nitrate stick.

Papillomata of the conjunctiva are frequently confounded with polypi, but are distinguished from them by their surface being not smooth, but papillary—that is, nodulated, like a raspberry or cauliflower. They are either provided with a pedicle or are sessile, having a broad base and extending in a flat layer over quite a large section

of the conjunctiva. They start most frequently from the region of the caruncle, but may also take their origin from other portions of the conjunctiva, and sometimes several papillomata at once are present in different portions of the conjunctiva. They must be removed very thoroughly, since they are extremely apt to recur.

A third form of tumor, having an external resemblance to polypi of the conjunctiva, are the *granulation tumors*. These, like polypi, form small, mushroom-like, pedicled tumors. Unlike polypi, however, they are not covered by conjunctiva, but consist of naked granulation tissue. They develop in places where a loss of substance exists in the conjunctiva, whether as a result of ulcers (or even large efflorescences in cases of conjunctivitis eczematosa) or as a result of injuries or of operations (most frequently after tenotomies, at the site of the conjunctival wound, and, after enucleation, at the bottom of the conjunctival sac). In the case of chalazia, also, which have broken through the conjunctiva of the lids, we quite often see a granulation mass growing out of the opening. After existing for some time granulation tumors become more and more constricted at their base by the cicatrical contraction of the surrounding conjunctiva, so that they ultimately fall off of themselves if they have not been previously removed.

The three varieties of tumors above mentioned often contain an abundance of dilated vessels; in fact, some cases of very vascular polypi have been described as pediculated angiomata of the conjunctiva. It is hence easy to conceive how these tumors can readily give rise to repeated hæmorrhages, especially if they are ulcerated in spots and are mechanically injured, as, for example, by the rubbing of the lids upon them (see page 223).

Hæmangiomata of the conjunctiva are, as a rule, those which were originally situated in the lids and have gradually passed over upon the conjunctiva. Primary angiomata are of rare occurrence in the conjunctiva, and are generally found in the region of the inner angle of the eye. They are for the most part congenital, and increase in size after birth. For treatment, see § 620.

Other benign tumors occurring, though very rarely, in the conjunctiva are fibromata, myxomata, and lymphangiomata. Furthermore, there are small connective-tissue growths which consist of plasma cells and are hence called *plasmomata*. It is not yet determined whether these are to be classed among chronic inflammatory growths or among neoplasms in the narrower sense of the word.

185. Malignant Tumors.—Of malignant tumors, epithelioma and sarcoma of the conjunctiva occur. These originate ordinarily in the limbus conjunctivæ and thence extend both into the conjunctiva and into the cornea.

Epithelioma of the conjunctiva forms a non-pigmented, flat, sessile tumor with a broad base. It remains for a long time confined to the superficial layers of the conjunctiva and cornea, its extension in the latter often resembling a pannus (Figs. 66 and 67). The tumor shows a great tendency to superficial ulceration.

The *sarcomata* that start from the limbus are generally pigmented (melano-sarcomata). In opposition to epitheliomata, they grow more in height than in breadth, and are attached to the substratum by only a slender base. They hence form dark-colored, very prominent mushroom-like growths, which often overlie a great part of the cornea, although, if we lift them up, we find the cornea beneath for the most part normal (Figs. 68 and 69). Both epitheliomata and sarcomata develop in advanced life, and, if they are not removed, spread steadily; sarcomata, especially, grow until at

length they form gigantic tumors. At last the patient succumbs from exhaustion or from metastases which form in the internal organs. The tumors must therefore be removed as early and as radically as possible. As long as they are still quite small and superficial, radical removal can be performed with preservation of the eyeball. The tumor is removed as completely as possible, partly with the knife, partly with the sharp spoon, and

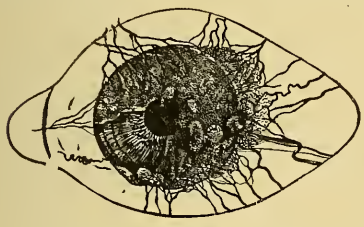

FIG. 66.

FIG. 67.

FIG. 66.—EPITHELIOMA OF THE LIMBUS AND CORNEA. The patient, a man of fifty-six years of age, had noticed, as much as thirteen years before, that a pellicle was beginning to grow on the outer border of the cornea in the left eye. With the exception of its lower inner quadrant the cornea is entirely covered by a gray deposit, the surface of which is coarsely nodular and is traversed by numerous vessels which arise from the limbus. In the deposit can be seen with the aid of a magnifying glass scattered bright dots (epithelial pearls). Owing to these the deposit in spots acquires a greasy aspect. The new growth passes from the cornea on into the limbus and at the temporal side a little way into the conjunctiva of the eyeball, which at this spot has also taken on a nodular and in places gelatinous character.

FIG. 67.—SECTION THROUGH THE ANTERIOR SEGMENT OF THE SAME EYE. The section starts from the upper and outer part of the eye and runs downward and inward.

the spot which it has occupied is thoroughly burnt with caustics, or, still better, with the actual cautery. [Radium has been used successfully in some cases (Treacher Collins).—D.] If complete removal of the growth is no longer possible in these ways, the eye, too, must be removed, even though it still possesses visual power.

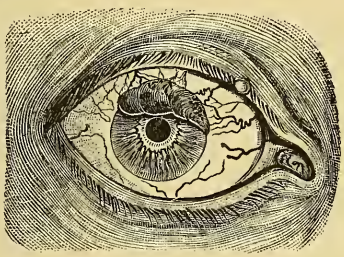

FIG. 68.

FIG. 69.

FIG. 68.—MELANO-SARCOMA OF THE LIMBUS ENCROACHING UPON THE CORNEA. The dark brown, sausage-shaped tumor starts from the limbus and overlies the upper third of the cornea. On its surface can be seen dilated veins. In the sclera the anterior ciliary veins are conspicuously visible, emerging at some distance from the margin of the cornea. The border of the upper lid presents near the inner angle of the lids, a small limpid cyst. This is situated right in the row of lashes and is derived from a gland of Moll.

FIG. 69.—A PERPENDICULAR SECTION OF THE SAME EYE. The tumor simply lies upon the cornea without penetrating into it.

[Malignant tumors of the conjunctiva, especially sarcomata, often recur after even thorough removal. Verhoeff and Loring (cited by De Schweinitz) say that 80 per cent of epibulbar sarcomata recur, and hence believe that with these tumors we should not try to save the eye, but should

enucleate at once. Superficial pigmented spots at the limbus should always be regarded with suspicion, and should either be removed at once or should be kept under observation and removed the moment they begin to enlarge (Weeks); cf. pages 223–229.—D.]

We must distinguish between epitheliomata and sarcomata which originate in the surrounding structures, especially the lids, and secondarily pass over upon the conjunctiva, and those which from the beginning are situated upon the conjunctiva and are hence to be looked upon as primary conjunctival tumors. These latter originate mostly from the limbus. The predilection shown by *epitheliomata* for the boundary line between the conjunctiva and cornea is to be considered as an analogue of the fact that, in other parts of the body also, epitheliomata occupy by preference those spots where one kind of epithelium passes into another, as, for instance, the boundary line between skin and mucous membrane (anus, lips, margins of the lids, etc.). An additional factor giving rise to the development of epitheliomata at the limbus is

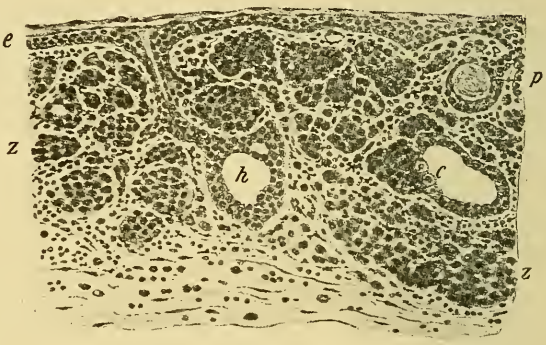

Fig. 70. Fig. 71.

Fig. 70.—Nævus Pigmentosus Limbi. Natural size.

Fig. 71.—A Portion of the Same Nævus. Magnified 113×1. The tumor is formed of cells which, being packed closely together, are arranged in spherical agglomerations, *z*. These are separated by slender bands of connective tissue which carry pigment cells. The epithelium, *e*, of the conjunctiva in many places penetrates into the depth of the growth between the agglomerations of cells. There are thus formed either solid plugs composed of cells, many of these containing pearls of laminated epithelium, *p*, or hollow gland-like invaginations, *l*, whose lumen in its deeper portion is ordinarily dilated to form rather large cavities, *h*. In many of these spaces, e. g., at *c*, there are found within the epithelial cells coccidia, which settle by preference in protected portions of the conjunctiva and act as cellular parasites.

probably furnished by the anatomical character of the latter. The limbus is the only place in the ocular conjunctiva in which small papillæ are normally present. Between these papillæ we sometimes find, even in the healthy eye, a proliferation of epithelium, the latter growing in the form of conical processes into the depth of the tissue.

[The dissemination of epitheliomata takes place mainly by the lymph rather than the blood channels (Treacher Collins).—D.]

Sarcomata of the conjunctiva are almost always pigmented, differing in this respect from the epitheliomata. Melanotic sarcomata, as is known, develop in those localities in which pigment is already normally present. They hence occur upon the conjunctiva of the lids, and above all upon the limbus, two divisions of the conjunctiva which even under physiological conditions contain pigment. Apart from the brownish spots that are sometimes found as a physiological condition (page 223), real pigment moles (*nævi pigmentosi*) occur. These are most frequent in the limbus (Fig. 70) and at the border of the lid. They are small, flat, superficially smooth tumors which are sometimes but

little, sometimes very much, pigmented. Their structure is analogous to that of the soft nævi of the skin, cells being deposited in the conjunctiva which are grouped to form nests or globes composed of cells (Fig. 71, *z*). The origin of these cells is contested, since some derive them from the endothelial cells of the lymph vessels (Recklinghausen), others from the surface epithelium (Unna) or from the chromatophores (Ribbert). Apart from this, there is also a proliferation of the epithelium, the latter growing down into the deeper parts in the form of gland-like invaginations which are sometimes solid, sometimes hollow (Fig. 71, *l*). This fact is the reason why such tumors have been classed by some authorities as epitheliomata. If such nævi, as not seldom happens, start to grow and develop into malignant tumors, it is always a sarcoma that forms from them, because the growth of epithelium soon falls quite into the background in comparison with the growth of the cell nests.

It is impossible to make a radical removal of epibulbar epitheliomata and sarcomata and at the same time preserve the eye, in cases in which these tumors have so great an extent superficially that the conjunctiva has to be extensively sacrificed; for in that case there would occur as a result of the operation so extensive a formation of cicatrical tissue, with consequent distortion and fixation of the eyeball, that the latter would become unserviceable for purposes of vision, and it would have been better to extirpate it at the beginning. So, also, the eyeball must be sacrificed in those cases in which the tumor grows down into the depth of the tissues at any one point—a thing which is especially apt to happen along the anterior ciliary vessels. Such an occurrence is often not discovered until after the removal of the superficial growth, or may even not be discovered at all. In the latter case a recurrence in the same locality occurs soon after the apparently radical removal. The following history serves to show the malignancy of these tumors which in the beginning are of such insignificant minuteness:

In the year 1879 there appeared in the eye-clinic, at that time directed by Arlt, a woman fifty-seven years of age, having a melano-sarcoma on the right eyeball. This had developed from a minute red spot which had already existed for a series of years, and which had begun growing larger the year before. It had the dimensions of a large pea, was of reddish-brown color, and was situated in the conjunctiva on the outer side of the cornea. The base of the tumor projected from the limbus somewhat into the cornea, but nevertheless did not extend into the pupillary area, so that the visual power was perfectly normal. I extirpated the tumor by cutting through the conjunctiva some distance from the edge of the tumor, and then removed the latter as carefully as possible from its base. The wound surface thus formed, which lay for the most part in the conjunctiva and to a smaller extent in the cornea, was scraped, and then the edges of the wound in the conjunctiva were united by a suture. Healing followed by first intention, and for a time the patient remained well. It was not till May, 1886—that is, seven years later—that the patient returned, having again an epibulbar melano-sarcoma on the right eye, which, however, this time was situated on the limbus at the inner side of the cornea, and formed a brown tumor half the size of a lentil. The thin cicatrix remaining from the first tumor was still present, quite unchanged, at the outer margin of the cornea; the limbus at the upper and lower margins of the cornea also was quite normal. For this reason it was impossible to regard the melano-sarcoma, now situated at the inner corneal margin, as a recurrence of the tumor removed seven years before from the external corneal margin. In fact, it could only be referred to a disposition toward the formation of tumors, inherent in the limbus, so that after the removal of a tumor at one spot a similar one developed in another. (The same thing holds good for a case of epithelioma that I observed, which developed simultaneously and quite independently in both eyes, and in both at the inner margin of the cornea.) The small tumor was removed, and the place where it had been situated was superficially cauterized with the galvano-cautery loop. Recurrences, however, followed

this extirpation in rapid succession. Four months later, in September, 1886, the woman returned with a recurrent growth at the upper corneal margin, and upon the removal of this, after another four months, two nodules formed in the conjunctiva to the inner and lower side of the corneal margin and at some distance from it. In order the more certainly to remove everything that was diseased, I resolved this time to enucleate the eye, although it was still serviceable for purposes of vision. In spite of this, a solid nodule made its appearance, not more than six months later, on the floor of the orbit. The woman put off having this removed, and did not come to the clinic until five months later. Meanwhile the glands in front of the ear, at the lower jaw, and on the anterior aspect of the neck had become enlarged and could be readily felt. Although now a radical operation, consisting of a complete exenteration of the orbit and the removal of all discoverable glands, was performed, nevertheless only a few months afterward enlarged glands were again observed. Since then (in February, 1890) the woman has succumbed to an extension of the growth to the internal organs.

Cylindromata also occur, although very rarely, in the conjunctiva.

The *plica semilunaris* and the *caruncle* which is situated upon it participate in the inflammations of the conjunctiva, so that it is unnecessary to treat of the disease of these parts separately. Sometimes the little hairs which the caruncle always has upon it are so long as to irritate the eye; in that case the hairs must be epilated. The new formations of the caruncle are designated by the old name encanthis;[21] benign new formations, such as simple polypoid or papillary outgrowths of the caruncle, are called encanthis benigna; malignant new formations are called encanthis maligna.

[21] From ἐν, in, and κανθός, angle of the eye.

CHAPTER II

DISEASES OF THE CORNEA

Anatomy

186. The *cornea,* together with the sclera, represents the outer fibrous envelope of the eyeball, of which the cornea forms the transparent portion. Seen from in front, the cornea has the form of a horizontal ellipse, the horizontal diameter of its base, which is 12 mm., surpassing the vertical diameter, which is 11 mm. It is thinner in the center than at the edges, where its thickness amounts to about 1 mm. Hence it follows that the curvature of the posterior surface is somewhat greater than that of the anterior. The latter has on the average a radius of curvature of 7.5 mm. Since the radius of curvature of the whole eye is more than this—amounting, in fact to 12 mm.—the curvature of the cornea is greater than that of the rest of the eyeball; the cornea therefore rests on the sclera like a watch crystal. The same comparison also obtains for the method in which the cornea is inserted into the sclera; for, in its posterior layers the cornea extends farther toward the periphery than in the anterior, where the sclera, as it were, laps over the edge of the cornea. The microscope, however, does not show any sharp boundary line between cornea and sclera; on the contrary, the fibers of one pass continuously into those of the other.

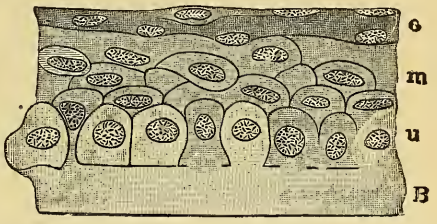

Fig. 72.—Epithelium of the Cornea. (After H. Virchow.) Magnified 660×1.

B, Bowman's membrane; *u* lowest, *m* middle, *o* uppermost layer of cells.

The healthy cornea is transparent. Almost all morbid changes of the corneal tissue make themselves known at once by a diminution of this transparency. In advanced age, however, a cloudiness makes its appearance even in the healthy cornea (arcus senilis corneæ or gerontoxon[1]). This consists of a narrow gray line which runs near the corneal margin, and is concentric with it. It shows itself under the form of a gray arc, first at the upper, soon after at the lower, margin of the cornea; finally the two arcs unite at the outer and inner side of the cornea to form a closed ring. The outer boundary of the arcus senilis is sharply defined, and is separated from the limbus by a strip of perfectly clear cornea; on its inner aspect, or the one turned toward the center of the cornea, on the other hand, the cloudiness gradually shades away until it is lost in the transparent cornea.

[1] From γέρων, an old man, and τόξον, bow.

While the cornea, viewed from in front, appears elliptical, it is circular if looked at from behind. Hence the elliptical form of the front surface of the cornea is due to the fact that both sclera and conjunctiva overlap it farther above and below than at either side.

The *arcus senilis* consists of very minute drops of fat which lie in the lamellæ of the cornea as far back as Descemet's membrane (Fig. 73). In addition to these there are often flakes of hyalin, the size of which varies from that of the finest dust to pretty large concretions. Sometimes also there is found a deposit of lime granules. The cause of the arcus senilis is supposed to be a senile atrophy of the limbus conjunctivæ with destruction of a part of the vascular loops contained in it. [It occurs not infrequently in the middle-aged or even the young, either as the result of premature degenerative changes or from the continuous action of irritants (pitch dust, etc.).—D.]

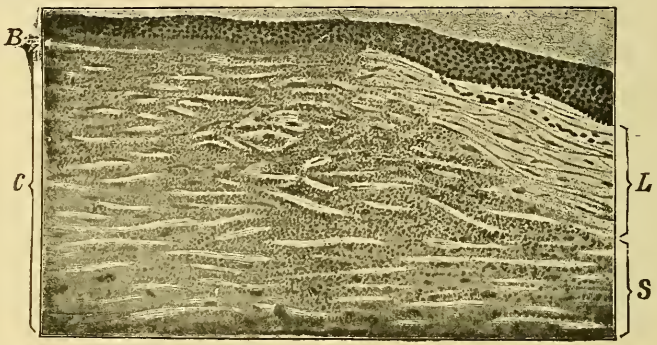

Fig. 73.—Arcus Senilis. (After Takayasa.) Magnified 75×1.

Bowman's membrane, *B*, is rendered cloudy by a deposit of extremely fine fat granules. This is still more the case with the lamellæ of the cornea, *C*, where the granules are larger and lie inside of the lamellæ, not in the interspaces between them, which in the drawing are left light. In the most superficial lamellæ the deposit of fat granules stops where Bowman's membrane ends and the conjunctiva of the limbus, *L*, begins. The deeper we go, the further toward the periphery do the granules extend, finally reaching even as far as the sclera, *S*.

187. The cornea consists of the following layers:

1. The *anterior epithelium* (Fig. 72; Fig. 74, *E*). This is a pavement epithelium consisting of several layers; the lowermost cells (foot cells, Fig. 72, *u*) are cylindrical, then follow rounded cells (Fig. 72, *m*), and finally flat cells (Fig. 72, *o*).

2. *Bowman's membrane* (lamina elastica anterior, Fig. 74, *B*) is a thin, homogeneous membrane closely connected with the corneal lamellæ lying beneath it. It may be said to represent the uppermost layer of the stroma of the cornea, which has become homogeneous and destitute of cells. From the epithelium it is separated by a sharply defined border; and, under pathological conditions as well as after death, the epithelium separates readily from Bowman's membrane.

3. The *stroma* (Fig. 74, *S*). This is composed of a ground substance and of cells. The ground substance in its ultimate constitution consists of minute fibers of connective tissue, united by a cement substance into flat bundles. The bundles are so applied to one another that lamellæ (Fig. 74, *l*) are produced; by the arrangement of these lamellæ in layers one above

another the cornea is built up. The latter has, therefore, a laminated structure. The individual lamellæ are connected together so that it is impossible to separate the cornea into its lamellæ perfectly and without tearing many fibers. Moreover, there are minute elastic fibers in the cornea, which form a specially dense network in front of Descemet's membrane.

Between the lamellæ lie the cells of the stroma of the cornea, the *corneal corpuscles*, which are of two kinds—non-motile and motile cells. The former are the *fixed corpuscles* of the cornea. They are cells with a large

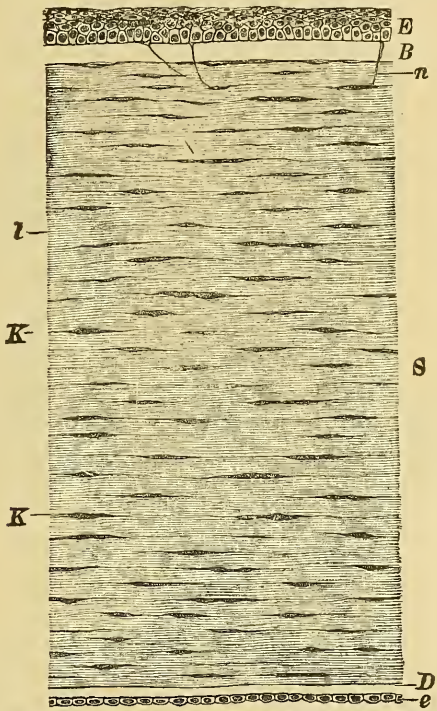

Fig. 74.—Cross Section through a Normal Cornea. Magnified 100×1.

E, anterior epithelium; *B*, Bowman's membrane; *S*, stroma, composed of the corneal lamellæ, *l*, and the corneal corpuscles, *K*; *D*, Descemet's membrane; *e*, posterior epithelium; *n*, nerves extending through Bowman's membrane and the epithelium.

nucleus and a very flat, protoplasmic cell body, the numerous branched processes of which are connected with the processes of adjoining cells so that in this way there is formed a system of connected protoplasmic bodies (cells and their processes, Figs. 75, 76, and 77).

The second variety of cells belonging to the stroma are the *motile corpuscles* of the cornea (wandering cells), which were discovered by Recklinghausen. These are nothing but white blood-corpuscles which have made their way into the cornea and which move about in its system of lymph passages. In the normal cornea they are present in very small amount; but

whenever any irritation acts upon the cornea they at once increase considerably in number, since they escape from the network of blood-vessels forming the marginal loops and pass into the cornea. These cells play an important part in inflammation of the cornea.

4. *Descemet's membrane* (*D*, Fig. 74) is a homogeneous, hyaloid membrane which forms the posterior boundary of the cornea. Unlike Bowman's membrane, it is quite sharply separated from the stroma of the cornea; from which also it is chemically different. It is very resistant to chemical

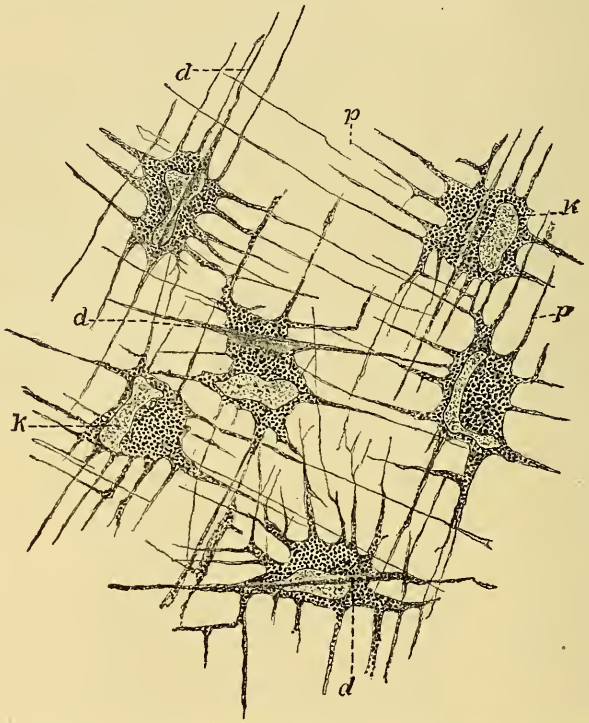

Fig. 75.—Fixed Corneal Corpuscles of the Frog. (After v. Ebner.) Magnified 325 × 1.
k, nuclei of the corneal corpuscles; *p*, processes of the cells, which either cross each other at right angles or anastomose with each other; *d*, pressure-ridges, so called because the way they are produced is that the cell protoplasm is squeezed into the gaps adjoining the lamellæ, and being compressed there are moulded into a rectilinear, ridge-like shape.

reagents, and likewise to pathological processes going on in the cornea. When the entire stroma of the cornea has broken down into pus, we often see the thin Descemet's membrane still for days offering resistance and remaining unimpaired (see § 199).

5. The *posterior epithelium* (endothelium, *e*, Fig. 74). This, under the form of a single layer of flattened cells, coats the posterior surface of Descemet's membrane.

188. The cornea at its margin abuts against three membranes, the conjunctiva, the sclera, and the uvea (iris and ciliary body). Embryology

teaches us that the cornea consists of three superimposed layers, each of which corresponds to one of the membranes adjacent and may be said to represent its continuation over the most anterior portion of the eyeball. The cornea hence consists of three divisions—a conjunctival, a scleral, and a uveal. The conjunctival portion of the cornea (the so-called conjunctiva corneæ) consists of the anterior epithelium and of Bowman's membrane, and also in the marginal portions of the cornea of the most anterior lamellæ

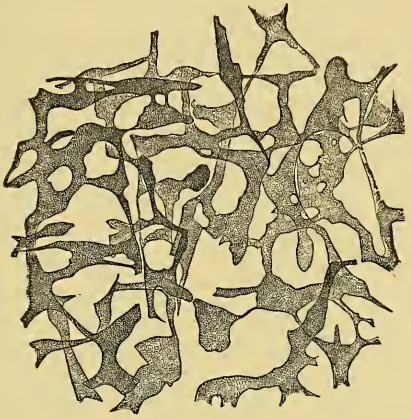

Fig. 76.—Corneal Corpuscles of Man. Magnified 145 × 1.

The description of corneal corpuscles ordinarily given is taken from the corneæ of animals, and it corresponds to the picture given in Fig. 75 of the frog's cornea. The figure here given is drawn from a section which was made through the cornea of an adult man parallel to its surface, and which was stained while still quite fresh with gold chloride. The section comprises several successive lamellæ and hence shows corneal corpuscles which lie in different planes, those which are drawn of lighter shade being the deeper. The processes of the cells are few and thick; the nuclei of the cells in this preparation are not stained enough to be visible. [Cf. Fig. 77.]

Fig. 77.—Corneal Corpuscles of Man. Magnified 250 × 1.

From another cornea, also stained while still fresh with gold chloride. The corpuscles represented all lie between the same two lamellæ and consequently in the same plane. They are connected by thick processes with each other and appear to form a single protoplasmic network, since the boundaries between the separate cell territories are not visible. The nuclei lie grouped together.

of the corneal stroma, which at the limbus merge into the tissue of the conjunctiva (Fig. 73, *L*). The most posterior lamellæ of the corneal stroma and Descemet's membrane, together with the posterior epithelium lining it, belong to the uvea, while the middle lamellæ of the cornea represent the continuation of the sclera (Waldeyer).

In the fully developed eye these three divisions are fused into a common whole, although their community of origin with the adjacent

membranes still finds expression under pathological conditions; the conjunctival division suffering most of all in diseases of the conjunctiva, the uveal division in diseases of the uvea.

189. The cornea contains no *vessels*. These cease at the margin of the cornea, forming there at the limbus the network of marginal loops which is supplied by the anterior ciliary vessels (see page 137, and Figs. 43 and 44). From the marginal loops the blood-plasma passes over into the stroma of the cornea.

The *nerves* of the cornea arise partly from the ciliary nerves, partly from the nerves of the bulbar conjunctiva. They are very numerous, particularly in the uppermost layers of the stroma, from which the nerve fibers pass forward through Bowman's membrane into the epithelium as far as the most anterior layers of the latter (Fig. 74, *n*). The cornea is therefore extremely sensitive to the touch. In the induction of narcosis the reflex squeezing together of the lids that follows from touching the cornea is employed to test the depth of the narcosis, since this reflex act is among those that are the last to disappear. Lesions of the cornea are particularly painful whenever they affect the uppermost layers, which are so rich in nerves, as, for instance, in the case of exfoliation of the epithelium, by which the numerous fibers of the epithelial plexus of nerves are laid bare.

Clinical Examination of the Cornea

190. An examination of the cornea must have regard to the following points:

1. The *size* and *form* of the cornea. Both may be altered either in consequence of congenital defects or because of morbid processes. Overlapping of the cornea by the limbus to an unusual extent, or the presence of marginal opacities in the cornea, not infrequently simulates a diminution in size or an irregularity of form.

2. The *surface* of the cornea must be examined with regard to its curvature, its evenness, and its polish. In respect to (*a*) the *curvature* of the cornea as a whole, marked anomalies are recognizable at the first glance; slighter changes, however, require more precise examination by means of the reflex images (see pages 77 and 79). The cornea acts as a convex mirror, the greater the curvature of which the smaller is the reflected image. In order to be able to judge whether the reflex image given by a particular cornea is of abnormal dimensions, we must compare it with the image reflected from another cornea which is healthy, and most conveniently with that from the cornea of the other eye in case it is normal. The diagnosis is easy when the curvature varies at different portions of the same cornea (as, for instance, in keratoconus, in which the central portions have a greater curvature than those at the periphery). In this case we cause the eye to move about in such a way that the reflection of a window opposite falls successively upon different portions of the cornea, and then we see the

reflex image becoming larger or smaller according to the varying curvature of the cornea. The *evenness* (*b*) of the corneal surface, together with the perfection of its polish, gives to the normal cornea its brilliant luster. Here, again, the reflex images afford the best means of testing both of these properties. These images in the normal cornea are *regular* and *clear-cut*. If there is unevenness of the surface, the image at the spot where the cornea is uneven is clear-cut but *irregular;* it looks distorted because its outlines are irregularly bowed in or out (Fig. 78, II). The form and extent of the inequality may be deduced from the kind of distortion of the reflex image. The inequality of the corneal surface becomes particularly evident when we use Placido's keratoscope (see § 788 [and Figs. 398 and 399]). By the ophthalmoscope, too, we can recognize inequalities of the corneal surface, on account of the irregular astigmatism which they produce (page 110). The uneven spots upon the corneal surface are either depressions (losses of sub-

Fig. 78.—Behavior of Reflex Images on the Cornea.

I. On the *normal* cornea. Reflex image brilliant, sharp and regular.

II. In *unevenness* of the surface. Reflex image sharp and brilliant, but irregular. Over the old opacity of the cornea which is associated with flattening, the image of the window is broader because the surface is less convex.

III. In *dulness* of the surface. Reflex regular, but ill-defined and not brilliant. The cornea is stippled and hence the outlines of the reflex image show a very fine zigzag indentation.

IV. In combined *unevenness* and *dulness* of the surface. Reflex image irregular, ill-defined, and not brilliant. IV represents II after the cornea has become dull by the development of increase of tension.

stance) or elevations. Furthermore, the cornea may be uneven from being wrinkled (rhytidosis[2] corneæ) or from being entirely collapsed (collapsus corneæ). Both of these conditions occur in cases of great diminution in the tension of the eyeball, hence particularly after the escape of the aqueous or vitreous humors. If (*c*) the *polish* of the cornea is entirely lost, the latter becomes lusterless (dull); it looks like glass that has been breathed upon, or looks as if it had been smeared with grease. The reflex images show their normal size and form, but have *lost their sharp outline.* The cornea, therefore, may be smooth as a whole but at the same time may be dull, just as the surface of a ball of ground glass is. Lack of luster of the cornea is also caused by the presence of inequalities so minute as to be nearly or quite imperceptible to the naked eye. Such inequalities—in contrast with those of larger size—are situated only in the epithelium and may consist either of elevations or depressions (Fig. 79). The latter are produced by the detachment of isolated epithelial cells in a number of places, thus leaving minute excavations; the surface of the cornea looks as if *stippled* by needle pricks. In other cases we see the roughness of the cornea produced in the opposite way—that is, by its surface being covered with many small elevations, in

[2] From ῥυτίς, a wrinkle.

which case the cornea looks as if it were made of *shagreen*. Here we have to do with multiple elevations, produced by a lifting of the epithelium, and occurring under the form of minute vesicles.

3. The *transparency* of the cornea is a property which belongs to its parenchyma, not its surface; which latter, in fact, is only a mathematical concept, an expression for the superficies that forms the boundary between the cornea and the air.

Dense opacities of the cornea are visible from a distance; but for the recognition of slight opacities lateral illumination and often also the use of the magnifying glass are indispensable. By means of these aids to diagnosis we determine the form, extent, and denseness of the opacity. We make out whether it is found in the superficial or in the deep layers of the cornea, and further whether it is diffuse or is composed of separate small dots, spots, or striæ. Many an opacity which appears diffuse when seen with the naked eye proves, when viewed with a magnifying glass, to be com-

Fig. 79.—Dull Cornea. Magnified 103 ×1.

The deeper layers of the epithelium are normal. In the superficial layers many of the cells are swollen, and others have ruptured in consequence of being swollen. The former cause small elevations, the latter small depressions in the corneal surface. Cf. also Fig. 22.

pounded of smaller opacities. Such an opacity may subsequently become really homogeneous by enlargement and confluence of its components. [Changes in the transparency and evenness of the cornea, and particularly distortions of the corneal reflex and the presence of minute elevations and depressions, are often best recognized if the observer places the patient in a semi-reclining position with his face to a window, and, standing behind him, examines the cornea from above with a strong magnifying glass, while the patient is told to move his eye slowly in different directions and the image of the window is focused sharply on the cornea by another lens.

To demonstrate defects in the corneal epithelium we use fluorescein (see page 243).—D.]

4. The *sensitiveness* of the cornea is tested by touching it with the end of a thread or a shred of paper. In many diseases the sensitiveness of the cornea is diminished, or is abolished altogether.

I. Inflammation of the Cornea

General Statement

191. In the course of an inflammation of the cornea (keratitis[3]) we observe the following *stages*: The inflammation begins with an *infiltrate* (Fig. 80). Cells in increased number make their appearance within the parenchyma of the cornea, and these constitute the exudate. As a consequence of this the diseased part loses its transparency. The level of the

[3] From κέρας, horn.

cornea over it is either normal or is elevated; in no case, is it depressed. The epithelium suffers to the extent of losing its polish, so that this portion of the cornea appears dull upon the surface. The clinical signs of the existence of an infiltrate hence are cloudiness of the cornea, and loss of luster over the clouded portions, but no depression of the surface. The subsequent fate of the infiltrate varies. It may go on to resorption or to suppuration.

(*a*) *Resorption* occurs in those cases in which the inflammatory process does not proceed to the point of producing actual destruction of the corneal lamellæ with their exfoliation. In that case no loss of substance is produced in the cornea, but when the height of the inflammation is past, the exudate gradually disappears from the cornea—second or regressive stage of the inflammation. In favorable cases, when the exudate has disappeared by resorption, the diseased spots may become perfectly normal once more and

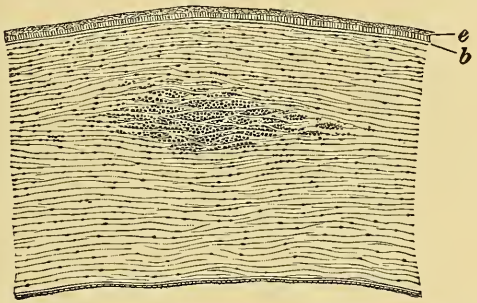

Fig. 80.—Infiltrate in the Cornea. (After Saemisch.)
The epithelium, *e*, and Bowman's membrane, *b*, over the infiltrate are preserved.

regain their transparency (healing without permanent sequelæ—i.e., without opacities). But it may also happen that the stroma of the cornea may not be absolutely destroyed by the deposition of the exudate, and may yet have experienced such an alteration of its structure that, even after the disappearance of the exudate, it never again becomes perfectly transparent. Or it may be that the exudate deposited between the lamellæ of the cornea does not disappear completely by resorption, but in part becomes organized and is left permanently fixed in the cornea. In both of these cases the infiltrate disappears by a process of healing, but leaves a permanent opacity behind. All those cases in which resorption of the exudate occurs without breaking down of the corneal stroma are grouped together under the common name of *non-suppurative keratitis*.

(*b*) The infiltration goes on to *suppuration*, when the exudation is such as to be no longer compatible with the maintenance of life by the corneal stroma, so that the latter breaks down. The inflammation then enters upon its second stage, that of suppuration, which is associated with a localized destruction of the cornea. These cases of keratitis are known as *suppurative keratitis*.

The *histological processes* occurring in inflammation of the cornea have been the subject of the most zealous investigations, and especially of investigations experimentally conducted, because the attempt was made to study in this field the problem of inflammation in general. For such studies the cornea is particularly adapted on account of its transparency, and also on account of the characteristic form of the fixed elements of its tissue. There is no doubt that in every keratitis there occurs an increase in the number of the cellular elements, whose accumulation causes the cloudiness of the cornea, visible to the naked eye, and finally, if very considerable, terminates in the formation of pus. Nevertheless, observers could not agree as to the source of the new cells which made their appearance in the cornea. Some, the leader of whom was Cohnheim, looked upon them as white blood corpuscles which had migrated into the cornea from the vessels of the corneal margin. Others, especially Stricker, believed them to be derived from an increase in number of the normal fixed cells of the cornea. It is now certain that both processes occur, although a different significance attaches to the two. Whenever an irritant productive of inflammation affects the cornea numerous white blood corpuscles wander into the latter, entering it by two ways. The great bulk of the cells is derived from the vessels of the corneal margin, from which they make their way between the lamellæ of the cornea until they reach the inflammatory focus. In certain cases these cells form a ring-shaped infiltration (invasion-ring, Fig. 99) about the diseased area. A few cells wander into the diseased area of the cornea from the conjunctival sac. This occurs when an epithelial defect present in the affected part gives the cells access to the parenchyma of the cornea. The proliferation of fixed corneal corpuscles takes but an insignificant part in the formation of the exudate, but, on the other hand, produces most of the material for the reparative process, which follows the inflammation, and which results in the development of new tissue. (For vascularization see page 244.)

192. The most frequent form of suppurative keratitis is the *corneal ulcer* in which the process of purulent disintegration begins in the most anterior layers of the cornea (Fig. 81). In this way a superficial loss of substance is produced which is recognizable as a depression in the corneal surface. In the beginning, the loss of substance represents only those parts which are most markedly infiltrated, and hence are the first to break down. Surrounding these are layers, which likewise are infiltrated with the exudate, although not to as high a degree. Hence both the floor and the walls of the ulcer are still infiltrated, for which reason we call it a *foul* (coated, infiltrated, or *progressive*) ulcer. Later on the infiltrated portions forming the floor and the wall of the ulcer, so far as they are incapable of living, are also cast off; but those parts of the cornal tissue which have retained their ability to live remain, are freed by a process of resorption from the exudate which infiltrates them, and become once more transparent. Thus, the ulcer has, it is true, become somewhat larger than before, but the cloudiness surrounding it has disappeared; the ulcer has acquired a smooth, transparent base and edges; it is a cleansed (*regressive*) ulcer (Fig. 82).

Among the clinical signs by which we diagnosticate an ulcer, the most important is the unevenness of the corneal surface, the latter presenting a depression or loss of substance. In foul ulcers this depression is surrounded by clouded corneal tissue, which, moreover, is dull upon its surface; the

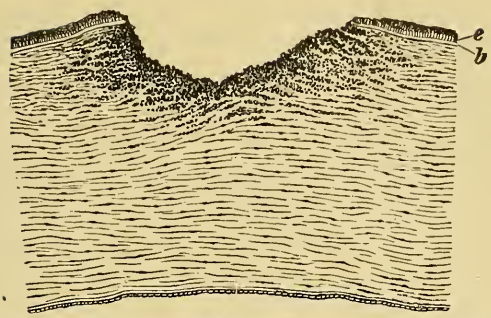

FIG. 81.—CORNEAL ULCER IN THE PROGRESSIVE STAGE. (After Saemisch.)

The floor of the ulcer is formed by an accumulation of pus cells, which also push their way some distance in between the lamellæ of the cornea that adjoin the ulcer. At the edges of the ulcer, which are somewhat raised, the epithelium, *e*, and Bowman's membrane, *b*, end as if cut short off.

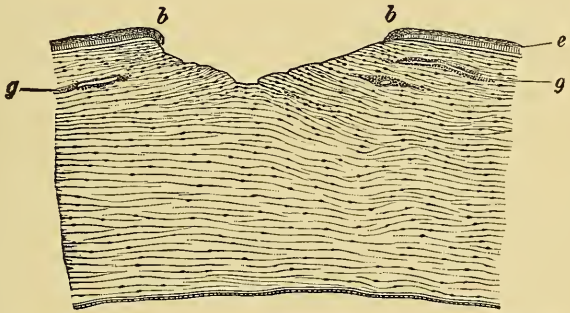

FIG. 82.—CORNEAL ULCER IN THE REGRESSIVE STAGE. (After Saemisch.)

The floor of the ulcer is formed by the denuded lamellæ of the cornea; a slight increase in the number of cells between them can still be made out. At the edges (*b*) of the ulcer the epithelium, *e*, is beginning to grow out over the base. Newly formed blood-vessels (*g*), lying in the upper layers of the cornea, run to the ulcer.

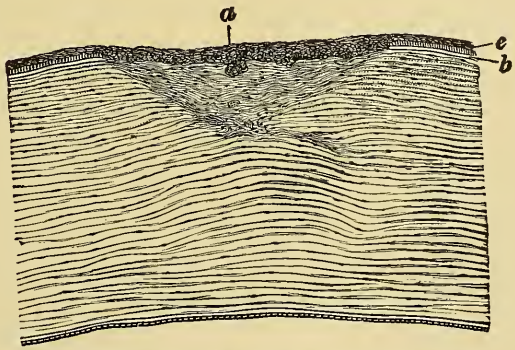

FIG. 83.—CICATRIX OF THE CORNEA. (After Saemisch.)

The epithelium, *e*, is everywhere present, but over the cicatrix it is irregular, and in places (at *a*) is thickened. Bowman's membrane, *b*, is wanting at the site of the cicatrix. The latter itself is distinguished from the tissue of the normal cornea by its denser and less regular texture.

floor of the ulcer also is gray and uneven. In clean ulcers the cloudiness surrounding them is very slight, or is wanting altogether, and the floor and the edge of the loss of substance are smooth and shining; the ulcer gives a mirror-like reflex.

Every keratitis suppurativa entails a loss of substance in the cornea. This must be filled up again by newly formed tissue, in doing which the process enters upon its third stage—that of *cicatrization*. The newly formed tissue is not corneal tissue, but connective tissue, and is accordingly opaque (Fig. 83). Hence suppurative keratitis always leaves a permanent opacity after it. The opacity, indeed, is the principal clinical sign of a cicatrix, for the corneal surface has completely reacquired its luster, because its epithelium is restored to the normal state, and the excavation or loss of substance has disappeared, at most a slight flattening of the surface being present.

In the *healing* of the losses of substance produced by inflammation, the regeneration of the epithelium and the regeneration of the stroma require separate consideration. The epithelium is replaced by the growth of the epithelium from the edge of the ulcer. Losses of substances which affect the epithelium only, heal with a perfect restoration to the normal state and without leaving a permanent opacity. On the other hand, every loss of substance of the corneal stroma is filled up by cicatrical tissue which forms from the bottom and sides of the ulcer. The chief part in this is taken by the fixed cells of the cornea in the immediate neighborhood of the ulcer, as these increase by division, and the cells which thus arise grow into connective-tissue fibers. The tissue thus formed is, however, essentially different from the normal tissue of the cornea (Fig. 83). The regular arrangement of the normal fibers of the cornea is wanting, and so, too, are the stellately branched fixed corneal corpuscles, which are replaced by ordinary connective-tissue cells. Bowman's membrane is never regenerated (Figs. 83, 87, 88, 89). The epithelium (Fig. 83, *e*) hence lies directly upon the cicatricial tissue, from which, being thinner in some places and thicker in others, it is separated by an irregular line.

As a consequence of these peculiarities, the cicatricial tissue never possesses the perfect transparency of the normal cornea, and is hence recognizable even with the naked eye as an opacity. In time, the texture of the cicatricial tissue approximates somewhat more closely to the regular structure of the corneal tissue. It accordingly gains in transparency, and a "clearing" of the opacity is observed to take place—a process, however, which never gets to the point of producing a complete disappearance of the opacity except in the case of quite small and superficial cicatrices.

In deep losses of substance the restoration of the normal *epithelial coating* does not wait to make its appearance until the ulcer itself has been completely filled up by cicatricial tissue. On the contrary, from the moment when the ulcer has become clean the epithelium begins to grow over the latter, and hence begins to cover it at a time when there is no cicatricial tissue present, or only a very thin layer of it (Figs. 87 and 88). At this time the ulcer (on account of the absence of opaque cicatricial tissue) is still almost completely transparent, and, as a consequence of the restoration of the epithelial covering, is smooth and shining. The formation of cicatricial tissue now goes on beneath the epithelium, and by this process the latter is gradually lifted up to its normal level. In proportion as the layer of cicatricial tissue becomes thicker, the degree of opacity naturally increases; but it would be a very great mistake for the physician to conclude from this fact that the disease was advancing.

193. Recapitulation.—As has been stated above, non-suppurative keratitis has two stages, the stage of infiltration and that of resorption. In suppurative keratitis, on the other hand, we distinguish three stages: the stage of infiltration, the stage of suppuration, and the stage of cicatrization (repair). The stage of suppuration is composed of two periods, the progressive (foul ulcer) and the regressive (clean ulcer).

In making the clinical diagnosis of the form and the stage of an inflammation of the cornea we proceed in the following manner: We first examine the corneal reflex. *If the surface over the clouded area is dull, we are dealing with a recent affection, and in that case, if there is no loss of substance, with an infiltrate; but if a loss of substance is present, with a foul ulcer. If the surface is lustrous, the affection is an old one, and, if a loss of substance is present, is a clean ulcer; but if no loss of substance is visible, we are dealing with a cicatrix.*

The signs before given, having regard to changes in the luster, evenness of surface and transparency of the cornea, serve for the *diagnosis* of the variety of corneal disease that is present. The recognition of these changes requires a certain amount of practice and experience. Particularly often is it the case that the alteration in luster of the cornea escapes the beginner, and, especially so when the cornea has tears running over it. These fill up the small inequalities in the corneal surface and cause a dull cornea or actually one which is a little uneven, to appear smooth and even and therefore of normal luster. Moreover, the changes that are characteristic of the different stages of corneal inflammation are not always found combined in a manner as schematic as has been represented. Some examples may show in what way exceptions to them occur. The opacities that are present upon the cornea may be old, and yet the cornea, because of a coincident increase of tension, may look dull and stippled. The surface of the cornea is not always smooth when there are infiltrates, nor yet when there are cicatrices. In the case of infiltrates, a bulging forward of the surface of the cornea often takes place on account of the deposition of an excessive quantity of exudation; in the cases of cicatrices, on the other hand, a flattening may take place in consequence of an insufficient filling in of the loss of substance. In doubtful cases, we must take still other factors into consideration, which may furnish points that will establish our diagnosis. Thus, in the case of a cicatrix, the outline of the opacity is commonly better defined than in the case of a recent opacity due to inflammation. The color of the latter form of opacity varies from gray to yellowish white and to yellow; cicatrices, on the contrary, present rather a pure white, or, if thin, a bluish-white hue. Recent inflammatory processes are associated with attendant inflammatory symptoms (ciliary injection, etc.), which are wanting in the case of cicatrices. By putting all these facts together the right diagnosis can almost always be made.

Fluorescein is used to make more apparent those spots upon the cornea that are destitute of epithelial covering (erosions and ulcers). If a one-per-cent aqueous solution of potassium fluorescein is instilled into the conjunctival sac, the highly fluorescent liquid penetrates solely into those portions of the cornea that are unprotected by epithelium, and such portions consequently are stained a vivid green and, when the excess of potassium fluorescein is removed by washing the eyeball off with water, contrast very distinctly with the uncolored surrounding parts. [Fluorescein is of especial value in demonstrating the very slight defects present in recurring erosions. See § 247. In very slight defects, the staining also may be very slight, simply a punctate stippling of green showing. In deep ulcers going down to Descemet's membrane only

the sides of the ulcer stain with fluorescein, since Descemet's membrane itself does not stain (Butler).—D.]

194. Vascularization of the Cornea.—In inflammation of the cornea we very frequently observe the development of vessels which grow in upon the cornea from its margin. This occurs more frequently during the *process of healing* of corneal ulcers. At the time when the ulcer begins to become clean, we see vessels starting out from that part of the limbus lying next to the ulcer. These vessels lie in the most superficial layers of the cornea and extend toward the ulcer, whose edges they soon reach (Fig. 82, *g*). Their chief office seems to consist in supplying the necessary material for filling in the loss of substance. Their advent therefore is to be regarded as favorable; we know that at the spot where the vessels have reached the ulcer the further progress of the latter is no longer to be apprehended—that, on the contrary, it will there enter upon the process of healing. After the loss of substance has been filled in, the vessels gradually disappear, so that a corneal cicatrix contains fewer of them as it grows older. Nevertheless, the blood-vessels never entirely disappear from the large cicatrices.

In other cases the formation of new vessels accompanies the *progress of the inflammatory process*, and hence belongs, like the exudation itself, to the clinical picture of the inflammation. The best example of this is afforded by the vascular form of parenchymatous keratitis (§ 234).

A kind of vascularization differing from both of these varieties is that which forms one of the symptoms of *pannus*. Here the vessels do not lie in the cornea itself, but in a newly formed tissue deposited upon it, of which tissue they form an essential constituent (Fig. 53).

It is very important to determine the situation of the vessels in the cornea, that is, whether superficial or deep—since by this fact alone we can

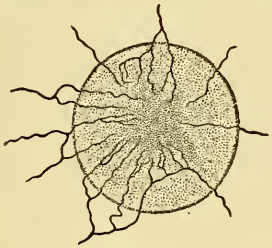

Fig. 84.

Fig. 85.

Fig. 86.

Fig. 84.—Superficial Blood-vessels in Pannus. Magnified 2×1.
Fig. 85.—Deep-Lying Blood-vessels in Parenchymatous Keratitis. Magnified 2×1.—Recent case with brush-like branching.
Fig. 86.—Deep-Lying Blood-vessels in Parenchymatous Keratitis. Magnified 2×1.—Old case with broom-like branching of vessels.

often diagnosticate what sort of keratitis we are confronted with. The type of superficial vascularization is afforded by pannus, that of deep vascularization by keratitis parenchymatosa.

The signs which enable us to distinguish the two kinds of vascularization from each other may be contrasted as follows:

Superficial Vessels (Fig. 84)	Deeply Situated Vessels (Figs. 85, 86)
spring from the net-work of marginal loops of the limbus, and can therefore be followed from the cornea into the limbus, and thence on to the vessels of the conjunctiva.	spring from the vessels of the sclera close to the margin of the cornea, and hence appear to come to an end suddenly at the limbus, as they disappear behind the latter to enter the sclera.
On account of their superficial position are clearly visible and well-defined, and have a vivid-red color.	Are not distinctly recognizable, or are even unrecognizable, except as a diffuse red coloration, and have a dirty red (grayish-red) hue; the reason for these appearances being that the vessels are veiled by the clouded layers of cornea lying in front of them.
The vessels branch in an arborescent fashion.	The vessels either form fine twigs running "broomstraw-fashion"—i.e., parallel to each other—or else diverge like the hairs of a paint-brush.
The corneal surface is uneven, because the vessels raise up the epithelium lying directly over them.	The surface of the cornea is lusterless, it is true, but not uneven.

195. Participation of Neighboring Organs in the Process.—Every keratitis is accompanied by inflammatory symptoms, the most important of which are—

(*a*) The *injection* of the blood-vessels. The characteristic mark of corneal inflammation is ciliary injection. When the inflammation is great conjunctival injection also makes its appearance, and may conceal the ciliary injection to a greater or less extent. Very violent suppurative inflammations of the cornea are accompanied by œdematous swelling of the conjunctiva and even of the lids.

(*b*) The iris and even the ciliary body become inflamed in cases of marked keratitis, so that we see the symptoms of *iritis* and *irido-cyclitis* set in (see §§ 336 and 342). These accompanying inflammations may be intense enough to bring about the destruction of the eye. The exudate poured out by the uvea very often, especially in suppurative keratitis, appears under the form of—

(*c*) *Hypopyon.*[4] This is the accumulation of a purulent exudate at the bottom of the anterior chamber. It forms a frequent symptom in keratitis

[4] From ὑπό, beneath, and πύον, pus.

suppurativa. We then find collected in the lowest portion of the chamber a yellow mass which, because it is fluid, is limited above by a horizontal line, and for the same reason is seen to change its place and seek the deeper portions of the chamber when the head is moved. In other cases the mass is gelatinous, and then when seen from in front often has a border that is convex upward, or it lies quite like a solid ball at the bottom of the chamber and does not change its position with the movements of the head. The amount of the pus varies greatly, ranging from a scarcely perceptible crescent lodged in the lowest sinus of the chamber up to masses of such size that the entire chamber is filled by the hypopyon. Hypopyon may disappear by resorption, this process taking place the more rapidly the more fluid the hypopyon is. Pretty thick masses of exudation may become organized and lead to occlusion of the pupil, or in rare cases, to adhesion of the iris to the posterior surface of the cornea.

(*d*) The *subjective* phenomena associated with keratitis are pain and photophobia, together with the lachrymation and blepharospasm which these conditions cause, and also disturbance of vision—symptoms which are met with in very varying degree.

The *exudation into the anterior chamber*, which accompanies every well-marked keratitis, depends upon the fact that irritant substances from the inflamed cornea diffuse into the aqueous and then act upon the vessels of the uvea (i. e., the iris and ciliary body). From the latter both fibrin and white blood corpuscles pass into the aqueous. Owing to the increased amount of fibrin in the aqueous, coagula are formed on the walls of the anterior chamber; a layer of fibrin is precipitated upon the posterior surface of the cornea (Fig. 170), and often also upon the anterior surface of the iris (Fig. 168). In such cases the cornea, in addition to the circumscribed opacity that corresponds to the area diseased, presents a faint diffused cloudiness produced by the layer of exudate upon its posterior surface. The margin of a fibrinous clot of this sort (which is usually much larger than the inflammatory mass in the cornea) can sometimes be recognized as a faint, gray, circular line which surrounds the corneal mass at some distance from it. If the coating of fibrin lines the cornea throughout, a diffuse cloudiness is produced, which is clinically indistinguishable from a cloudiness of the aqueous. This latter cloudiness is caused by leucocytes, which emigrate from the vessels of the iris and ciliary body and become suspended in the aqueous. Later, by sinking to the bottom of the anterior chamber, these cells form the *hypopyon*. The latter, therefore, originates not from the cornea, but from the vessels of the uvea, as, indeed, is also apparent from the fact that many of the pus corpuscles contain pigment granules, which they have transported with them from the inflamed uvea. For this reason, too, we understand why a hypopyon is found to be free from pus cocci. It is owing to this freedom from germs that the hypopyon is tolerated by the tissues bordering the anterior chamber and produces no special reaction in them. If ordinary pus containing cocci is injected into the anterior chamber of a rabbit, the eye is rapidly destroyed by panophthalmitis. But the pus that constitutes a hypopyon is not only tolerated by the eye with impunity, but can even undergo absorption without leaving any injurious effects behind.

The *resorption* of the hypopyon takes place chiefly through the meshwork of the ligamentum pectinatum. The rapidity with which absorption proceeds varies exceedingly. In many cases a hypopyon of considerable size disappears so completely that

after twenty-four hours scarcely a trace of it is to be found; in other cases the hypopyon remains lying at the bottom of the chamber so long that it becomes organized. Sometimes we observe a rapid alternation in the height of the hypopyon which at times decreases, and again increases once more.

Subdivisions of Keratitis

196. The subdivision of keratitis into keratitis suppurativa and keratitis non-suppurativa corresponds most fully to practical requirements. Every suppurative keratitis, since it is associated with destruction of corneal tissue, leaves behind it a permanent opacity, which in many cases injuriously affects the visual power. On the contrary, so long as purulent dissolution of the cornea has not taken place—that is, in non-suppurative keratitis—a complete restoration of its transparency, and with it of the normal power of vision, is possible, and, in fact, often does take place. Moreover, the above subdivision also corresponds to the essential characters of the corneal inflammations. For it is not merely a matter of accident, nor something that depends altogether on the degree of inflammation, that determines whether an infiltrate in the cornea goes on to suppuration or to resorption. On the contrary, the forms which tend to the production of suppuration ordinarily present from the very beginning characters differing from those borne by forms in which there is no progress toward suppuration, so that these two categories are distinct not only in their consequences but also in their clinical aspect. The suppurative keratitides develop as a rule from the entrance of pyogenic bacteria into the cornea from its surface (ectogenous infection). They have their origin, therefore, generally in external morbific agents; and in the majority of cases, only one focus of disease is present, which starts at some point on the surface and from this spreads continuously either along the surface or into the depth of the cornea. The deep forms of non-suppurative keratitis, on the contrary, are caused as a rule by constitutional disease. The morbific agent is carried to the cornea by the circulation of blood or lymph, and, since it exerts its action on many points of the corneal parenchyma at once, many distinct foci of disease are produced. Hence the cloudiness in the cornea can be resolved by the loupe into numerous small spots which are the separate infiltrates. These lie in the middle and deep layers of the cornea.

To each of the two categories of keratitis a number of different forms belong, the most important of which are set forth in the following list:

A. *Keratitis Suppurativa.*

1. Ulcer of the cornea.
2. Ulcus serpens corneæ.
3. Keratitis e lagophthalmo.
4. Keratomalacia.
5. Keratitis neuroparalytica.

B. *Keratitis Non-Suppurativa.*

(*a*) Superficial forms.

1. Pannus.
2. Keratitis with the formation of vesicles.

(*b*) Deep forms.

3. Parenchymatous keratitis.
4. Keratitis profunda.
5. Sclerosing keratitis.
6. Keratitis starting from the posterior surface of the cornea.

A. *SUPPURATIVE KERATITIS*

1. *Ulcer of the Cornea*

197. Symptoms and Course.—Every ulcer of the cornea develops from an *infiltrate.* In the beginning we find one spot upon the cornea cloudy and the surface over it dull (infiltrate). Then the epithelium exfoliates upon the surface of the affected spot, and soon, by the breaking down of the most strongly infiltrated portions of the cornea, a loss of substance forms in the parenchyma of the cornea, so that an ulcer is produced. This is at first surrounded by infiltrated portions of the cornea—a fact which we recognize by the base of the ulcer being gray and uneven, and its walls likewise gray and clouded. The walls of the ulcer are often surrounded for quite a distance by a gray area, or slender gray striæ, extending from the ulcer in different directions into the transparent cornea. This is an unclean or *progressive* ulcer (Fig. 81). In a favorable case, only so much of the corneal tissue breaks down during the further progress of the disease as was from the very beginning too strongly infiltrated to live. In this case the ulcer rapidly becomes clean without attaining great dimensions. But it very often happens that, simultaneously with the breaking down of the parts that are most strongly infiltrated, the inflammatory cloudiness keeps spreading, new portions of the cornea being constantly attacked by the infiltration. And if these, too, break down into pus, the ulcer grows constantly larger. This progressive growth of the ulcer takes place sometimes more in the direction of its depth, sometimes more along the surface. In the former case, perforation of the cornea is to be apprehended; in the second case, larger and larger areas of the cornea may be destroyed, and thus extensive opacities be produced. Progressive growth along the surface often takes place chiefly in one direction—a fact which can be easily recognized by a particularly marked gray, or even yellow, cloudiness of the ulcer wall on the corresponding side. It may even happen that the ulcer keeps constantly advancing in one direction, while on the opposite side it heals just as fast, so that apparently it goes creeping over the cornea (*serpiginous*[5] ulcer).

The progressive stage of the ulcer is accompanied by *symptoms of irri-*

[5] From *serpere*, to creep.

tation, like ciliary injection, lachrymation, photophobia, and pain, which not infrequently reach a considerable height; moreover, in this stage hyperæmia and even inflammation of the iris make their appearance (evidenced by turbidity of the aqueous humor, hypopyon, discoloration of the iris, contraction of the pupil, posterior synechiæ). There are, however, cases of ulceration in which the irritative symptoms are very slight, or are wanting altogether—cases constituting what are called *torpid* or asthenic ulcers—which, nevertheless, may be very dangerous.

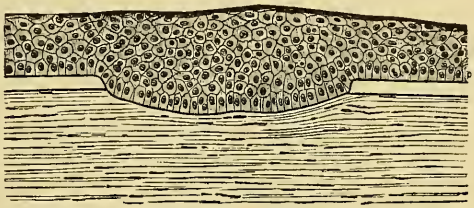

FIG. 87.—HEALED ULCER OF THE CORNEA. Magnified 345×1.

The loss of substance developing as a result of conjunctivitis eczematosa affects Bowman's membrane, *B*, and the most superficial lamellæ of the cornea, *C*. The loss of substance is filled up with epithelium, *E;* right beneath the epithelium, however, the nuclei in the cornea are aggregated somewhat more closely than normal, so that perhaps we are to assume that there is here a very thin layer of connective-tissue fibers. In the eye during life, there was no opacity of the cornea visible; nor was there any facet either, since the surface of the epithelium lies everywhere at the same level.

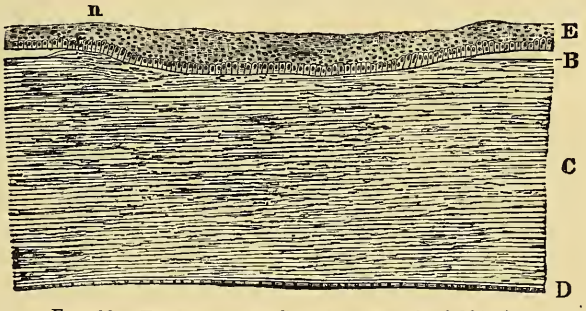

FIG. 88.—FACET OF THE CORNEA. Magnified 90× 1.

In the area of the facet the epithelium *E* shows a shallow depression; Bowman's membrane, *B*, is wanting here, and so are the superficial layers of the cornea, *C*. The larger portion of the loss of substance is filled with epithelium only; only at *n* is there a layer of connective tissue between epithelium and corneal parenchyma. Corresponding to this connective tissue there was a delicate semi-circular opacity which marked one edge of the scar. In its other parts the scar was almost entirely transparent and could be recognized only by the flattening of the surface of the cornea. *D*, Descemet's membrane, with its endothelium.

198. When the infiltration has finally come to a standstill, the ulcer enters upon its *regressive* stage. The tissue that has been destroyed is cast off, that which has not been destroyed becomes transparent once more from resorption of the exudate. The ulcer "cleanses" itself (Fig. 82). A clean ulcer presents a smooth base and edges with little or no opacity, and is to be diagnosticated chiefly by the excavation of the surface of the cornea, which we recognize when examining the corneal reflex. In proportion as the ulcer becomes clean, the associated symptoms of irritation disappear.

After the ulcer has become entirely clean, *cicatrization* begins. Vessels extend from the nearest portions of the limbus to the ulcer, which latter, in

consequence of becoming filled with the opaque mass of the cicatrix, becomes again more clouded, but at the same time constantly shallower, until finally it reaches the level of the adjacent normal cornea. Not infrequently, however, the new formation of the cicatricial mass comes to a standstill before the loss of substance has been quite filled up, so that the surface of the cicatrix remains permanently a little sunken. When such cicatrices are small they are, on account of the thinness of the cicatricial tissue, almost or quite transparent, and disclose their presence by a flattening of the cornea (*corneal facet*), only discernible upon examination of the corneal reflex (Figs. 87 and 88). On the other hand, cicatrices not infrequently occur which project above the level of the surrounding cornea. Such are the cases in which the cornea on the floor of the ulcer, having become thinned, does not offer resistance to the intra-ocular pressure, and

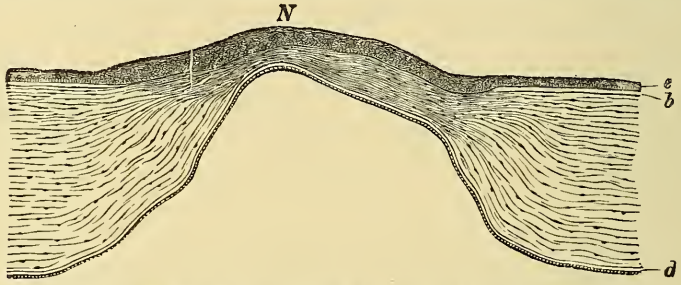

Fig. 89.—Keratectasia Produced by an Ulcer. Magnified 25 × 1.

The thinned and protruding cicatrix is distinguished by its denser texture from the adjacent normal cornea. The epithelium, *e*, over it is thickened, while Bowman's membrane, *b*, is wanting. On the other hand, Descemet's membrane, *d*, with its epithelium, is everywhere present—a proof that the ulcer has not perforated.

bulges forward. The bulging may disappear, owing to the contraction of the cicatricial tissue; but it may also remain permanently (*ectatic cicatrix*, keratectasia[6] ex ulcere, Fig. 89). The formation of ectatic cicatrices is, however, of much more frequent occurrence after perforation of the cornea.

The physician who is called to a patient with an ulcer of the cornea must, after examining the eye, have acquainted himself not only with the diagnosis but also the *prognosis*; he must tell the patient beforehand to what extent his sight will suffer permanent impairment, in order that such impairment may not afterward be charged against the medical treatment. The prognosis for vision depends upon the situation, the extent, and the density of the opacity which the ulcer has left behind it. Small opacities, even when dense, are generally less injurious to vision than those which are less dense but extensive (§ 263). It is hence less serious for an ulcer to extend into the depth of the tissues than upon the surface. If an ulcer is progressing in the direction of the center of the cornea, every millimetre of advance causes additional injury to the vision, while an extension toward the corneal margin is almost a matter of indifference. No further disintegration is to be apprehended at those portions of the margin of the ulcer to which vessels have already penetrated, and so, too, a portion of the cornea, covered by pannus, is protected against suppuration in gonorrhœal conjunctivitis. In every instance the ulcer is arrested at the limbus, as it never makes its way into this or

[6] From κέρας, horn, and ἔκτασις, distention, from ἐκτείνειν, to stretch out.

into the adjacent sclera. The only exception to this is formed by those ulcers which not infrequently develop from the nodules of conjunctivitis eczematosa situated in the limbus (Figs. 92 and 93). Even extensive suppuration of the cornea, as in gonorrhœal conjunctivitis, ulcus serpens, etc., always leaves a narrow rim of cornea intact, which, to be sure, is often not of sufficient size to render possible an iridectomy for the restoration of vision.

[The prognosis and course of keratitis are sometimes also much affected by the patient's general condition, and the keratitis is not completely or at least rapidly relieved until the proper constitutional treatment is applied.—D.]

199. Perforation of the Cornea.—The course which an ulcer takes is much more complicated when the latter perforates the cornea. Perforation takes place when the ulcer has penetrated down to the deepest layers of the cornea. The patient suddenly experiences violent pain, and feels a hot liquid (the aqueous humor) gushing out of the eye, after which, not infrequently, the severe pains previously existing subside. Perforation may occur spontaneously or in consequence of a sudden increase of the intra-ocular pressure, such as may be caused by bodily exertion (even, for example, stooping), or by coughing, sneezing, squeezing together of the lids, crying (in children), etc. The increase in intra-ocular pressure, which develops under these circumstances is to be referred to two causes: it is partly a result of the increase of blood pressure (from the straining of the muscles, and also from congestion in the district drained by the superior vena cava), partly produced by direct compression of the eyeball, and particularly by the pressure which the ocular muscles, and especially the orbicularis palpebrarum, at such times exert upon it. The perforation produced by such causes may develop with violent symptoms and entail very damaging results.

After perforation has occurred we find the anterior chamber obliterated in consequence of the escape of the aqueous humor; the iris and, in the region of the pupil, the lens, also, are applied to the posterior surface of the cornea. If the aperture made by the perforation is of suitable position and size, we see lying in it the iris which has been swept into the wound by the jet of escaping aqueous. The eye feels quite soft.

Perforation is sometimes preceded by a *keratocele*.[7] For, Descemet's membrane being distinguished by the great resistance which, in comparison with the corneal lamellæ, it offers to the inflammatory process, it often happens that the stroma of the cornea is destroyed throughout its entire thickness by suppuration, while Descemet's membrane still remains resistant. In that case it is protruded by the intra-ocular pressure under the form of a transparent vesicle which is visible upon the floor of the ulcer, or which may even project above the level of the adjacent cornea (keratocele or descemetocele). When this vesicle, too, ruptures, the perforation becomes complete. Sometimes the ulcer heals without the keratocele's either rupturing or being flattened out. The latter then remains permanently under the

[7] From κέρας, horn, and κήλη hernia.

form of a vesicle which projects above the surface of the cornea, and which, itself transparent, is surrounded by a cloudy, cicatricial ring.

The direct *effect* of a perforation upon the course of the disease is for the most part favorable, inasmuch as not only the pain and the other symptoms of irritation abate, but the progress of the ulcer also is, as a rule, arrested, and the ulcer rapidly becomes clean. (See page 249). [On the other hand, perforation may produce disastrous immediate effects, such as dislocation and expulsion of the lens and intra-ocular hæmorrhage, and, oftener still, remote ill effects (flattening, ectasia, and fistula of the cornea, glaucoma, intra-ocular suppuration). See pages 254, 255.—D.]

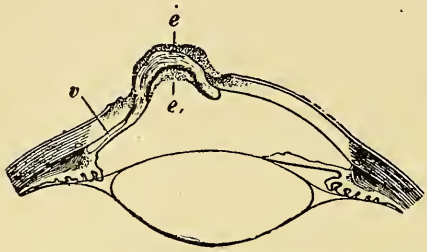

FIG. 90.—PARTIAL PROLAPSE OF THE IRIS (SCHEMATIC).

The iris, which is thickened by the process of infiltration, and is covered on its anterior and posterior surface by the exudate, *e*, e_1, rises up between the sharp edges of the margins of the perforation, which are still infiltrated. Peripherally from the prolapse the iris is approximated to the cornea, although here a remnant, *v*, of the anterior chamber still exists.

200. The method in which the perforation in the cornea closes again varies according as it is placed in front of the iris or the pupil. If the opening is found *in front of the iris*, as is generally the case, it is quickly covered by the iris, which, after the escape of the aqueous humor, is driven forward as far as the cornea. In this way it becomes possible for the anterior chamber to be restored within a very short time, although, to be sure, the iris at the site of the perforation remains permanently connected with the cornea. If the perforation is quite small, the iris simply applies itself to it from behind and there becomes solidly adherent. In case, however, the perforation is larger, the iris, as a rule, is driven into it by the escaping aqueous humor, and thus a *prolapse* or *hernia* of the iris is produced (Fig. 90). This is represented by a hemispherical prominence which, while recent, has the gray or brown color of the iris. Soon, however, this color is changed because of a layer of gray exudation (*e*, Fig. 90) which covers the prolapse like a cap and may be removed with a forceps. When the prolapsed portion of the iris is much stretched, the proper color of the iris is lost and the prolapse looks black, because of the retinal pig-

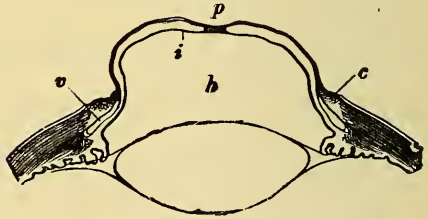

FIG. 91.—TOTAL PROLAPSE OF THE IRIS (SCHEMATIC).

Only the marginal portions, *c*, of the cornea are preserved, and these are still partially infiltrated. Between them bulges the iris, which is driven strongly forward and which consequently is thinned —so that the pigment, *i*, upon its posterior surface shines through it and gives the prolapse a blackish hue. The pupil, *p*, is closed by a membrane, which is continuous with a thin layer of exudate that covers the entire iris. The space, *h*, between the iris and the lens is the enlarged posterior chamber. Of the anterior chamber only the shallow, slit-like, annular space, *v*, is left. This no longer communicates anywhere with the posterior chamber (seclusio pupillæ).

ment on its posterior surface, which appears through the thinner stroma. This is particularly often the case in large prolapses of the iris. The extent of the prolapse of the iris is proportional, first of all, to the size of the perforation. In the worst cases the perforation may comprise the whole cornea, which has suppurated throughout; in that case the iris prolapses through its entire extent (*total prolapse* of the iris, Fig. 91). The pupil is then generally closed up by a plug of exudation (*p*). But the way in which the prolapse occurs has also an influence upon its size. If the perforation takes place with great force (e.g., while the patient is straining hard), or if the patient behaves in a restless manner after it has taken place, a relatively larger portion of the iris will be driven into the perforation.

201. The *cicatrization* of a corneal ulcer, which is associated with a prolapse of the iris, occurs in the following manner, if the prolapse is left to itself: The prolapsed iris in the first place becomes solidly agglutinated to

Fig. 92.

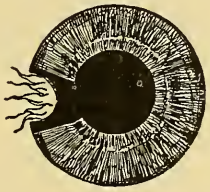

Fig. 93.

Fig. 92.—Scar after Perforation of a Marginal Ulcer with Inclusion of the Iris. The scar is situated very peripherally, partly in fact in the sclera, a thing which can happen only as the result of ulcers due to conjunctivitis eczematosa. The dark central portion of the scar corresponds to the incarcerated iris, the white margin to the solid scar-tissue lying between the iris and the border of the former perforation aperture. The pupil is pear-shaped, with its pointed end displaced toward the scar, and corresponding to this the iris on the opposite side is put on the stretch. Consequently, the pupillary zone and the ciliary zone are broadened, and furthermore the zone of peripheral crypts, which otherwise would be concealed behind the limbus, becomes visible under the form of a dark stripe at that portion of the periphery of the iris opposite the scar.

Fig. 93.—Scar Without Inclusion of the Iris. Here there was also an eczematous ulcer which had perforated. The prolapsed iris had been excised in the proper way. Consequently, it is no longer connected with the scar, but over an area corresponding to the scar shows a coloboma. The pupil is permanently dilated because of the gap made in the sphincter pupillæ.

the walls of the opening caused by the rupture, and wherever it is exposed it is converted by inflammation into a sort of granulating tissue, so that the prolapse soon loses the color of the iris and becomes grayish-red. Subsequently there develops from the proliferating tissue of the iris cicatricial tissue, which first becomes visible under the form of isolated gray bands. By the contraction of these latter, constrictions are formed upon the surface of the prolapse. As the formation of the cicatrix proceeds, these bands become broader, fuse together, and render the prolapse constantly flatter and flatter. Hence in favorable cases the process terminates in the formation of a flat cicatrix situated at the level of the rest of the cornea, and at the site formerly occupied by the bulging prolapse. This cicatrix being mainly a portion of iris that has become cicatricial, it follows that the remainder of the iris still lying in the anterior chamber is solidly united to it. Such an adhesion of the iris to a corneal cicatrix is called an *anterior synechia*.[8]

[8] From συνέχειν, to connect. The term leucoma adhærens (from λευκός, white) is also employed to denote a cicatrix of the cornea with anterior synechia.

Owing to the fact that the iris is drawn forward to the scar, the pupil loses its round shape and is drawn in toward the site of the adhesion. To what extent this is the case depends upon where the perforation is situated and what part of the iris is prolapsed. In peripherally situated ulcers the pupil is drawn strongly toward the site of the perforation; it has the shape of a pear, the tapering end of which is directed toward the site of the synechia (Fig. 92). If, however, the perforation is situated near the center of the cornea (as in Fig. 90), the pupillary portion of the iris becomes engaged in it in healing and in this case the distortion of the pupil is slight, or entirely absent. If the perforation is so large that the entire pupillary margin of the iris is involved in the prolapse and becomes attached to the cornea in healing the pupil is permanently closed by the cicatrix that is formed (Figs. 122 and 123); there are produced occlusio and seclusio pupillæ, together with their destructive consequences.

In the healing of large perforating ulcers of the cornea, the shrinking of the cicatricial tissue is often so great that the corneal cicatrix appears flattened when compared with the normal curvature of the cornea. This flattening, moreover, may extend beyond the cicatricial spot to the portion of the cornea which is still transparent, and then the cornea as a whole becomes flatter (*applanatio corneæ*). If the cornea has been entirely destroyed by suppuration, so that a total prolapse of the iris has developed, the latter ultimately becomes reduced to a small and perfectly flat cicatrix, which takes the place of the cornea (*phthisis corneæ*). The distinction between applanatio and phthisis of the cornea is as follows: In the former the cornea is still present, although it is in part cicatricial, and thus as a whole is flattened. In the latter, on the other hand, nothing of the cornea is left except a very narrow marginal rim which generally withstands the destructive process of ulceration. The flat scar which takes the place of the cornea is, in this case, the cicatrized iris.

The healing of a prolapse of the iris with the *formation of a flat cicatrix* must be regarded, even though the latter is opaque, as a comparatively favorable outcome of a large corneal perforation. Such an eye, of course, is seriously affected, so far as its function is concerned, but yet after the process has run its course, it remains in a state of quiescence, and generally causes its owner no further trouble. The case is different when healing takes place with the formation of an *ectatic cicatrix*. This occurs in the following manner: The prolapsed iris becomes covered with cicatricial tissue, but this is not strong enough to effect the flattening of the prolapse. Hence the latter becomes consolidated in its original form as a protrusion, and is converted into an ectatic cicatrix with inclusion of the iris (*staphyloma corneæ*). A large-sized perforation opening and restlessness on the part of the patient favor the formation of such an ectasis. Whenever a prolapse of the iris has become so extensive that it is constricted by the margin of the per-

foration (or hernial orifice), and hence has acquired a mushroom shape, the formation of a flat cicatrix, without artificial aid, becomes altogether impossible.

202. If the perforation in the cornea is not in front of the iris, but lies *in the region of the pupil*, it cannot be covered by the iris. Its occlusion in this instance takes place more slowly by an outgrowth of new formed tissue (cicatricial tissue) from the margins of the opening, until the opening is filled up. In such a case the anterior chamber remains absent for a somewhat longer time, and meanwhile the lens is in contact with the posterior opening in the cornea. The lens may bear away permanent marks of this contact, most frequently in the form of a circumscribed opacity at its anterior pole (anterior capsular cataract; see § 474). Sometimes the perforation does not close completely by cicatrization, so that a minute aperture remains permanently (*fistula of the cornea*). This appears under the guise of a small dark point, surrounded by opaque, whitish cicatricial tissue; the anterior chamber is absent, the eye is soft. If the fistula persists for a long time, the eye gradually perishes. The cornea flattens, the eye grows softer and softer, and at length goes blind from detachment of the retina. On the other hand, as soon as the fistula closes, an increase of tension is apt to set in, that may lead to a renewed rupture of the occluding substance, which is still but slightly resistant. An alternation of this sort between an open fistula with softness of the eyeball, gradual closure of the fistula, and a consequent steady increase of pressure until the cicatrix ruptures anew, may be repeated for a long time, until finally a severe inflammation or an intra-ocular hæmorrhage occurs, which leads to the atrophy of the eye, and thus puts an end to the process.

Fistulæ of the cornea occur mainly as a sequel of perforations that lie opposite the pupillary margin of the iris, so that the iris cannot block the opening completely, but simply lies with its pupillary border in the perforation orifice. In this case often there is no solid connection present between the iris and the cicatricial tissue, minute intervals remaining between the two. Hence, corneal fistulæ do not as a rule present wide canals lined with epithelium, and yet the scar-tissue filling the perforation opening is not dense, but is permeated by fissures through which the aqueous keeps oozing until it reaches the exterior surface of the cornea (Czermak). The origin of other fistulæ is that the prolapsed iris splits apart under the strain produced by the pressure of the aqueous, and the opening thus formed never closes solidly again. Lastly, in very extensive prolapses of the iris, it may happen that in the process of cicatrization a fistula is left at the spot corresponding to the pupil.

203. Other bad consequences of perforation of the cornea, which are sometimes observed, are:

(*a*) *Luxation of the Lens.*—After the escape of the aqueous humor, the lens, to reach the cornea, must be pushed forward through the entire depth of the anterior chamber (2.5 mm.), a movement associated with a considerable degree of stretching of the zonula of Zinn. If the forward movement

takes place very suddenly, or if the fibers of the zonula have been rendered fragile by disease, the zonula ruptures. In consequence the lens may become tilted, or, if the perforation is large enough may even be expelled from the eye.

(*b*) *Intra-ocular hæmorrhages* are the result of the rapid diminution in tension, by virtue of which an increased quantity of blood flows into the vessels of the interior of the eye, which have been thus suddenly relieved of external pressure, and causes their rupture. Hæmorrhage takes place if the perforation occurs very suddenly, or if the eye beforehand was under an abnormally high tension. The latter is the case in glaucomatous and staphylomatous eyes, in which, furthermore, there is generally also a degeneration of the vessels associated with an increased fragility of their walls. The hæmorrhage may be so great that the entire contents of the eyeball are extruded by reason of it; nay, more, the patient may almost be in danger of bleeding to death.

(*c*) Suppuration starting from the cornea may be carried over into the deeper parts of the eye and lead to the destruction of the latter by *severe irido-cyclitis*, or even by *panophthalmitis*. This occurs mainly in the case of extensive destruction of the cornea, especially if, at the same time, the suppuration is of a particularly virulent character, as in gonorrhœal conjunctivitis or in ulcus serpens.

204. The Clearing of Corneal Cicatrices.—After a long time has elapsed—months or years, that is—the cicatrix left by an ulcer appears less large and less opaque than it was directly after the healing of the ulcer was completed; the cicatrix has partially "cleared up." In this way quite small cicatrices may become altogether invisible. The extent to which clearing takes place depends principally upon two circumstances: upon the thickness of the cicatricial tissue and upon the age of the individual. The deeper the cicatrix penetrates into the cornea, the less it clears up; perforating cicatrices of the cornea, if they are ever so small, remain permanently opaque. (A fine example of this is afforded by the punctures which the discission needle makes, and which remain visible as gray points upon the cornea all during life.) The age of the individual influences the process of clearing, in that the latter makes greater advances the younger the patient is. For this reason cicatrices after ophthalmia neonatorum often clear up in a wonderful way.

205. Etiology.—With reference to their etiology, all inflammations of the cornea may be divided into two great groups; primary and secondary keratitides. By the former, we understand those which have their starting point in the cornea itself; by the latter, on the contrary, those which have passed over to the cornea from other structures, and most frequently from the conjunctiva. This distinction, true with regard to keratitis in general, is especially so with respect to ulcers of the cornea.

Primary ulcers of the cornea most frequently owe their development

to traumatism. Under this head belong not only injuries in the narrower sense of the word, but also a lesion of the cornea by means of small foreign bodies, by faultily placed cilia, by papillary growths on the free border of the lids, etc. Ulcers, furthermore, develop after the separation of eschars produced by burns or the action of caustics. Other ulcers are dependent upon a disturbance in the nutrition of the cornea, as the ulcers in eyes with absolute glaucoma where the cornea has become insensitive, or ulcers which develop in old cicatrices of the cornea (atheromatous ulcers).

Secondary ulcers are the result of an affection of the conjunctiva. All inflammations of the conjunctiva may be complicated with inflammation of the cornea; and in severe inflammations of the conjunctiva, as in gonorrhœal conjunctivitis and diphtheria, this is quite the rule.

In accordance with our present views regarding suppuration we must expect to find that in the majority of cases the direct cause of the formation of ulcers in the cornea is constituted by the entrance of micro-organisms into the corneal tissues. Micro-organisms are found very frequently in the secretion of the diseased and even of the normal conjunctiva. If, now, owing to slight traumatism, to detachment of the epithelium by the formation of a vesicle (in herpes corneæ), or to any other cause, the protective epithelial covering of the cornea is injured at some spot, the door is opened for the entrance of bacteria into the tissue of the cornea. In people of the working class ulcers of the cornea occur much more frequently than among the well-to-do classes, because they very often suffer from neglected chronic catarrhs, and also very often expose themselves to injuries of the cornea.

Ulcers of the cornea are among the most frequent affections of the eyes, and special significance attaches to them because the opacities that they leave very often impair the sight. Ulcers of the cornea, if we except those resulting from conjunctivitis eczematosa, are found much more frequently in adults, and especially in elderly people, than in children. It seems that in the later years of life the cornea is less well nourished, and is hence more disposed to disintegrate than in youth.

The results of *bacteriological examination* in some cases of corneal ulceration are negative; in other cases we find the same bacteria that are familiar to us as occurring in the conjunctival sac. This should not surprise us, since the cornea certainly is most often infected by invasion from the conjunctival sac. A perfect correspondence between the individual variety of corneal ulcer present and a definite morbid germ, so that from the appearance of the ulcer one could diagnosticate the germ with certainty, exists only for the keratitis produced by mold fungi and to a less extent for ulcus serpens, which, at least in the majority of cases, is caused by the pneumococcus. [The pneumococcus, however, may also cause ordinary ulcers of the cornea and particularly the clear facetted ulcer (page 259), which is likewise produced by severe inflammation caused by the Koch-Weeks bacillus or mixed infections (Butler).—D.] Marginal ulcers such as occur in conjunctival catarrh are often produced by the diplobacillus of Morax-Axenfeld or the allied bacillus of Petit, sometimes also by a bacillus described by zur Nedden. [According to Butler, except in mixed infections, the Morax-Axenfeld diplobacillus is not very often a cause of corneal ulceration.—D.] In any event, the bacterial findings in the case of corneal ulcers vary greatly.

[Some ulcers are due to constitutional causes (see pages 260, 261 and 281); and in others that are not produced directly in this way the depressed general state of the patient often affects the course unfavorably and, unless attended to, may lead to destructive changes in the cornea. This is particularly the case with the ulceration of the cornea occurring in marasmic infants affected with ophthalmia neonatorum. Here ulceration may progress in spite of all local measures, and every effort must be made to reinforce the patient's resisting power by general treatment. Again, many believe that deficient nerve power is at the bottom of many cases of ulceration following vesicle formation (herpes of the cornea, recurrent erosion; see §§ 226–230 and 247), and in others hypothyroidism seems to be a factor.—D.]

206. Varieties.—Corneal ulcers occur under many various *forms*, some of which are well characterized, partly by their etiology, partly by their aspect and course. These may be enumerated as follows:

1. In *conjunctivitis eczematosa*, as well as in *conjunctivitis ex acne rosacea*, we find small, superficial, generally marginal ulcers, which, as a rule, get well rapidly. There are, however, cases of conjunctivitis eczematosa in which the ulcers, without spreading along the surface, keep on steadily penetrating deeper and deeper until abrupt crater-shaped losses of substance are produced which speedily perforate the cornea. These ulcers, too, are commonly situated at the margin of the cornea, and hence leave behind those peripheral incarcerations of the iris with marked displacement of the pupil that are characteristic of a conjunctivitis eczematosa which has run its course (Fig. 92).

2. The *vascular fasciculus* (keratitis fascicularis) is likewise observed in conjunctivitis eczematosa, and is produced by an ulcer making its way from the margin of the cornea farther and farther into the latter, and trailing after it a leash of vessels from the limbus (page 194).

3. *Catarrhal* ulcers are characterized by their crescentic shape, as well as by their position near the corneal margin, and concentric with it. Several crescentic ulcers may be present in the same eye at different parts of the circumference of the cornea; nay, more, by their confluence an annular ulcer may be formed, completely encircling the cornea. In the latter case the annular opacity which is left has a great resemblance to the arcus senilis corneæ. In cases where such an annular ulcer has penetrated deeply, the very detrimental result of a permanent ectasia of the cornea has been observed. For by a stretching of the floor of the ulcer the base of the cornea at the point where the ulcer is situated is pushed forward, and the whole cornea assumes an oblique position. If the ulcer completely surrounds the cornea, the latter may give way before the intraocular pressure and move forward en masse. In this case the region of the cornea inclosed by the annular ulcer lies like a watch glass on top of the marginal portions of the cornea (keratectasia ex ulcere, § 271). [Other forms of ulceration observed in catarrhal conjunctivitis are the clear facetted ulcer (see No. 5 below), minute deep ulcers like gimlet holes, and minute superficial ulcers in a circle midway between the center and the circumference of the cornea. These last occur specially in children and heal quickly (Butler).—D.]

4. In *pannus trachomatosus* small ulcers frequently occur, which develop from the infiltration at the margin of the pannus. Sometimes along the margin of the pannus a whole series of such ulcers is found, which also may coalesce into one large crescentic ulcer. Other ulcers develop in the midst of the pannus in spots where the infiltration penetrates more deeply into the cornea and leads to ulcerative disintegration.

5. The *central, non-irritative* ulcer occurring in trachoma [and also in Koch-Weeks, pneumococcus, and mixed infections] develops generally in the center of the cornea. It is distinguished by the absence of accompanying symptoms of irritation, so that

often the disturbance of vision is the only thing that calls the patient's attention to his trouble. Objectively, the ulcer is distinguished by the fact that even during the progressive period it is but very slightly clouded, so that it scarcely gives any evidence of its presence, except the loss of substance that it produces; it may therefore be very easily overlooked if we do not examine the cornea by carefully testing its reflex. It has a tendency to fill up incompletely with cicatricial tissue, so that a central facet remains which causes very great deterioration of sight by the production of irregular astigmatism.

6. Ulcers in *gonorrhœal conjunctivitis* and in *diphtheria of the conjunctiva* generally spread rapidly, and often lead to destruction of the entire cornea, or even to panophthalmitis. [Multiple superficial ulcers may also be caused by metastatic gonorrhœa (see page 156).—D.]

7. *Traumatic* ulcers of the cornea are, as a rule, small and superficial, and occur for the most part in elderly people. They are located in the zone of the cornea that lies in the palpebral fissure; the upper third of the cornea, which is covered by the

Fig. 94.—Forms of Ulcer that Develop from Herpes Febrilis Corneæ.

A.—Large but Quite Superficial Ulcer. The central gray ring corresponds to an earlier stage of the ulcer, which since then has advanced nearly to the margin of the cornea.
B.—Keratitis Dendritica. To the left of the large branched ulcer lies a group of delicate minute maculæ, representing the remains of the herpetic efflorescences.
C. Keratitis Stellata. In the inner half of the cornea, which is moderately clouded all over, are four large and two very small ulcers, showing a stellate branching.

upper lid, therefore generally is exempt from them. But besides these ulcers, which run a rapid course and are benign, there also occurs—usually after inconsiderable injuries—the dangerous sort known as ulcus serpens (see § 217).

For the ulcers which develop as a result of desiccation of the cornea, see keratitis e lagophthalmo (page 277).

8. *Small marginal* ulcers of the cornea occur frequently in elderly people, especially of the male sex, without any conjunctival lesion or external traumatism being discoverable as their cause. They develop with pretty violent complications, but are scarcely of the size of a pin's head, and heal rapidly without penetrating deeply. They are particularly troublesome from the fact that they are prone to recur, so that many people have to go through with attacks of this kind of keratitis one or more times every year. The uratic diathesis appears to be a frequent cause of these ulcers, and general treatment directed against this diathesis and consisting of the proper dietetic regulations or the use of mineral waters often puts an end to the recurrence of the ulceration.

9. *Herpes corneæ febrilis* (rarely also *herpes corneæ zoster*, see § 228) may give rise to ulcers formed from the ruptured herpetic vesicles. These have the property of not penetrating deeply, but of being very prone to spread superficially. This superficial extension may take place in two ways; either the ulcer extends in all directions uniformly, in which case we have a large but quite superficial loss of substance everywhere surrounded by a narrow, sharp, usually festooned, gray, and infiltrated margin, which

pushes its way farther every day (Fig. 94 A); or extension takes place in certain directions only. In the latter case, from the loss of substance, which originally is small, gray striæ extend in one or more directions into the transparent cornea, and grow constantly longer, at the same time becoming forked, and also sending out lateral branches. Thus there is produced in the cornea a very pretty gray figure which is branched like a tree, and often bears nodular swellings at the extremities of its branches —*keratitis dendritica* (Emmert). This branched infiltrate breaks down into an ulcer having the form of a deep, branched furrow, with gray margins inclosing it (Fig. 94 B). Then this ulcer becomes clean and heals, leaving behind it an opacity, whose branched form allows us to recognize, even some time afterward, the nature of the antecedent affection.

[Dendritic keratitis was first described by Kipp, who pointed out that in this country at least it is caused in nine cases out of ten by malaria, and is relieved by the administration of quinine. The same is true of some cases of the ordinary form of herpes febrilis corneæ (De Beck).—D.]

In many cases of herpes, instead of a single large ulcer, numerous minute ulcers develop which are star-shaped and provided with short processes (*keratitis stellata;* Fig. 94 C).

All these forms of keratitis are characterized by long duration (one to three months).

[10. Akin to the above conditions is *superficial linear keratitis* (Spicer and Greeves). This is characterized by an acute onset with pain, congestion, and the formation in the cornea of a number of gray superficial epithelial ridges, usually running vertically and tapering at the ends. The ridges present a series of denser spots or nodes, which often stain with fluorescein, although the ridges themselves do not. The tension is usually markedly lowered, and the sight greatly impaired. The attack subsides in a few days or one or two weeks, leaving a grayish opacity, which at length disappears. If, however as often happens, there are several attacks, a permanent opacity is left which may render the eye partially or completely blind. The ridges on the cornea differ from those of dendritic keratitis in that they are multiple, straight, and have no bud-like outgrowths from them. They are caused by a wrinkling and elevation in Bowman's membrane with the formation of new fibrous tissue in the underlying substantia propria.—D.]

11. *Ulcus rodens* (Mooren). [*Chronic serpiginous ulcer* (Mayou).] A superficial ulcer develops from the margin of the cornea (usually the upper margin) with marked inflammatory complications. From the sound portion of the cornea it is limited by a gray, clouded margin, which is evidently undermined. This latter symptom is characteristic of ulcus rodens. After a short time the ulcer begins to grow clean and to cicatrize, becoming covered with vessels from the limbus. Just when one supposes the process to be nearing complete recovery, a relapse sets in with a return of the symptoms of irritation, and in this the ulcer pushes its way forward somewhat farther in the cornea. So the disease goes on with discontinuous attacks and intervening remissions, until the ulcer has covered the entire cornea. The latter is thus everywhere deprived of its superficial layers, and hence remains permanently clouded throughout its entire extent, so that vision is very greatly diminished. [The ulcerated area, as Mayou remarks, shows but little tendency to fill up with new tissue and may show very little clouding.] Perforation of the cornea in this affection has never been observed. This rare disease attacks elderly people, and not infrequently invades both corneæ either simultaneously or in succession. It was regarded as incurable as long as surgeons were unacquainted with the cauterization of the cornea by means of the actual cautery. If, however, we destroy the margin of the ulcer by this means, the ulcer in most cases is brought to a cure.

12. *Keratitis marginalis superficialis* is another rare disease, found in persons in middle life. A quite superficial ulceration spreads over the cornea, starting from its

margin. It does not, however, start from all parts of the margin at the same time, nor does it advance uniformly. Consequently the ulcerated marginal zone of the cornea is demarcated from the transparent central portion by a sinuous border formed by a fine gray line (Fig. 95). This variety of keratitis drags on for a long time—sometimes for years—periods of intermission alternating with relapses, which are associated with moderate symptoms of irritation. It is distinguished from ulcus rodens in that the ulcer is extremely shallow, and hence the cornea within its limits shows only a very faint and grayish opacity. Moreover, the edge of the ulcer, scarcely visible in any case, is not undermined. The ulceration never reaches the center of the cornea, so that the extremely faint opacities that remain do not interfere with sight. Keratitis marginalis superficialis often gives rise to a drawing of the conjunctiva up over the cornea in the form of a pseudo-pterygium (see page 217).

The vascular fasciculus, keratitis dendritica, ulcus rodens, and keratitis marginalis superficialis have the common trait of creeping along slowly in the cornea, for which reason they are also designated by the name of serpiginous ulcers of the cornea.

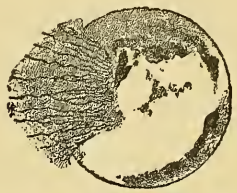

Fig. 95.—Keratitis Marginalis Superficialis.
At the inner margin of the cornea is a pseudo-pterygium.

13. *Atheromatous* ulcers develop in old scars of the cornea when the latter have undergone degeneration through the deposition of lime or hyaline masses or when they are exposed to mechanical injuries (as, for example, when at the apex of a corneal staphyloma). The result is exfoliation of the epithelium and consequently necrosis of the poorly nourished cicatricial tissue ("sequestrating cicatricial keratitis"). As the necrosis frequently extends through the entire thickness of the scar, perforation often occurs and as a sequel to this even panophthalmitis.

14. In eyes rendered blind by *glaucoma absolutum*, purulent ulcers, usually under the form of ulcus serpens, may develop. These are ordinarily associated with considerable hypopyon, and frequently terminate either in perforation of the cornea, with resultant hæmorrhages from the eyeball, or in panophthalmitis. Like atheromatous ulcers, they are caused by insufficient nutrition and innervation of the cornea, an insufficiency already made apparent from the insensitiveness of the latter. With both varieties of ulcers, enucleation of the blinded eye is sometimes the only remedy that permanently relieves the patient of the repeatedly recurring, tormenting ulceration.

15. Likewise due to impaired nutrition are the *marantic ulcers* which occur in persons who are run down as a result of a serious disease of the liver (cirrhosis, carcinoma). These ulcers are torpid in course but may, nevertheless, produce destruction of the cornea. As they are associated with xerosis of the conjunctiva and with hemeralopia, they occupy an intermediate position between the cases of simple hemeralopia with xerosis (§ 569) and the keratomalacia of small children (see page 279).

207. Treatment.—Ulcers of the cornea are very amenable to proper and energetic treatment. They hence in general afford a favorable prognosis if they come under treatment early; in the great majority of cases it is possible to put a stop to their progress, and produce regular cicatrization. The treatment varies according to the stage in which the ulcer comes under treatment.

(*a*) Recent ulcers that are still *foul* (progressive) require, most of all, the consideration of the *causal indication*. In traumatic ulcers any foreign bodies that are still present must in every case be removed. Cilia which

are directed against the cornea must be epilated; papillomata of the edges of the lids, when causing trouble in the cornea, must be removed. In the numerous cases in which the ulcer of the cornea is caused by a conjunctival lesion, the treatment of the latter forms, as a rule, the most important part of our therapeutics, and under it, moreover, the ulcer advances toward recovery. Hence, in corneal ulcers resulting from catarrh, trachoma, or gonorrhœal inflammation of the conjunctiva, we must by no means desist from cauterization of the latter, if it is required by the conjunctival trouble. [So too, in cases of diplobacillus conjunctivitis, we continue using zinc sulphate (see page 145) after ulcers have developed, and with good effect on the latter.—D.] The only precaution that must be observed is that the caustics applied should not come into contact with the cornea itself—a contingency which can be avoided by carefully washing off from the conjunctiva any excess that may be present. Furthermore, we should cauterize with the silver solution only, and not with the copper stick, which is too irritating. Moreover, as long as progressive ulcers are present in the cornea, no irritating collyria, such as the collyrium adstringens luteum and the like, should be instilled, as they would then come into contact with the cornea.

[To fulfill the causal indication, we must in many cases apply constitutional and hygienic treatment. Thus, if the disease has a malarial basis as in many cases of dendritic keratitis, we must use quinine; in herpetic and other cases in which there is evidence of nerve disturbance, we use arsenic (which, indeed, seems helpful in a variety of corneal conditions). In some intractable forms thyroid extract may be required, although this is more often indicated in non-suppurative keratitis. Fresh air and good diet, careful regulation of the bowels, and, in general, measures to promote nutrition and prevent accumulation of waste matter are indicated in all cases, but particularly in the marantic ulcers (page 261), the uratic ulcers (page 259), and the ulcers of eczematous conjunctivitis. In the last-named condition minute doses of calomel are of service (see page 201). Finally we should look for and remove any latent focus of infection in the teeth, tonsils, nasal cavities, etc.—D.]

208. The *indicatio morbi* requires in most cases the application of a *protective dressing* (see page 54). This, as a rule, is to be kept on until the ulcer gets clean and becomes lined with an epithelial covering which protects the cornea against exterior influences. When the floor of the ulcer is thinned and shows a tendency to bulge, a *pressure dressing* must be applied and kept up until the freshly formed cicatrix is sufficiently strong to offer resistance to the intra-ocular pressure.

A contraindication against the bandage is furnished by a profuse secretion, because the latter would be retained in the conjunctival sac by the closure of the lids, and would remain in constant contact with the ulcer. For this reason, in ulcers resulting from conjunctivitis the bandage must be very often dispensed with. In quite small children, also, the bandage is generally useless, since it soon gets displaced; and a badly applied bandage is more hurtful than any exposure of the eye could be.

Next to the bandage *atropine* plays the most important part in the treatment of ulcers. It combats the inflammation of the iris, hence diminishes the general state of irritation, and so reacts favorably upon the ulcer itself. It must be instilled as often as is necessary, in order to keep the pupil steadily dilated.

[In cases that promise to be transient, atropine may tentatively be replaced by homatropine.—D.]

209. With these two remedies alone—the bandage and atropine—we attain our object in mild cases. For those ulcers, however, which, from the purulent hue or from the great infiltration of their surrounding parts, show a rapidly *progressive* character, we must employ still other remedies. Some of those most used are—

Moist hot compresses (see page 56) applied every day for an hour or more, the dressing being left off each time for the same period.

Very finely powdered *iodoform* sprinkled upon the ulcer or applied in 10-per-cent ointment.

Absolute *alcohol* or tincture of *iodine* [or a solution of iodine in vasogen applied to the ulcer with a cotton-tipped swab. This very effective, though very painful application can be used with considerable freedom, as it rarely does injury to the intact cornea.—D.]

[*Nitric acid* or 95-per-cent *carbolic acid*. To apply the former we use a wooden toothpick soaked in the acid and then dried (Jackson). We must be careful that neither agent comes into contact with the sound cornea.—D.]

[In phlyctænular and other superficial ulcers, powdered *calomel* dusted into the eye or in the latter stages 1-per-cent *yellow oxide of mercury ointment* (see page 200).—D.]

Subconjunctival injections (see page 65) of a mercury oxycyanide solution or a 10-per-cent salt solution made beneath the bulbar conjunctiva.

[In some cases *phototherapy* (page 57), *actinotherapy*, or *iontophoresis* (page 58) with zinc sulphate have been serviceable.—D.]

The minute branched ulcers of keratitis dendritica are usually arrested if a piece of bluestone whittled to a fine point is carried carefully along the furrow made by the ulcer. [But these cases usually require constitutional treatment (see page 260).—D.]

210. If in spite of these remedies the ulcer is evidently spreading, we must proceed to the operation of *cauterizing* the ulcer by means of the actual cautery (Gayet). For this purpose we use a small sharp-pointed cautery iron, or the galvano-cautery loop, or Paquelin's thermo-cautery. With one of these instruments the ulcer is cauterized wherever it shows a gray coating. In the case of extensive ulcers it is not necessary to cauterize the entire ulcerating surface, but it is sufficient to destroy the most infiltrated portion of its margin, at which an advance of the ulcerative process is to be expected. Before cauterizing we make the cornea insensitive by repeatedly instilling a 5-per-cent solution of cocaine hydrochloride.

The introduction of the actual cautery, which we owe principally to Gayet, was a great step forward in treatment; for the ulcers against which we were often powerless before, namely, those which are deeply infiltrated and rapidly progressive, are just the ones that are usually arrested at once by this means. The application of the cautery is painless when cocaine is employed, and does not, as might be supposed, cause any marked irritation of the eye. On the contrary, after its application the pain often ceases instantly, while the other symptoms of irritation abate. In private practice, when one has no other means to resort to, the head of a probe or of a knitting needle, heated red-hot, may be employed for cauterization. The chief thing is to make the cauterization extensive enough. Perforation of the thinned floor of the ulcer can, with care, be easily avoided; should it occur, however, it has no bad results beyond what the perforation itself gives rise to, since the hot point is cooled at once by the outpouring aqueous humor. An opacity remains permanently at the cauterized spot; but since we only cauterize such places as would otherwise go on to purulent disintegration, the final opacity is not greater than it would have been in any case.

211. Another potent remedy for combating rapidly spreading ulcers is *paracentesis* of the anterior chamber (for the method of its performance see § 847). People were led to do this operation by observing that ulcers after spontaneous perforation generally went on to rapid healing (page 252). In a similar manner, artificial perforation—i.e., puncture of the cornea—performed early prevents the extension of the ulcer and its threatened rupture. Why should we not wait until the ulcer spontaneously perforates the cornea? Because in the meantime the ulcer keeps enlarging superficially and thus would produce a more extensive opacity, and, furthermore, because after ulcerative perforation of the cornea a prolapse of the iris almost always develops, leading to the formation of an anterior synechia, while with a properly performed puncture this is not the case.

If perforation is imminent, and we prefer not to bring it about artificially by puncture, we take care to have the patient kept quiet—a thing best done by making him lie in bed, in order that the perforation may take place gradually, and that as little as possible of the iris may be driven into the opening.

[For *transplantation* of the conjunctiva in rapidly advancing ulcers, see § 823.]

212. (*b*) When *perforation* of the cornea has taken place treatment has to aim at the following objects: in the first place, that the iris shall not adhere to the cornea, or at least shall do so to the smallest possible extent; in the second place, that a firm and flat (not ectatic) cicatrix shall be formed. The two objects are attained as follows:

1. If the perforation is quite small the iris does not prolapse into it, but simply becomes applied to its posterior orifice. In such cases, rest, bandage, and atropine suffice for the treatment. There then remains only a punctiform adhesion of the iris to the corneal cicatrix, and this is often subsequently drawn out into a thin filament. In particularly favorable cases no anterior synechia at all may be produced; for instance, if the iris,

before it has become firmly adherent to the site of perforation, is pushed away from it again by the reaccumulating aqueous.

2. If—in the case of a perforation of greater size—the iris has prolapsed, it should be excised. A replacement of the iris into the anterior chamber (reposition) in most cases would be impossible of performance, and even if it should succeed, would have no lasting results, since the iris would continually prolapse again. For the performance of *excision* we first make the cornea insensitive by means of cocaine. Then with a sharp-pointed instrument (conical sound) we separate on all sides the adhesions of the prolapse to the perforation opening, draw the iris as far as possible out of the wound with the forceps, and snip it off close to the cornea (Leber). If the iris still projects anywhere into the opening, it must be freed with a spatula and pushed back into the anterior chamber. If the operation has been successful, the iris ought no longer to be attached anywhere to the margin of the aperture; on the contrary, there should be a coloboma of the iris (Fig. 93) with free pillars, as after a regular iridectomy. In this way we obtain a firm cicatrix without inclusion of the iris.

The performance of excision is possible only in recent prolapses (prolapses a few days old), as afterward the prolapsed iris becomes so solidly adherent to the margins of the perforation that the separation of the iris from the latter is no longer feasible. Similarly it is not to be recommended in the case of a very large perforation. In these two cases—i.e.,

3. In old or very extensive prolapses of the iris, we abstain from releasing the iris from the cornea; we confine ourselves to the attempt to transform the prolapse into a firm and flat cicatrix. In many cases a pressure bandage applied for a long time accomplishes this end. If we cannot attain our object in this way, as is particularly the case when the prolapse is constricted at its base like a mushroom, we must produce flattening of the prolapse by repeatedly puncturing it or by excising a small portion. In the case of a very bulging total prolapse of the iris, it is advisable to split it transversely, and then, after opening the anterior capsule, to expel the lens. If there is a remnant of sound cornea left broad enough for the performance of an iridectomy, this operation is an excellent means for securing the formation of a flat cicatrix.

4. In *keratocele,* the maintenance of rest, the application of a bandage, and ultimately the puncture or cauterization of the protruding vesicle, are employed.

5. In *fistula of the cornea,* in order to effect its closure, everything must be avoided that might temporarily increase the ocular tension, and thus force the fistula open again just as it is closing. For this purpose we order rest in bed, with the application of a light bandage to both eyes, while at the same time we instill a miotic (eserine or pilocarpine) in order to diminish the pressure in the anterior chamber. An iridectomy has a very good effect,

but this can be performed only when the anterior chamber has been, at least to some extent, temporarily restored. If these measures fail, we must remove the cicatricial margins of the fistula either by excising or by cauterizing them (with a blunt galvano-cautery or thermo-cautery point) so as to fill the fistula up with new, firm scar tissue. Here again, however, if we are going to perform cauterization, there must be some remnant, even though a shallow one, of the anterior chamber present, as otherwise we should singe the anterior surface of the lens.

It is usually hard to effect firm union in fistulæ of the cornea. If the fistulæ will not close at all, we may often attain success by sewing over the fistula a flap taken from the adjacent conjunctiva. The flap by growing fast to the surface of the scar, whose epithelium had previously been removed, closes the fistulous opening. Another procedure consists in excising by means of the corneal trephine the fistula, together with the cicatricial tissue surrounding it, and implanting in the opening an equally large piece of healthy cornea (see § 851).

213. (*c*) The treatment of ulcers in the regressive period, or *period of cicatrization*, should aim at filling the loss of substance completely with a resistant cicatrix, and at rendering the latter as transparent as possible. For the attainment of both objects irritants are employed. We begin cautiously with the weaker remedies, passing gradually, if these are well borne, to the stronger ones. One of the mildest of irritants is powdered calomel; more energetic is the action of the yellow-precipitate ointment (from 1- to 4-per cent), the collyrium adstringens luteum,[9] tinctura opii crocata, and dionin. [Calomel and the yellow ointment can also be used with advantage in the earlier stages of corneal ulceration (see page 200).—D.] In applying the yellow-precipitate ointment, we insert it into the conjunctival sac with a brush or glass rod, and then by rubbing it about with the upper lid perform a sort of massage upon the clouded cornea. Another irritant remedy that is recommended is the application of hot steam (vaporization) or hot air (see page 56). It is advisable to continue the application of these irritants for a long time in order to secure the greatest possible clearing up of the cornea, but in so doing the remedies must be changed from time to time, as otherwise the eye gets accustomed to them and they lose their efficacy.

2. *Ulcus Serpens Corneæ*[10]

214. Symptoms.—A recent ulcus serpens appears under the form of a grayish-white or yellowish disk, which occupies nearly the center of the cornea. The opacity of the disk is greater at its edges than in the center, and generally the edges themselves show a particularly well-marked gray or yellow opacity in one special direction. The disk is surrounded by a delicate gray area, and frequently fine, radiating, gray striæ extend from the margin

[9] [See page 150.]
[10] Synonyms for ulcus serpens (Saemisch) are hypopyon keratitis (Roser) and abscess of the cornea.

of the disk into the transparent part of the cornea. The surface of the cornea over the disk is dotted, and often at the beginning is raised some distance above the level of the surrounding parts. Soon, however, this spot is seen to be depressed, although not with abruptly depressed edges, as in the case of an ulcer, but rather under the form of a shallow dimpling. Moreover, the rest of the cornea that is not occupied by the serpent ulcer proper is less lustrous, being covered with a delicate uniform cloudiness. These changes in the cornea are always associated with a violent iritis. The aqueous humor is turbid, a hypopyon lies at the bottom of the anterior chamber, the iris is discolored and is fastened by posterior synechiæ to the lens capsule. Corresponding to the severity of the inflammation is the violence of the irritative symptoms: slight œdema of the lids, intense injection of the conjunctival and ciliary vessels, photophobia, and pain, which latter often reach a very considerable height. Nevertheless there are also torpid cases, which are associated with very slight symptoms of irritation.

215. The subsequent *course* consists in the enlargement, both superficially and in depth, of the serpent ulcer. The superficial enlargement takes place chiefly in that direction in which the margin is marked by a specially dense opacity—an opacity which not infrequently looks like a yellow crescent placed upon the serpent ulcer. Inasmuch as the anterior lamellæ of the cornea within the region occupied by the ulcus serpens keep breaking down constantly more and more, there is formed an extensive ulcer, the floor of which is coated with pus. Soon after this, generally, those lamellæ of the cornea which now form the base of the ulcer are also destroyed, so that an extensive perforation of the cornea is produced. The contents of the anterior chamber, consisting of aqueous humor and pus, are discharged, and a large prolapse of the iris forms.

While the ulcus serpens is going through with its process of development, the accompanying iritis keeps on increasing in the same proportion up to the time of perforation of the cornea; the hypopyon, too, keeps growing until it fills the greater part of the anterior chamber, and the pupil is closed by an exudation membrane.

After the perforation of the cornea has taken place the irritative symptoms generally abate and the suppuration may now come to a standstill. In other cases, however, the purulent disintegration of the cornea keeps on just the same, so that the latter is entirely destroyed, with the exception of a narrow marginal rim. Panophthalmitis even may result from the suppuration passing over into the deep parts.

An ulcus serpens always leaves a very dense corneal cicatrix which cannot be cleared up, and in which the iris is almost always incarcerated. Furthermore, in consequence of the iritis, there are usually left adhesions of the iris to the capsule (posterior synechiæ), and even a closure of the pupil by a membrane (occlusio pupillæ). The corneal cicatrix itself is in favorable

cases flat, in unfavorable cases ectatic, so that the ulcus serpens ends by forming a staphyloma. If panophthalmitis has followed upon the ulcus serpens, a shriveling up of the eye (phthisis bulbi) takes place.

The clinical picture which is characteristic of ulcus serpens and by which the diagnosis is made is present only in the beginning of the disease. Its important features are *the disk-like form and central situation of the opacity, the more pronounced opacity of the margin in comparison with the center, the character of the corneal surface, which, at the site of the ulcus serpens, shows only a shallow depression, and finally the early onset of hypopyon and iritis.*

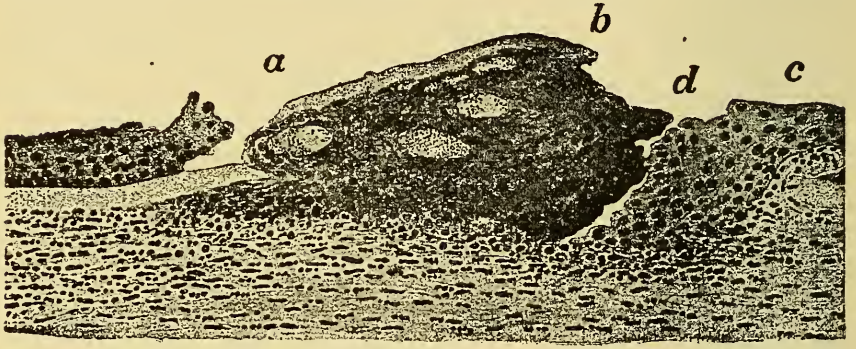

FIG. 96.—RECENT ULCUS SERPENS. Magnified 355 × 1.

An ulcus serpens developed from an erosion, the latter showing a faint gray cloudiness and, on the 3rd to the 4th day after it had started, getting a more pronounced gray border. Since such an early stage of ulcus serpens could not otherwise be had for anatomical examination, I inoculated the cornea of an eye that was destined for enucleation with a culture of pneumococci which had been made from a case of ulcus serpens. The eye was enucleated 3 days later; at the site of inoculation there was a gray infiltrate which rose somewhat above the level of the adjacent cornea and which had a more pronounced gray border like a recent ulcer. The cross section shows a plug consisting of the swollen uppermost layers of the cornea. In this lie only a few poorly stained (necrotic) pus corpuscles, and great quantities of pneumococci, which in particular quite fill up the large gaps visible in the plug. Bowman's membrane over the plug is also necrotic. At *a* the swollen masses push through the membrane, out upon the anterior surface of the latter, while the sharp edge of the plug juts forward like a wedge beneath the membrane. On the other side Bowman's membrane is wanting between *b* and *c*. Through this gap the epithelium *d* grows down into the deeper parts, and separates the necrotic plug sharply from the living corneal tissue. The lamellæ of the cornea at the lower side of the plug are infiltrated with pus corpuscles whose number increases toward the border *a*. At this spot both in the necrotic border itself, and in the surrounding infiltrated lamellæ of the cornea are present still living pneumococci. Hence at this side an extension of the suppuration would be expected to take place.

The *prognosis* of ulcus serpens is always serious, as, on account of the malignancy of its course, it belongs to the most dangerous of the diseases of the eye, and, if not checked early, it generally ends by producing blindness through an incurable opacity of the cornea. And even in the favorable cases, which either spontaneously or with the help of art come to a stop early, a dense, centrally situated opacity remains, so that usually the sight can be restored only by the performance of an operation (iridectomy).

216. Morbid Anatomy.—Anatomical researches on human eyes affected with ulcus serpens give the following results:

The pneumococci enter at the site of the superficial injury, then multiply, and cause a circumscribed necrosis of the cornea. About the necrotic plug (Fig. 96, *ab*) is found an infiltration with pus cells. The necrotic plug is approximately lenticular in shape,

its summit reaching to the surface of the cornea while its sharp edge pushes its way between the adjoining superficial layers of the cornea (Fig. 96 beneath *a*). Then the main body of the plug is exfoliated, so that a loss of substance is produced which does not look as deep as it really is (forms, indeed, simply a rippling depression), because the corneal lamellæ forming its floor are swollen. The border of the plug, extending between the lamellæ that are still preserved, or, in any event, the infiltration surrounding this border, remains, appearing in cross section as a wedge-shaped mass of cells, whose apex is directed toward the periphery of the cornea (Fig. 97, *a* and a_1). In it lie pneumococci which are still living, and hence the infiltration keeps insinuating itself farther and farther along between the lamellæ of the cornea and as fast as it does so it first lifts and then detaches the overlying layers. It is this marginal zone of infiltration that corresponds to the yellow, progressive border which is visible clinically. The contrast between this border and the floor of the ulcer is soon heightened by the fact that the purulent masses are thrown off from the floor, so that the latter is made up of lamellæ which are but little or not at all infiltrated, are swollen to form a homogeneous mass, and in distinction from the yellow border are but slightly cloudy.

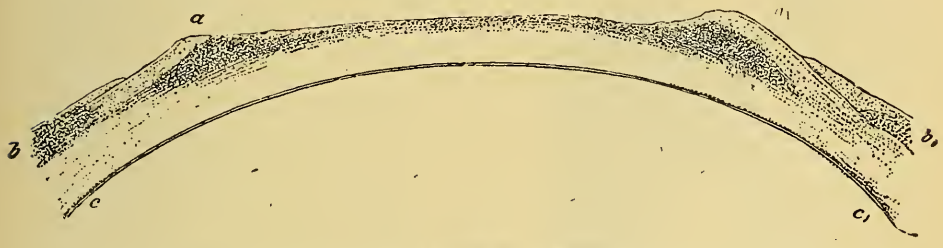

FIG. 97.—ULCUS SERPENS. Magnified 10 × 1.

The ulcer is large, is still covered with pus in every part, and has a yellow border *a* a_1, surrounding it on all sides. This border is elevated because the anterior lamellæ of the cornea are raised by the subjacent infiltration. Layers that are infiltrated with pus form the floor of the ulcer. The layers that succeed these deeper down show, to be sure, no infiltration, but neither do they show any living corneal corpuscles, so that the cornea here is necrotic in its entire thickness. At the margin of the cornea there is marked infiltration beneath the limbus *b* b_1, and under this lie living corneal lamellæ presenting a moderate infiltration which only towards the most posterior layers *c* c_1, becomes more marked. A posterior abscess is wanting in this case.

In many cases of ulcus serpens the infiltration of the margin soon disappears at some portion of its circumference, so that the ulcer advances in one direction only. This progressive portion of the ulcer's border then looks, not like a ring but a crescent (Fig. 98 A, *a*) applied to the disk-shaped ulcer, which itself is often so little clouded that one can scarcely recognize it except by the shallow depression that it produces in the surface of the cornea. In this case wherever in the living eye the yellow margin is no longer visible anatomical dissection shows the wedge-shaped infiltrate to be absent (Fig. 98 C, *b*). At this point the epithelium passes over the edge of the ulcer and out upon its floor, covering the latter in an irregular uneven layer, often as far as the advancing portion of the border. This fact explains why such ulcers give an almost mirror-like reflex. It would, however, be erroneous to assume that the portions of the cornea that have once more become covered in this way with epithelium have healed. Nature tries to cover every wound with epithelium as rapidly as possible, in order to protect it from the outside world. In such a case it often happens that the epithelium covers masses of dying tissue, and even masses of pus. So also in Fig. 98 C, we see at *a* how the epithelium has grown over the advancing portion of the border, which is on the very point of disintegration. Moreover, those lamellæ of the cornea that lie directly beneath the epithelium and form the floor of the ulcer are no longer capable of surviving; they are swollen up, destitute of corneal corpuscles, and contain simply a few pus

cells. The deeper-lying lamellæ are apparently normal; but upon careful examination it is found that no corneal corpuscles susceptible of staining can be distinguished in them, so that they also in large part are on the way to destruction. Hence it is that although in ulcus serpens the purulent infiltration does not go very deep, nevertheless a much more extensive perforation of the cornea occurs than we should expect from the intensity of the inflammation.

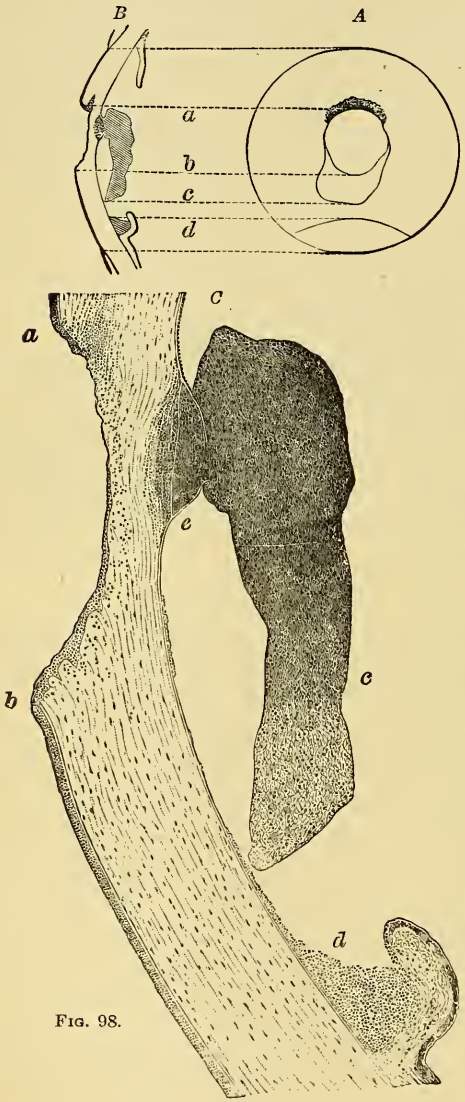

Fig. 98.

The changes which simultaneously take place at the *posterior surface of the cornea* contribute to the production of perforation. Here an accumulation of pus corpuscles takes place early, and these migrate toward the inflammatory deposit, making their way mainly along the posterior surface of Descemet's membrane. They are derived from the vessels of the uvea, and chiefly from the vessels surrounding the sinus of the anterior chamber; and as proof of the origin, many of them contain small granules of pigment derived from the uvea. The pus corpuscles congregate to form a mass of pus upon the posterior wall of the cornea; they then penetrate into Descemet's membrane and ultimately into the cornea itself. In this latter, therefore, there is formed a sort of posterior abscess (Fig. 98 C, *e*) at a point corresponding to the site of the ulcus serpens. Directly in front of this abscess lie those corneal lamellæ which, as already mentioned, are destitute of corneal corpuscles and are undergoing dissolution; and their necrosis in conjunction with the abscess gives rise to the perforation of the cornea.

Fig. 98.—Ulcus Serpens. A, front view; B, vertical cross section. Magnified 2.5 × 1.—C, longitudinal cross section. Magnified 20×1. In this, as in most of the cases of ulcus serpens that have been examined anatomically, the eye was affected with absolute glaucoma. The ulcer occupied about the center of the cornea; its upper, advancing border, *a*, was clearly recognizable as a yellow crescent, while the lower border, *b*, being but slightly opaque, did not specially stand out against the subjacent masses of pus which lay in the anterior chamber and extended as low down as *c*. Farther below, between the cornea and the iris, is seen the hypopyon, the upper border of which is convex (A, *d*). Owing to the glaucoma, the iris has, rather peculiarly, become adherent by its periphery to the cornea. *e*, posterior abscess in the cornea.

The pus of the posterior abscess, which lies within the cornea itself, is in direct communication with the purulent masses that are applied to the posterior surface of

the cornea. These latter form coherent lumps (*c*, Fig. 98 C), which sink to the bottom of the anterior chamber until they unite with the hypopyon (*d*, Fig. 98 C) present there.

The hypopyon has usually a border that in front view appears convex upward (Fig. 98 A, *d*). It is, moreover, agglutinated to the posterior surface of the cornea (Fig. 98 C, *d*), so that when we look into the anterior chamber from above we can see down between the hypopyon and the iris.

The older authors were well acquainted with these appearances, but gave them a different interpretation. They regarded the thread of pus extending down into the anterior chamber as a hypopyon situated in the cornea itself, assuming that the pus settled down between the corneal lamellæ. They explained the flattened shape of the hypopyon and the convex curve of its upper border as being due to the contracted space occupied by the pus inclosed between the corneal lamellæ. On account of the convexity of its upper border they compared the hypopyon to the lunule of the finger nail, and hence called it *unguis* or *onyx* (nail). These expressions would therefore denote a settling of pus down between the lamellæ of the cornea—a phenomenon, however, that does not actually occur.

In the clinical examination of an ulcus serpens it is only by careful investigation with the loupe or with a corneal microscope, and not always even then, that we can discriminate between that part of the gray or yellow disk which lies in the cornea itself and that which lies on the posterior wall of the cornea, that is, in the anterior chamber. If the infiltrations of the anterior layers of the cornea, which constitutes the ulcus proper, and the posterior abscess—in case one is present, which is not always the case—and the masses of pus on the posterior surface of the cornea, lie right behind each other, they in large part conceal one another. But often they are displaced laterally with regard to each other; the yellow pus on the posterior wall of the cornea projects on one side or even all around beyond the limits of the ulcer and makes the latter appear more extensive than it is. In cases in which a large part of the anterior chamber is filled with pus it is absolutely impossible to determine the limits of the ulcer with certainty, since the ulcer does not contrast with the yellow background; it is only after the pus has been withdrawn from the anterior chamber by splitting the cornea that the limits of the ulcer become evident. The faint gray circular line which not infrequently surrounds a small ulcus serpens at a certain distance from it is generally not located in the cornea itself but is the margin of a disk-shaped clot on the posterior wall of the cornea.

217. Etiology.—An ulcus serpens originates in infection of the cornea by organisms (the pneumococcus) which set up in it a purulent inflammation. Such infection presupposes two conditions: first, a lesion of the corneal epithelium, which in the normal state protects the cornea against the entrance of micro-organisms; and, second, the presence of pyogenic organisms which find their way to the spot where the epithelium is wanting. Both of these conditions occur in many cases of *injury* of the cornea. The body which inflicts the injury may itself be the carrier of infection and inoculate the cornea with germs. Much more frequently the injury, by producing a loss of substance in the epithelial covering, simply affords the opportunity for the entrance of infection, the infecting germs being furnished by the secretion contained in the conjunctival sac. The injuries which in this manner lead to the formation of the ulcus are, as a rule, very slight, consisting in a simple scaling off of the epithelium, such as is caused by a rough cloth, a leaf, or a branch grazing the cornea, small foreign bodies flying into the

eye, and scratches produced by contact with slivers of wood in those engaged in wood-chopping or fragments of stone in those engaged in breaking stone. Even in those cases in which a typical ulcus serpens has appeared to originate spontaneously, it is probable that there has been an antecedent injury, since such slight injuries of the cornea as these are readily overlooked by the patients. In exceptional cases severe perforating injuries, and likewise operation wounds, may also give rise to an ulcus serpens. Associated with the injury, and constituting the second factor in the production of ulcus serpens, is the presence of a chronic lesion of the conjunctiva (catarrh or trachoma), or an infection of the lachrymal sac (present in about one third of the cases of serpent ulcer), by which the infecting secretion is furnished.

Typical ulcus serpens attack adults exclusively, and especially those belonging to the working class. These are more frequently exposed to injuries of all kinds, and, besides, more often suffer from neglected affections of the conjunctiva and lachrymal sac than do members of the well-to-do classes. Great heat favors the formation of ulcus serpens, which is hence much more frequent in the hot season than in winter. For this reason reapers are not infrequently affected with the disease, since in cutting the grain they scratch their eyes with its awns, and, besides, do their work during the hottest days of the year. Stone masons [and coal miners] also are particularly apt to be attacked by ulcus serpens.

Ulcus serpens also occurs in *acute infectious diseases*, such as smallpox, scarlet fever, measles, typhus, etc. The form that results from *variola* is most frequently observed. In this case it makes its appearance not at the height of the disease but in the stage of desiccation, and, in fact, sometimes even in patients who have already left their beds. These variolous ulcers are found in children as well as in adults, and not infrequently affect both eyes so that total blindness may be produced by them.

Since the ulcus serpens in variola develops such a length of time after the stage of eruption, it obviously cannot be regarded as a smallpox pustule that has been localized upon the cornea. Such pustules do occur, but only in the conjunctiva. It is true they may then, if they lie near the margin of the cornea, give rise to a suppurative infiltration of the adjoining portions of the latter, but not to the typical picture of the centrally situated ulcus serpens. When the latter develops in the stage of desiccation of variola, it must be attributed, like a traumatic ulcus serpens, to an infection of the cornea from without. There is no lack of opportunity for such infection to take place, since the free border of the lids is a favorite seat for variolous pustules, which thus can come into direct contact with the cornea.

In typical ulcus serpens the pneumococcus is found so extremely often (Uhthoff and Axenfeld) that ulcus serpens has been called by the name of pneumococcus ulcer of the cornea. Exceptionally, it is true, other bacteria, above all the diplobacillus (that

of Morax-Axenfeld and a variety of it described by Petit), produce the clinical picture of ulcus serpens. That these germs found in ulcus serpens are really pathogenic for the cornea can be readily proved by making inoculations with them in the cornea of an animal. Traumatism alone, without infection, does not give rise to suppuration. We may cut, scrape, crush, or, in short, mechanically injure, or even cauterize the cornea of an animal without getting any purulent inflammation of it; in every case simply a gray cloudiness develops, which generally disappears again quickly. But when, by repeatedly brushing the conjunctiva with nitrate-of-silver solution, we have artificially produced a conjunctival catarrh, and in this way have given the opportunity for the production of infection, we then see purulent infiltration follow upon these same lesions of the cornea. What is true of the cornea of animals is also true of that of man. Provided we avoid infection by cleanliness and antiseptic measures, we can with impunity subject the cornea to operations both light and severe; even crushing of the cornea, such as, for example, is often enough produced in the expression of a cataract, does not always by any means lead to suppuration. But if we undertake the same operation in the presence of a conjunctival catarrh or a suppuration of the lachrymal sac, we risk the loss of the eye from a purulent infection of the wound.

In what way does infection of the cornea by pus germs lead to the *development of a suppurative keratitis?* We owe our knowledge in regard to these processes and the true explanation of them chiefly to the investigations of Leber, who made inoculations of various kinds of germs upon the corneæ of animals. The morbid processes that he observed to result from these inoculations he refers to the toxic effect which the products of the tissue metamorphosis of the germs induce. He assumes that the chemical substances produced by the germs exert upon the cell protoplasm an irritant action, when but slightly concentrated, and, when more concentrated, a paralyzing and ultimately fatal effect. When pus cocci are introduced into the cornea by inoculation they first increase in number within the corneal tissue. Then the cornea for a certain distance about the colony of cocci dies, because the toxic substances excreted by the cocci are present within this area in a state of strong concentration. Accordingly, the colony of cocci now lies in the center of the necrotic area (Fig. 99). In the meantime violent inflammatory symptoms have made their appearance in the eye. The toxic substances by diffusion have reached the margin of the cornea, and there cause dilatation of the vessels and increased permeability of the vessel walls, entailing as a necessary consequence increased diapedesis of the blood-plasma. In addition to this diapedesis of serum an emigration of white blood-corpuscles also takes place from the vessels. This is effected by active movements of the leucocytes, which, irritated by the toxic substances, emigrate toward the focus of inflammation (*chemotaxis*). This migration of the leucocytes may be explained in the following manner: The degree of concentration of the toxic substances diminishes gradually from the spot where the irritation originates to the periphery. Hence, that side of the body of a leucocyte that is turned toward the starting point of the irritation is in contact with a more irritating fluid than is the side which is turned in the opposite direction. Hence the protoplasmic processes push out more on the former side than on the latter, and the whole cell consequently moves toward the source of irritation. The leucocytes, however, do not make their way into the necrotic district itself, the pus cells that are found there being such as have

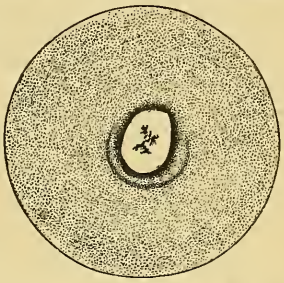

Fig. 99.—Inoculation Keratitis. (After Leber.) Magnified 3 × 1.

Surface section through a rabbit's cornea into the center of which a dilute suspension of staphylococcus aureus had been injected three days before. In the middle of the cornea is seen the mass of proliferating cocci, surrounded by a necrotic zone. This latter is bordered by a broad migration ring, adjoining which below there is a second one, narrower and not complete.

emigrated from the conjunctival sac. In fact, the leucocytes derived from the margin of the cornea are paralyzed at the border of the necrotic area owing to the great degree of concentration of the toxic substances at this spot. Thus it happens that a constantly increasing number of cells are arrested at the margin of the necrotic spot and die there. In this way is produced the infiltration (or migration) ring, which is apparent to the naked eye. Now leucocytes have the property of dissolving by a kind of digestive action tissues in which they are present in large quantities. They effect by this means the exfoliation of the necrotic area, and give rise to a delimiting suppuration. The inflammatory phenomena in the cornea, consequently, appear under the guise of a process having a definite purpose to subserve, the principal end and object of which are to eliminate the necrotic area, and with it the morbific agents that it contains. But besides this the pus corpuscles, as experiment has shown, have the additional property of directly inhibiting the growth of germs, so that they oppose the diffusion of those organisms that may have chanced to grow out beyond the necrotic mass.

Since the cornea is an organ which extends mainly in one plane, the migration zone does not form a spherical shell, but a ring. Yet, according to Leber, migration is not wanting on the posterior surface of the cornea also. The way in which this occurs is, that first the endothelium of Descemet's membrane over the necrotic area becomes detached and a clot of fibrin is precipitated from the aqueous upon this portion of the posterior wall of the cornea. Then leucocytes migrate into the clot, so that soon a plug of pus can be seen on the posterior surface of the cornea at the site of the inoculation. This pus by sinking to the bottom of the anterior chamber forms the hypopyon.

Leber's experiments were all made upon animals, in which it is not possible to produce a morbid picture perfectly similar to the ulcus serpens in man (see page 268).

It is a striking fact that typical ulcus serpens is observed only on adults. In children neglected disease of the conjunctiva and lachrymal sac, which affords the material for infection of the cornea, is rare, and probably, too, the cornea in childhood is less predisposed to infection with pneumococci. However, it is not quite immune to it. Rare cases of pneumococcus ulcers do occur in children. These ulcers accordingly correspond to the ulcus serpens of the adult, and like it take a malignant course. They do not, however, have the typical appearance of ulcus serpens, perhaps on account of the tendency to marked swelling that the cornea shows in childhood, for they form large infiltrates which often are so greatly distended as to project above the surface of the cornea. They are uniformly gray or are yellow like pus throughout, and rapidly break down.

218. Treatment.—In consideration of the rapid progress which an ulcus serpens usually makes, and which threatens the entire cornea with destruction, a particularly prompt and energetic interference is required. The treatment is partly medicinal, partly operative.

The *medicinal* treatment is the same as in pus-infiltrated ulcers of the cornea—namely, the application of a bandage, atropine, iodoform, moist and warm compresses, and subconjunctival injections of mercury [oxycyanide]. A specific remedy is optochin (ethyl-hydrocupreine hydrochloride), which has a specially destructive action on pneumococci and which is applied either by instilling a few drops of a 1-per-cent solution every hour or by painting the ulcer with a 2-per-cent solution. Painting the ulcer with a 20-per-cent zinc solution has also been advised. At the same time, any lesion of the conjunctiva or lachrymal sac that may happen to be present is to be suitably treated. This treatment is only adapted to the case of small recent

ulcers without an excessively large hypopyon. It should be undertaken only under the condition that the disease is closely watched, so that in case the latter progresses in spite of it, we may immediately proceed to operative treatment.

Operative treatment must be initiated without delay in all severe cases of ulcus serpens, but is also required in the milder cases when they resist the mild treatment. It consists either in the cauterization of the ulcer by means of the actual cautery or in its incision. *Cauterization* is performed in the same way as in the case of progressive ulcers of the cornea; special attention must be paid to the destruction of the progressive portion of the margin. Cauterization has the advantage over incision of not causing a perforation of the cornea, and hence of not giving rise to inclusion of the iris. It is suitable, however, only for those ulcers that have not yet undergone perforation, and in which the hypopyon is not excessively large, for the latter is not removed from the eye by this method; it can disappear from the anterior chamber only by resorption. *Incision* of ulcus serpens (§ 847), beside dividing freely the corneal lamellæ, which are saturated with pus, also effects the discharge of the hypopyon; it entails the disadvantage of a frequently extensive incarceration of the iris, which, however, would not have failed to occur without the operation, in those cases in which incision is indicated at all. Incision is suitable for very extensive ulcers, for those in which perforation is imminent, and for those which are associated with a deep hypopyon. We should not let the matter rest with a single performance of the incision, but must every day separate anew with a blunt instrument the edges of the wound, which speedily reunite, and we must keep this up until the ulcer begins to grow clean. At the same time that this operative procedure is being performed, the medicinal treatment above mentioned must be continued. Perforation and prolapse of the iris, when once they have occurred, must be treated according to the plan that has been laid down for perforating ulcers (page 264).

In ulcus serpens we may also *curette* the surface with a small sharp spoon. The loss of substance thus produced may be brushed over with antiseptic substances or with tincture of iodine. [Other methods that have been successful are *iontophoresis* with a ½-per-cent solution of zinc sulphate applied with a current of 2 M. A., and the injection of an *autogenous serum* derived from a blister raised on the patient's body (Römer).—D.]

Prophylaxis against the formation of an ulcus serpens is possible in the sense of our being able to remove in season the source of infection, as, for instance, the secretion from a diseased lachrymal sac. If, in such a case, a small erosion of the cornea exists, this is to be treated with special care by the application of disinfectant remedies.

In cases of *variolous ulcers* of the cornea, too, prophylaxis undertaken in season would often prevent the infliction of great injury. During an eruption of smallpox the lids are much swollen, and hence are not opened by the patient, and even the physician generally neglects to look at the eye from time to time. In that case, when the swelling of the lids goes down during the stage of desiccation and the patient opens

his eyes again, the morbid process in the cornea is often already in progress, and we are just so much behindhand in undertaking the treatment. Horner, therefore, is right in demanding that a physician treating a smallpox patient should prevent the agglutination of the lids by applying a pledget smeared with ointment, should examine the eyes every day, and should cleanse the conjunctival sac with antiseptic solutions. Careful watching will enable us to recognize the very commencement of the corneal disease, which in these early stages presents the most favorable conditions for treatment. At the time when smallpox was very widespread it formed one of the most frequent causes of blindness, so that about one third of all cases of blindness were produced by it. Since smallpox, owing to the introduction of vaccination, has become less prevalent, the blindness due to it has correspondingly diminished. Thus, in France, before the introduction of vaccination, 35 per cent—and after its introduction 7 per cent—of all the blind lost their eyesight by reason of smallpox (Carron du Villards). In Prussia, before the introduction of compulsory vaccination, 35 per cent—after its introduction 2 per cent—of all the blind people in the country were rendered so by smallpox.

219. Keratomycosis Aspergillina.—This variety of keratitis produced by mold fungi (Fig. 100) presents even upon external examination a clinical picture differing from that of the ordinary ulcus serpens. There forms in the central portion of the cornea an infiltrate which later undergoes superficial disintegration, and is distinguished by its peculiar, dry, crumbly surface. About this area a gray or yellow annular line of demarcation forms, which gradually deepens into a gutter and ultimately leads to the exfoliation of the inclosed portion of cornea, which in the meantime has become necrotic. The latter being thus detached en masse from the cornea, cicatrization of the resultant loss of substance ensues. Hypopyon is present, but the irritative symptoms are slight, and the whole course is very chronic. Examination of the sequestrum shows it to be permeated by the mycelium of the aspergillus fumigatus. It is probable that, as a general thing, this fungus is carried into the cornea by the foreign body that caused the original injury. [Occasionally other mold fungi are present besides the aspergillus. The inflammation produced by the latter is not always chronic and may be slight.—D.]

FIG. 100.—KERATOMYCOSIS ASPERGILLINA. Natural size. The prickly fruit of a horse chestnut had fallen into the eye of the patient, a woman thirty-five years of age, fourteen days before. The fungus mass is surrounded by a ring of demarcation but is still solidly connected with the underlying parts. Three small colonies of fungi have grown almost up to the ring of demarcation.

Keratitis Disciformis.—This is a keratitis that is allied to ulcus serpens and consists in the development in the middle layers of the cornea of a gray, disk-shaped opacity. In the center of the disk a small, more deeply clouded speck is commonly observed (Fig. 101). The periphery of the disk is sharply delimited by a border of deeper gray, which in many cases is made up of concentric lines (Fig. 102). This disk-shaped infiltrate never becomes yellow nor leads to disintegration of the cornea; only exceptionally a small loss of substance develops over a circumscribed area. The irritative symptoms are mostly not pronounced, and hypopyon is absent or scanty. The course is protracted, as it takes one or more months for the eye to become free from congestion and for the infiltrate to be transformed into a corneal opacity which is permanent. In the course of the disease scattered, superficial or deep-seated blood-vessels often develop and extend into the infiltrate. The cause of the disease, just as in ulcus serpens, is an infection of the cornea from without, the central grayer speck representing the point of entry of the bacteria. The epithelial lesion that precedes the infection may often be attributed to a slight injury and in some cases to an antecedent herpes corneæ febrilis; but in very many cases the cause remains unknown.

Schirmer has observed a typical keratitis disciformis when the cornea had been infected by vaccine virus (keratitis post-vaccinolosa). This occurs most frequently in

persons who in nursing children that have been recently vaccinated have acquired a vaccine blepharitis (§ 591), from which the cornea has become infected. These cases represent, we may say, a milder form of the ulcus serpens of variola and are instructive in two regards. They show that without doubt the ulcus serpens of variola does not originate, as was formerly supposed, in a metastatic way, but by infection from without, probably from the lid margin. They further prove that keratitis disciformis is closely related to ulcus serpens; is, we may say, a milder form of the latter, in which the inflammation does not go on to suppuration—a fact which probably depends on the slighter virulence of the micro-organisms that have entered the cornea.

Older authors, properly recognizing the relationship of keratitis disciformis to ulcus serpens (which they called abscessus corneæ), gave the former the name of abscessus siccus, to denote an abscess in which suppuration does not develop.

Keratitis disciformis has a certain resemblance to keratitis profunda (§ 242), in which also a deep-seated gray, non-ulcerating cloudiness develops in the center of the cornea. But this is composed of gray striæ and specks and merges gradually into the transparent cornea. In keratitis disciformis, on the contrary, the opacity appears uniformly gray and only on strong magnification is resolved into very minute clear-cut, white, closely packed dots; moreover, it is very sharply demarcated from the healthy cornea by the gray circular line along its border. Keratitis disciformis should not be confounded with keratitis centralis annularis (see § 235).

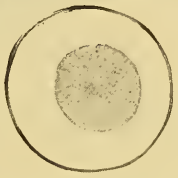

Fig. 101.

Fig. 102.

Fig. 101.—Keratitis Disciformis. Due to an erosion of the cornea.

Fig. 102.—Keratitis Disciformis. Due to herpes febrilis corneæ. The two maculæ at the inner lower margin of the cornea are on the site of a herpes eruption.

Annular Abscess of the Cornea.—This usually occurs after perforating injuries of the latter, and also after operations (especially cataract operations). No matter where the corneal wound that gives rise to it is situated, it develops in the central portions of the cornea as a yellow ring which is concentric with the limbus and is separated from it by a slightly cloudy marginal zone, 1 to 2 mm. broad. The ring itself has about the same width; the central portions of the cornea inclosed by it are again less cloudy and simply gray, not yellow. In the next few days, however, the yellow coloration spreads over the entire cornea; the latter disintegrates completely, and generally panophthalmitis ensues. Here accordingly we have to deal with an affection of the cornea of a peculiarly fulminating course, so that it is best to enucleate at once such an eye affected thus with annular abscess. Annular abscess may be caused by various bacteria; most often perhaps by the proteus fluorescens (Hanke) and the bacillus pyocyaneus.

3. *Keratitis e Lagophthalmo*

220. Keratitis e lagophthalmo originates in a defective covering of the cornea by the lids. The conjunctiva of the eyeball, wherever it lies constantly exposed to the air in the open palpebral fissure, appears reddened, and generally somewhat swollen as well. It secretes a small amount of discharge, drying upon the conjunctiva in crusts, which not infrequently also cover the exposed portion of the cornea. The latter is found to be dry on the surface, dull, slightly depressed, and at the same time clouded and gray. In the subsequent course of the disease the cloudiness becomes more and more intense, until finally disintegration of the superficial layers of the cor-

nea takes place, with the consequent formation of an ulcer (Fig. 103). At the same time there is iritis with hypopyon. The ulcer may heal without perforation, but leave an opacity behind it, or it may perforate the cornea, and thus lead to prolapse of the iris, or even to panophthalmitis.

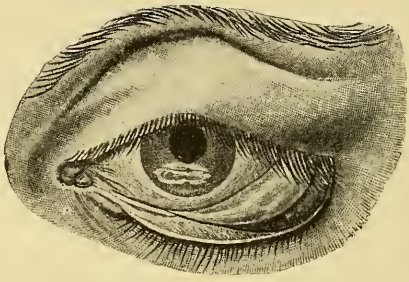

FIG. 103.—KERATITIS E LAGOPHTHALMO.

As a result of a healed caries of the upper and lower margins of the orbit, lagophthalmus had developed with ectropion of the lower lid. The skin of the upper lid, below the middle portion of the eyebrow, is drawn up toward the upper margin of the orbit and is attached to the latter by a scar one cm. long. As a result of this the free border of the lid is drawn up at its central portion, but, what is of most importance, the lid is prevented from going down when the attempt is made to close the eyes. There is a second scar at the outer end of the lower margin of the orbit. This, indeed, is concealed by the margin of the lid, but by palpation through the lid it can be made out that there is an irregular notching of the otherwise sharp and smooth orbital margin. At the same spot, the border of the lid is attached to the bone and in such a way that the entire lid is drawn outward and downward and everted, particularly in its outer half, so that here the conjunctiva of the tarsus, being exposed to the air, is particularly swollen and reddened, and consequently appears dark in the drawing. The lower lid, being fastened down, cannot be raised when the attempt is made to close the eyes. Hence, even in sleep the lower part of the cornea between the two lids remains uncovered. This part of the cornea presents an oblong ulcer with yellowish-white border and an excavated and hence somewhat darker center. Above and below, an areolate gray opacity adjoins the yellow margin of the ulcer.

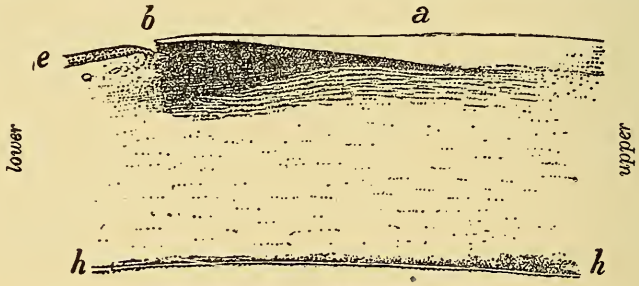

FIG. 104.—KERATITIS E LAGOPHTHALMO.

Vertical section through the lower part of the cornea. The epithelium, *e*, has been retained only in the neighborhood of the limbus, but over the dried portion of the cornea, *a*, it has been exfoliated. This portion of the cornea is necrotic, and no longer displays any stained cell-nuclei. Under it there is an infiltration of the cornea with pus corpuscles. This infiltration increases rapidly toward the lower margin of the cornea, here, at *b*, has already caused by liquefaction of the tissue a small loss of substance, and would later lead to elimination of the entire mass, *a*. Bellow *b*, a less marked infiltration extends to the lower margin of the cornea. This corresponds to the gray areola which adjoins the yellow border (Fig. 103). There is furthermore an infiltration of the cornea with pus cells on the posterior surface, *h h*. This at present is inconsiderable, but in the subsequent course would increase just like the infiltration on the anterior surface, and by growing out to meet this, would co-operate with it in producing disintegration of the cornea.

The *cause* of keratitis e lagophthalmo is the desiccation of the cornea in consequence of the defective closure of the lids (lagophthalmus). Owing to this desiccation the corneal epithelium becomes fissured and desquamates in spots. Then germs migrate into the corneal lamellæ thus exposed, and produce suppuration (Fig. 104).

The defective closure of the lids arises either from mechanical obstacles, such as contraction of the lids, marked protrusion of the eyeball, etc., or from paralysis of the orbicularis palpebrarum. In high degrees of lagophthalmus the cornea is uncovered all the time; in lighter cases, on the contrary, in which the closure of the lids is not impossible but only impeded, the danger of desiccation taking place is particularly present during sleep. In daytime, owing to the feeling of dryness of the cornea, the act of winking is pretty frequently excited through reflex action, and thus the cornea is repeatedly moistened. But in sleep the reflex winking of the lids is absent, and hence the cornea is unmoistened by this means and becomes dry whenever it lies exposed in the open palpebral fissure. The desiccation in this case always affects the lowermost part of the cornea, because in sleep the eyeball is turned upward, and hence the lower part of the cornea lies in the palpebral fissure. Here then an ulcer is produced which extends below as far as the margin of the cornea, while above it reaches a greater or less distance, according to the extent to which the cornea is uncovered, and ends in a horizontal border.

The same desiccation of the lowermost portion of the cornea occurs when the lids remain incompletely closed because the consciousness is clouded, as is the case in persons who, in severe diseases, lie unconscious for a long time. If such patients escape with their lives, they may have opacities of the cornea in both eyes in consequence of keratitis e lagophthalmo, or they may even lose their eyes altogether.

The *treatment* consists in taking care that the cornea shall be covered by the lids. In this way the development of a keratitis is prevented by prophylaxis, or, if a keratitis already exists, the chief condition is afforded for its cure. We must accordingly initiate the proper treatment for the cure of the lagophthalmus (see § 614), and in the meantime, until a cure has been accomplished, take pains to effect a perfect closure of the lids by means of a properly applied bandage. In slight cases of lagophthalmus it is sufficient to keep the eye bandaged through the night only. But if the lagophthalmus is considerable, or if keratitis has already set in, the eye must be kept bandaged all the time. If the treatment is initiated early, the prognosis is good, inasmuch as the process comes to a standstill as soon as the desiccation of the cornea is arrested.

4. *Keratomalacia.*

221. Symptoms and Course.—Keratomalacia[11] occurs only in childhood. The disease begins with night blindness (hemeralopia). This consists in the patient's visual power being perfectly good in bright daylight, but so very greatly reduced when the illumination is diminished (e.g., in twilight) that he is often no longer in a state to go about alone. In very small children who do not go about alone yet, this symptom naturally can-

[11] Softening of the cornea, from [κέρας, horn, and] μαλακός, soft.

not be made out. In such children, the first thing that strikes us is the dryness of the conjunctiva, which next develops, and which appears under the form of triangular xerotic spots on both sides of the cornea (see page 219). The conjunctiva in these spots is covered with a fine white substance like foam, and, as the lachrymal fluid cannot moisten it, looks as if smeared with grease. The dryness extends rapidly over the rest of the conjunctiva and also over the cornea. The latter becomes dull, insensitive, and uniformly cloudy. Soon the cloudiness in the center of the cornea increases, a gray infiltrate forming there. This spreads rapidly, takes on the yellow color of pus, and terminates in the disintegration of the cornea—a disintegration which, in bad cases, may take place within a few hours. In the beginning the affected eye is not discolored; afterward, when the cornea is already greatly involved, there appears about the latter a dusky venous injection. The lachrymal secretion is not increased, but rather diminished; moreover, other symptoms of irritation, like photophobia and blepharospasm, are slight or are wanting altogether. The striking contrast between the severity of the corneal affection and the insignificance of the accompanying symptoms of irritation, together with the dryness of the eye, stamps the disease with quite a peculiar character. This affection usually attacks both eyes.

Children suffering from keratomalacia show a disturbance of the general condition, which is generally pronounced even before the outbreak of the eye trouble, and which afterward grows still greater. The children become strikingly apathetic, have diarrhœa alternating with constipation, become rapidly emaciated, and often ultimately die either from exhaustion or from a complicating bronchitis or pneumonia.

The *prognosis* in very small children is bad, as in some cases they lose not only their eyes, but their lives as well. In somewhat older children the disease runs a less severe course, so that they escape with their lives and get off with smaller or larger cicatrices of the cornea.

222. Etiology.—Keratomalacia is the result of insufficient nutrition of the cornea. From this there results a necrosis either of the epithelium alone or of the corneal lamellæ themselves, and following this in turn is an entrance of bacteria from without and hence suppuration. The reduction in the nutrition of the cornea is only one of the symptoms of a serious disturbance of general nutrition which also shows itself in the associated hemeralopia. [The condition, in fact, is analogous to the much less serious idiopathic hemeralopia occurring in adults and due likewise to general disturbances of nutrition which diminish the sensitiveness of the retina to light (§ 569).—D.] Hence, keratomalacia develops, as a rule, in consequence of enfeebling influences affecting the children, and acting detrimentally upon their nutrition. Among these influences belong insufficient or unsuitable nourishment (rearing of children by hand), severe diseases like scarlet fever,

measles, typhus, etc., and particularly hereditary syphilis. The disease occurs in Russia much more frequently than with us, as there it attacks infants during and after the time of the great fast, because during this period the mothers lose their milk in consequence of fasting. For a similar reason it is frequently observed in Brazil among the badly nourished children of the negro slaves. In exceptional cases, children previously healthy are from some unknown cause attacked by keratomalacia, to which ensues a rapid decline of strength with a fatal issue. True keratomalacia does not occur in adults, although the kind of hemeralopia that occurs with xerosis of the conjunctiva and also the marantic ulcer of the cornea (see page 261), which likewise occurs chiefly in poorly nourished persons, may be a milder form of the same disease.

The chief task that *treatment* has to accomplish is to support the child's strength by means of fitting nourishment. In addition we must try to stimulate the vitality of the tissue of the cornea, a thing best performed by means of moist warm compresses placed upon the eyes. If the apathetic little patients do not close their lids properly, the corneæ must be protected from desiccation by bandaging the eye.

Rats fed on a chemically pure nutritive mixture and distilled water die, the cornea in the meantime becoming first xerotic and then undergoing suppuration. The illness of the animals is probably caused by the absence from their food of certain substances (vitamines) essential for life. Perhaps the keratomalacia of children depends on a similar dietetic error.

5. *Keratitis Neuroparalytica*

223. Symptoms.—In this affection, which occurs in consequence of paralysis of the trigeminus, the cornea becomes dull and slightly cloudy. Then, beginning in the center of the cornea, a depression shows itself in the epithelium as if the latter in that spot had been exfoliated. This depression spreads more and more peripherally until finally the only portion of epithelium that is left of normal thickness, is a strip 2 to 3 mm. broad at the margin of the cornea. This gives the cornea quite a peculiar appearance, such as is found in no other disease of it. In the meantime the cloudiness of the cornea also has increased. This is most marked in the center and there is uniformly gray; toward the margin it gradually decreases and may be resolved by the magnifying glass into separate maculæ. Subsequently the hue of the cloudiness becomes yellowish, hypopyon sets in, and ultimately the cornea in its center breaks down into pus. Thus a large ulcer forms, which cicatrizes with inclusion of the iris, and generally with flattening of the entire cornea. Not all cases, however, run so severe a course; the keratitis may get well without the occurrence of any purulent disintegration of the cornea, although there always remains an opacity of considerable dimensions, and often, besides, a flattening of the cornea.

The course of the disease is slow, and is characterized by the slightness

of the associated symptoms of irritation. There is, indeed, marked ciliary injection but no lachrymation, since the secretion of the lachrymal gland, due to reflex action, is diminished or abrogated. Owing to the coincident paralysis of the trigeminus, pain obviously is altogether absent.

The *prognosis* is unfavorable, treatment having very little influence on the course of the disease, which, whether the formation of ulcers does or does not take place, leads, almost without exception, to the production of a dense opacity over the entire cornea, and hence to an almost complete annihilation of the visual power.

Keratitis neuroparalytica has its *cause* in a paralysis of the trigeminal nerve, which induces trophic disturbances in the cornea. The paralysis of the trigeminus also causes the simultaneous arrest of secretion of the lachrymal gland as well as the absence of pain. The keratitis may set in no matter whether the lesion which causes the paralysis of the trigeminus affects the trunk of the nerve or its nucleus of origin in the brain.

The *treatment* consists in the application of a bandage, warm compresses, and atropine. Besides, we may try electricity, or, following Nieden's recommendation, strychnine (3 to 5 mg. by hypodermic injection beneath the skin of the temple).

224. Explanation of Neuroparalytic Keratitis and Allied Conditions.—The three affections of the cornea just described—keratitis e lagophthalmo, keratitis neuroparalytica, and keratomalacia—have been frequently confounded with each other. Thus the keratitis e lagophthalmo, which makes its appearance in patients during the death agony, has been regarded as a keratitis neuroparalytica, i. e., as caused by the reduction of the nervous energy. Conversely, some have explained keratitis neuroparalytica and keratomalacia as produced by desiccation of the cornea, and in this way have placed them in the same category with keratitis e lagophthalmo.

The theory of *keratitis neuroparalytica* was founded by Magendie, who found that after section of the trigeminus in animals a keratitis made its appearance. He referred this to trophic disturbances. Snellen and Senftleben explained it as being due to injuries of the cornea, and Feuer as being due to desiccation of the latter; the lesion in either case being the result of the insensitiveness of the cornea. Feuer's views have been shared by many authors, because in animals the movements of the lid are abolished and the cornea becomes dry on the side on which the trigeminus is divided. In opposition to this it is to be remarked that in this case animal experimentation is not conclusive in the case of man because:—(1) In man the reflex movements of the lids are always bilateral and hence take place as often on the side on which the trigeminus has been divided as on the other. (2) Neuroparalytic keratitis is observed even in cases in which either the eye has been protected by a bandage from the start (e. g., after extirpation of the Gasserian ganglion) or in which it has been kept permanently covered by reason of their being an associated ptosis. (3) The clinical picture of a genuine keratitis neuroparalytica is entirely different from that of keratitis e lagophthalmo.

The disturbance of nutrition in the cornea produced by a trigeminal paralysis makes itself first apparent in the corneal epithelium. If in a case of trigeminal paralysis in which the eye is still healthy, we hold the lids apart for one or two minutes with the fingers and thus prevent moistening of the cornea, small depressions, which soon enlarge and become confluent, appear in the epithelium. This takes place rapidly even when

the eye has been under a bandage up to the time the experiment is made, while a healthy eye does not show these pits at all or does so only after a long time. These pits prove a diminished resistance of the corneal epithelium to desiccation and consequently a disturbance of its nutrition which was present before the desiccation began. Diminished moistening is, therefore, certainly more injurious for such eyes than for normal ones, and in fact we often actually see a genuine keratitis e lagophthalmo in cases of trigeminal paralysis when the eye has been exposed more than ordinarily to desiccation. For the same reason, too, bandaging of the eye is indicated and often has a good effect even in neuroparalytic keratitis at the outset. But, as above stated, neuroparalytic keratitis may develop even when the cornea is kept permanently covered. Desiccation, therefore, is to be regarded only as a favoring factor, not as the final cause of keratitis neuroparalytica. Such a cause is found in a disturbance of nutrition of the cornea (and particularly of its epithelium) produced by the loss of innervation. As a result of this disturbance of nutrition the resisting power of the cornea is so reduced that the latter is thrown into a diseased condition by external influences which are so slight that a normal eye would not have been injured by them. (Similarly in hemiplegia a bedsore sometimes develops with extraordinary rapidity on the side which is paralyzed, and is absent on that which is not, although it is exposed to the same pressure as the other.) That keratitis does not develop in all cases of trigeminal paralysis is no proof of the contrary of what is here stated.

The confounding of the three forms of keratitis—keratitis e lagophthalmo, keratitis neuroparalytica, and keratomalacia—with each other has been favored by the fact that they display various features in common. Among these are the dryness which the eyes exhibit, and also the insignificance of the irritative symptoms in comparison with the severity of the keratitis, an insignificance shown in the absence of increased lachrymal secretion, of blepharospasm, and often also of pain. And yet the *dryness of the eyes* in these three forms of keratitis is to be referred to very different causes.

(*a*) In keratitis e lagophthalmo an actual desiccation of the cornea from evaporation exists. It affects only the exposed portion of the cornea, and may be relieved by closure of the lids. The desiccation in this case is the one cause of all the subsequent changes.

(*b*) In keratomalacia the cornea is not actually dry, but only looks so, because the lachrymal fluid does not adhere to its surface. This dry appearance is present even when the eye is swimming in tears or when it is kept constantly closed; evidently, bandaging is of no effect against this sort of dryness. It is caused by the fatty character of the epithelial surface, which consequently is not wetted by the lachrymal fluid.

(*c*) In keratitis neuroparalytica there is neither real desiccation of the cornea, as in keratitis e lagophthalmo, nor a peculiar fatty condition of its surface, as in keratomalacia; on the contrary, the eye looks dry simply because, in spite of the marked inflammation of the cornea, the lachrymation, which we usually see under these circumstances in other cases, is absent. The secretion of the lachrymal gland is in fact diminished or altogether abrogated; nevertheless, the moistening of the eye is quite sufficient, as indeed it is after extirpation of the lachrymal gland.

The *absence of marked symptoms of irritation*, which characterizes these three varieties of keratitis, is accounted for in the keratitis e lagophthalmo of very sick people and in keratomalacia by the general depression of the vital forces, and in keratitis neuroparalytica by the insensitiveness of the eye. The irritative symptoms, which in other cases are put in action through reflex impulses originating in the sensory nerves, are absent in the case of paralysis of the trigeminus.

The three forms of keratitis are hence, in spite of their external similarity, entirely different from each other, and can be readily differentiated by the clinical picture which they present. Keratitis e lagophthalmo occupies, as a rule, the lowermost part

of the cornea. Keratomalacia begins in the center of the cornea, and is found only in children who are the subjects of a rapid decline of nutrition. Finally, keratitis neuroparalytica is characterized above all by the rapid exfoliation of epithelium over the whole extent of the cornea, and does not occur except in conjunction with a trigeminal paralysis which can be diagnosticated at once.

The confusion between the three varieties of keratitis just described is furthermore favored by their nomenclature. The designation keratitis xerotica, chosen by Feuer for the keratitis of desiccation (keratitis e lagophthalmo), would be quite a good one if it did not lead to confusion with simple local xerosis of the cornea on the one hand and on the other with keratomalacia, in which xerosis of the conjunctiva and cornea likewise exists. And, as a matter of fact, some authors designate keratomalacia under the name of keratitis xerotica. In order to avoid this confusion, I have dropped the expression keratitis xerotica altogether; and as I do not wish to increase the number of epithets still further by the invention of a new name, I use the old expression keratitis e lagophthalmo for the keratitis of desiccation.

B. *NON-SUPPURATIVE KERATITIS*

(*a*) Superficial Forms

1. *Pannus*

225. Pannus consists in the new formation of a tissue resembling granulations beneath the epithelium of the cornea. Pannus is to be looked upon as an affection of the conjunctival layer of the cornea (conjunctiva corneæ, see page 136), and in every instance is simply one of the symptoms of a conjunctival disease—that is, either of conjunctivitis trachomatosa or conjunctivitis eczematosa. We hence make a distinction between pannus trachomatosus and pannus eczematosus. For further particulars, see under these two diseases of the conjunctiva.

2. *Keratitis with the Formation of Vesicles*

226. Vesicles on the cornea are generally small, and are filled with a limpid liquid. The anterior wall is very frail, for it is formed simply by the epithelium of the cornea, which is lifted up from Bowman's membrane by serum. More rarely larger-sized blebs (bullæ) occur, the anterior wall of which then generally consists of new-formed connective tissue (Fig. 105) in addition to the epithelium, and is hence more resistant. The small vesicles are ordinarily present in some numbers, while the large blebs generally occur singly. Violent symptoms of irritation, such as ciliary injection, lachrymation, photophobia, and more especially great pain, are usually present during the development of the vesicle. These, doubtless, are caused by the pulling upon the corneal nerves which pass into the epithelium, and which, in the process of formation of vesicles, are first stretched and finally torn in two. The irritative symptoms ordinarily disappear with the rupture of the vesicles. This occurs so quickly in the case of small vesicles that we generally do not get a sight of these themselves at all, but only of the subsequent small epithelial defects, to the margins of which the detached epi-

thelium still adheres in loose shreds. The large blebs, on account of the greater firmness of the anterior walls, are of longer duration. They are not tightly distended, but form a lax, tremulous, somewhat dependent sac. After their rupture the lax anterior wall still lies upon the cornea, and can be readily made out by the way in which it can be displaced by movements of the lids. The sensitiveness of the cornea to touch is ordinarily diminished or entirely abrogated in the cases with vesiculation.

There are the following varieties of keratitis with vesiculation:

227. (*a*) *Herpes*[12] *Febrilis Corneæ* (Horner).—In febrile diseases, especially of the respiratory organs (most frequently in epidemic influenza, next oftenest in bronchitis, pneumonia, ordinary influenza, etc.), less frequently in other febrile diseases, like typhoid fever, intermittent fever, etc., small vesicles often make their appearance on the lips, the alæ of the nose, the eyelids, the ears, etc.[13] At the same time an eruption of small transparent vesicles, which are associated with violent symptoms of irritation, may occur upon the cornea. These are scarcely the size of a pin's head, and are often disposed in rows or in groups. The vesicles very speedily rupture, leaving small abrasions, the floor of which shows a faint opacity. Generally these abrasions soon heal, so that after two or three weeks the disease is over, without leaving any lasting opacity of the cornea. In severe, and especially in neglected cases, however, large corneal ulcers, which not infrequently have a branching form (keratitis dendritica, see page 260), may develop from the small abrasions.

There is no doubt that the vesicles upon the cornea are entirely analogous to those that develop upon the skin. Just as the latter are generally present only upon one side of the face, so also the affection of the eyes is usually unilateral in its development, and is, moreover, confined to the same side as the vesicles upon the face. With careful treatment the prognosis is good, as in that case the disease generally gets well without leaving any opacity. The treatment is purely symptomatic, being that which is indicated for corneal ulcers generally—that is, in the main, the employment of a protective bandage and of atropine. [In accordance with the probable neuropathic origin of many of these cases, arsenic is often indicated (Verhoeff).

Iontophoresis (page 58) with zinc sulphate (½-per-cent solution) has been successful in some cases (Zahn).—D.]

228. (*b*) *Herpes Zoster Corneæ.*—This is one of the symptoms of herpes zoster[14] ophthalmicus—that is, zoster which is localized in the region of distribution of the trigeminus (see § 588). The cornea participates in the morbid process by forming small vesicles, which generally are arranged in groups and rupture speedily, as in the case of herpes febrilis. From the

[12] From ἕρπειν, to creep.
[13] Hebra's herpes facialis.
[14] = girdle-eruption, from ζωστήρ, girdle. We also speak of it as zona ophthalmica.

latter, however, herpes zoster is distinguished by running a considerably severer course, since the irritative symptoms persist after the rupture of the vesicles, the parenchyma of the cornea becomes deeply clouded at the spots where the vesicles were situated, and iritis is added to the other symptoms. Sometimes a regular keratitis profunda develops (page 299). It takes a longer time for the opacities to disappear, nor is it always the case that they disappear completely. The insensitiveness of the cornea to touch is especially pronounced in herpes zoster. Reduction of the intra-ocular tension not infrequently exists so long as the inflammation is still recent; in other cases increase of tension sets in. The prognosis of this variety of herpes, from what has just been said, is less favorable than that of herpes febrilis; the treatment is the same.

[Iontophoresis with quinine is serviceable, particularly in reducing the pain (McNab).—D.]

229. (*c*) *Keratitis Vesiculosa* (*et Bullosa*).—This variety occurs in eyes the cornea of which is more or less clouded and insensitive; in eyes with a large corneal cicatrix, or eyes which have been rendered blind by irido-cyclitis or by increase of tension. In such, with the accompaniment of violent

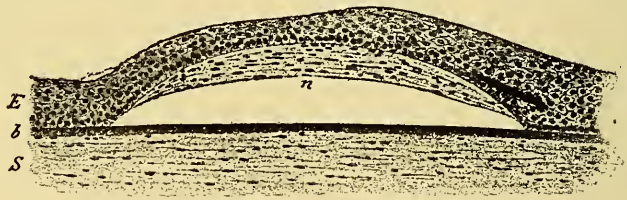

FIG. 105.—KERATITIS BULLOSA. Magnified 92 × 1.

The small vesicles of herpes corneæ are produced by detachment of the epithelium alone from Bowman's membrane, and since the corneal epithelium is quite delicate, are very evanescent. On the other hand, the larger bullæ in keratitis bullosa form in corneæ which have been already diseased and in which there has been a new growth of fibrous connective tissue between the epithelium and Bowman's membrane. This connective tissue is detached together with the epithelium by fluid and gives greater stability to the anterior wall of the bulla. The above figure is taken from an eye rendered blind by irido-cyclitis. Bowman's membrane, *b*, covering the stroma of the cornea, *S*, is stained dark because it is filled with lime granules corresponding to the zonular opacity of the cornea which is present (page 310). The epithelium, *E*, is detached over the area of half a millimetre. At the highest point of the bulla it is thin, and on its posterior surface lies the new formed connective tissue, *n*.

inflammatory symptoms, either vesicles which are small and of short duration form upon the cornea (keratitis vesiculosa), or large tremulous bullæ may develop, which last for several days before they rupture (keratitis bullosa; Fig. 105). In all cases the vesicles show a great tendency to take on frequent recurrences, in each of which the irritative symptoms set in anew.

The cause of the formation of vesicles appears to lie in the abnormal conditions of lymph circulation that are without doubt present in such eyes. By stasis of the lymph œdema of the cornea is produced; the œdematous fluid penetrates forward until it gets beneath the epithelium, and then lifts the latter up in spots from Bowman's membrane.

The prognosis is so far unfavorable in that the disease frequently recurs, on which account the affected eye, besides being useless for purposes

of vision, is the source of constant discomfort to the patient. Treatment should aim at relieving the condition of irritation produced by the eruption of vesicles, and at preventing the recurrences. The former object is attained by opening the vesicles, the smaller ones being pricked, and in the case of the larger ones the anterior wall being removed. When the vesicles recur frequently at the same spot we must try to modify the character of the base from which they develop. We remove the epithelium and also any deposit that may chance to be present in Bowman's membrane by shaving off the most superficial layers of the cornea, which we then paint with tincture of iodine. If nevertheless recurrences set in we may, in order to obtain a deeper effect, cauterize the surface of the cornea lightly with the galvanocautery loop. Sometimes we can check the recurrences only by an iridectomy; and we may actually have to enucleate the eye in order to give the patient ease.

230. Other Forms of Vesicular Keratitis.—Apart from the forms above described, the formation of vesicles upon the cornea is further, in rare cases, observed under special conditions—e. g., as a result of the action of various, and particularly of corrosive, substances upon the cornea, after burns, after a cataract operation beneath the bandage, etc. For the vesicles that usher in fresh recurrences of former erosions of the cornea see page 305. Cases also occur in which, without known cause, there develop upon a perfectly sound cornea vesicles or bullæ the formation of which we are inclined to attribute to nervous influences, as we do also in herpes febrilis and herpes zoster. Cases of this sort are usually characterized by periodical recurrence. One old lady for twelve years suffered from occasional attacks of inflammation in her eyes, which otherwise were sound. The inflammation occurred once or twice a year, and affected sometimes one eye, sometimes the other. It was associated with violent pain, great photophobia, and profuse lachrymation. In the first days of the attack the only things found were œdema of the lids, great ciliary injection, and a cornea covered with minute elevations, as if it had been strewn with sand. Then a large transparent bulla developed upon the cornea, after the rupture of which the inflammatory symptoms rapidly abated and the epithelial defect healed without leaving a trace behind. [An allied condition is superficial linear keratitis (page 260), which also tends to recur and is undoubtedly neuropathic.—D.]

231. Keratitis Punctata Superficialis.—This is a form of superficial keratitis related to herpes febrilis corneæ, but not associated with the formation of vesicles. It begins with the symptoms of an acute conjunctivitis. Either at the same time or not till some days or weeks afterward there are observed extremely minute dots which can be seen only with a magnifying glass and which within a few days grow to form faint gray round spots (Fig. 106). These are sometimes only ten to twenty in number, sometimes very abundant—upward of a hundred. As in the case of herpes febrilis these are often arranged in groups or in short rows. In every case, however, the marginal portions of the cornea are the part least covered by the spots. The spots lie in the most superficial layers of the cornea, which latter looks dull because the epithelium over the spots bulges out in the form of a nodule. The irritative symptoms soon vanish, but the spots, as well as the punctate look of the corneal surface, generally remain for

Fig. 106.—Keratitis Punctata Superficialis.

months almost unchanged, and then very gradually disappear. If the spots are not numerous, the sight remains undisturbed; if, however, many spots are present, particularly in the center, the acuity of vision is considerably reduced. Keratitis punctata superficialis is found most frequently in young people, and affects sometimes one, sometimes both eyes. It often begins at the same time with a catarrh of the air passages, just as herpes febrilis corneæ does, but is distinguished from the latter mainly by the absence of the formation of true vesicles. Hence, too, in keratitis punctata superficialis the superficial losses of substance which develop from the vesicles in herpes are wanting, and for the same reason the formation of ulcers in this variety of keratitis is observed only as a rare exception.

232. **Filamentary Keratitis.**—In various slight, superficial affections of the cornea, in which its epithelium is affected, we observe that fine filaments are formed, which adhere by one end pretty firmly to the surface of the cornea, while the other end, which is often swollen in a club shape, hangs down free (Leber, Uhthoff, Fischer). The filaments are produced by a process of outgrowth from the epithelial cells of the cornea (Hess, Nuel).

(*b*) Deep Forms of Non-Suppurative Keratitis

233. These forms have as a common characteristic the development of an infiltrate in the middle and deep layers of the cornea, an infiltrate, however, which shows no tendency toward purulent disintegration, but—generally not till after existing quite a while—disappears again by resorption. When this takes place, the cornea in favorable cases clears up again completely, while in other cases opacities of a varying degree of intensity are left, and are sometimes even accompanied by flattening of the cornea. In keeping with the deep position of the infiltrate in the cornea, the uveal tract and especially the iris and ciliary body are almost always implicated.

3. *Parenchymatous Keratitis.*[15]

234. Invasion.—This affection may run its course in two ways, according as it begins in the center or at the margin of the cornea. If the disease invades the *center* of the cornea first, we see small, dim, gray maculæ making their appearance in this situation, and lying in the middle and deep layers of the cornea; the surface of the latter is lusterless and dull. The number of maculæ gradually increases, so that they keep extending farther and farther toward the margin; but they are always massed most thickly in the center, where they frequently become confluent. Since even between the maculæ the cornea is not clear, but shows a fine diffuse cloudiness, the entire cornea may in severe cases get to look uniformly gray, like ground glass. As soon as the opacity of the cornea has advanced somewhat farther, vascularization begins by the penetration of vessels into the cornea from different spots upon the corneal circumference. We see the vascular trunks coming out from beneath the limbus, because—as opposed to the vessels in pannus—they arise from the deeply situated vessels of the adjacent sclera (Figs. 85 and 86). They branch in tufts, like a brush, in the deep layers of

[15] Synonyms: keratitis interstitialis, keratitis profunda, keratitis diffusa, uveitis anterior.

the cornea, and often appear quite indistinct and of a dirty-red or grayish-red color, because they are covered by the clouded superficial layers of the cornea.

In those cases in which the disease begins at the *margin* of the cornea, the first thing that strikes us is that the latter has grown lusterless and clouded at some spot upon its margin. The cloudiness is deeply situated, and when regarded with the naked eye looks uniformly gray, but with the magnifying glass can generally be resolved into separate maculæ or dim parallel streaks. Soon similar areas of cloudiness appear at other spots of the corneal margin, and then push their way forward concentrically from all sides toward the center of the cornea. Simultaneously with the appearance of the marginal opacities the corresponding portions of the limbus become injected and the vessels of the corneal margin begin to grow out. The formation of vessels, as far as it originates in the network of marginal loops in the limbus, soon comes to an end, so that the limbus advances only a little way out upon the cornea, where it looks red and swollen ("epaulet-like" swelling of the limbus). While this swelling soon goes down again, the deep vessels, which come out from beneath the limbus, grow farther and farther into the cornea, and follow closely upon the opacity which advances in front of them; it looks as if they were pushing the opacity before them. These vessels have, as in the case of those of the first form, the characters of deeply situated vessels, shown by their broom-like branching and by their dull, dead, grayish-red hue.

When parenchymatous keratitis has attained its acme, the cornea is often so opaque that we scarcely recognize the iris through it. At the same time it loses its luster completely, so that it looks as though smeared with grease (with the magnifying glass we recognize numerous minute elevations of the epithelium, which make the surface of the cornea rough, as if made of fine shagreen). Sight is so reduced that the patient can only count fingers held very close to him, or, still worse, can only recognize the movement of the hand before his eye. Now gradually the process of recovery begins, starting from the margin, where the cornea first becomes transparent again, while at the same time the vessels grow constantly fewer and fewer. The center of the cornea remains opaque the longest, but finally clears up, too, until only a fine diffuse cloudiness remains, which causes but little impairment of sight. This cloudiness, together with a few very minute blood-vessels which are visible only with the magnifying glass, can still be made out years afterward, and are certain signs of the previous existence of a parenchymatous keratitis.

235. Among the more frequently occurring *variations* in the clinical picture of parenchymatous keratitis the following may be mentioned:

In that form which begins with maculæ in the central portions of the cornea it not infrequently happens that the maculæ at a certain distance from the center are particularly dense, and thus form a very opaque ring, which in the subsequent course

contracts more and more toward the center of the cornea (*keratitis centralis annularis* of Vossius). Allied to these cases are those in which the central part of the cornea becomes particularly opaque through the confluence of the maculæ, which form a white disk pretty sharply separated from the less opaque, marginal portions of the cornea. I have seen several cases in which this central opacity remained permanently after the marginal portions had cleared up, and formed a dense, white, sharply circumscribed spot in the center of each cornea, just as if there had been a deeply penetrating central ulcer.

Sometimes the distribution of the infiltration in the cornea is such that the densest opacity occupies the lowest part of the cornea, as if the products of inflammation had arranged themselves there in obedience to the law of gravitation. In that case the opacity is bounded above by a convex line, or it forms a triangle, the base of which

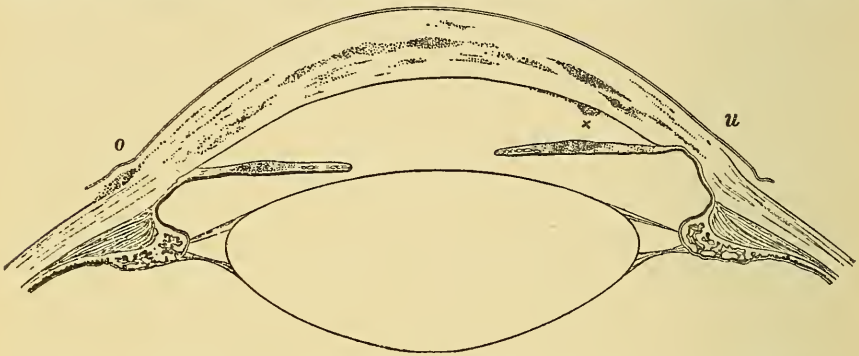

FIG. 107.—PARENCHYMATOUS KERATITIS. Magnified 7 × 1.

The patient, a girl of nineteen, had already passed through one attack of parenchymatous keratitis six years before. This recurred, the cornea became ectatic, increase of tension set in, and as the result of this the eye became entirely blind. Since the eye was inflamed and painful all the time, it was enucleated at the patient's own wish. At the time of enucleation, the cornea was densely permeated with little white spots which were situated in the middle layers of the cornea, and the latter was consequently so opaque that the iris could not be seen through it. In the preparation, contrary to the usual relations, the cornea is seen to be somewhat thicker in the center than at the edge. To these clinically demonstrable white spots correspond small foci, consisting of uninucleated round cells. These foci are largest and most numerous in the middle layers of the cornea. At the margin of the cornea are to be seen, even in the anterior layers, files of cells accompanied by new formed vessels. At the upper margin of the cornea, *o*, there is situated a small episcleral inflammatory focus. Near the lower margin of the cornea at *x*, there is a mass of exudate (precipitate) on the posterior surface of the cornea. The iris, as is the rule when there is an increase of tension, is adherent to the cornea by its periphery, and hence is driven bodily forward, so that it no longer is in contact with the lens. The marginal portion of the iris which is adherent to the cornea is very much thinned by atrophy and at the lower side, *u*, is pushed somewhat into the also attenuated sclera (beginning of an intercalary staphyloma). The free portion of the iris is rather less atrophic, and both in its upper and lower part contains an inflammatory nodule, which like the nodules in the cornea, consists simply of uninuclear cells. The ciliary processes are well preserved. They are at a greater distance than normal from the margin of the lens, because, owing to the increase of tension, the eye as a whole has become somewhat enlarged but the lens has not shared in the enlargement.

corresponds to the lower margin of the cornea, while its apex looks upward. The permanent opacity that results from this has the greatest similarity to those triangular opacities in the lower part of the cornea which are left by an irido-cyclitis, when an exudate at the bottom of the anterior chamber has remained deposited for a pretty long time upon the posterior surface of the cornea.

In irido-cyclitis resulting from acquired syphilis it not infrequently happens that isolated gray specks appear in the middle and deep layers of the cornea. These have been described by Mauthner, Hock, Purtscher, and others as *keratitis punctata syphilitica*. This is distinguished from keratitis punctata superficialis (page 287) not only by the etiology, but also by the situation of the specks in the deep layers of the cornea, and might therefore be appropriately characterized as *keratitis punctata profunda*.

236. Morbid Anatomy.—Anatomical examination of an eye affected with parenchymatous keratitis shows small foci of inflammation which correspond to the gray specks seen on clinical inspection and which are formed by an accumulation of uninuclear leucocytes in the middle and deep layers of the cornea. Similar nodules may also be found in the anterior portions of the uvea (iris, ciliary body most anterior part of the chorioid—see Fig. 107). The nodules sometimes contain giant cells, but yet are not typical tuberculous nodules. It may chance that disintegration of the corneal substance itself occurs at the site of the nodule, but there is not the excavation that is characteristic of tuberculosis. In the case represented in Fig. 108, instead of discrete nodules there

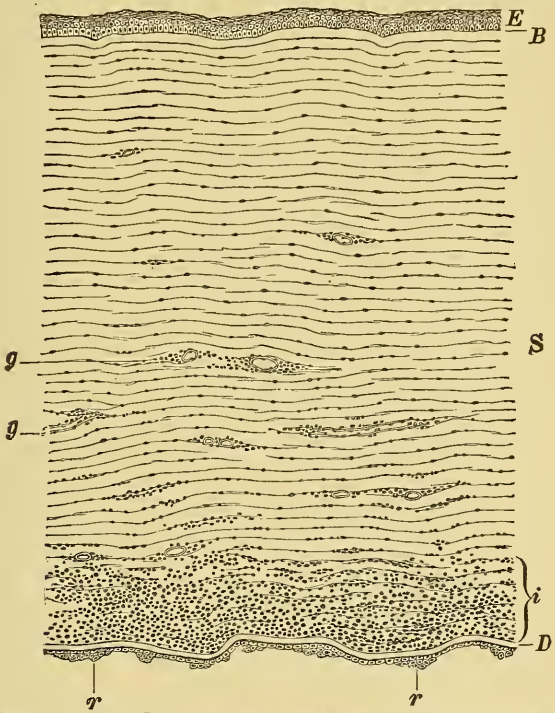

FIG. 108.—CROSS SECTION THROUGH A CORNEA WITH PARENCHYMATOUS KERATITIS. Magnified 100 × 1. (After a preparation of Dr. Nordenson's.)

The stroma, *S*, of the cornea shows an infiltration, which begins in the middle layers, and keeps on increasing more and more posteriorly, so that the deepest layers, *i*, have assumed the aspect of a granulating tissue. On account of the inequality in the degree of thickening of these layers, Descemet's membrane, *D*, is undulated; upon its endothelium there are deposited in places small accumulations of round cells, *r*. In the middle and deep layers of the cornea we see the transverse and longitudinal sections of newly formed blood-vessels, *g*, *g*, while the most anterior layers, and also Bowman's membrane, *B*, and the epithelium, *E*, are normal.

was a uniform dense infiltration of the most posterior layers of the cornea, so that the latter looked as if transformed into granulating tissue; furthermore, numerous new-formed blood-vessels (*g*) were seen in the most posterior and the middle layers. In deep-seated infiltration destruction of Descemet's membrane sometimes occurs in spots.

237. Course.—Parenchymatous keratitis always runs a chronic course. The inflammatory symptoms keep on increasing for one or two months, until the disease has reached its acme. Then the irritative symptoms very soon abate, and the process of clearing up of the cornea makes at first rapid

progress. Afterward, however, it goes on more slowly again, and the center of the cornea in particular remains for a long time opaque, so that sight is not restored until late in the disease. It takes from half a year to a year, or even more, for the cornea to acquire the full degree of transparency which it is possible for it to assume with an inflammation of the given intensity.

All the cases do not run their course in the way described. There are, for example, many lighter cases in which the changes do not go far, and which hence, too, are completed in a shorter time. Thus the process may go no further than the formation of a few maculæ, which gradually disappear again without the associated inflammatory symptoms being at any time marked. If the opacity begins at the margin of the cornea, it often remains confined to that section of it from which it originally started. In that case, if it pushes its way farther from the margin toward the center, only a sector, and not the entire cornea, is rendered opaque. Conversely, there are also very severe cases in which dense opacities remain as a permanency. Again, owing to the inflammatory infiltration, softening of the cornea may be produced, so that the latter gives way before the intra-ocular pressure, and keratectasia develops; in this case also the cornea remains permanently opaque, and to quite a marked degree. The worst cases are those in which, by subsequent shrinking of the exudate, the cornea becomes flattened, densely opaque, and of a tendinous appearance, in which case the sight is nearly or quite lost.

Just as great variations exist in regard to the density and extent of the infiltration, so they do also in regard to the *vascularization*. In many cases the cornea is so abundantly vascularized that it looks like a red cloth; in others, on the contrary, it is almost devoid of vessels, and is like white ground glass. Lying between these extremes are numerous cases in which vessels develop from only single spots upon the corneal margin, so that simply a sector of the cornea looks red, or so that only single tufts of vessels are discoverable. Hence we may distinguish between a vascular and a non-vascular form, according to the relations of the vessels. It must be remarked, however, that even in the non-vascular form one or two vessels can generally be made out with the aid of the magnifying glass.

When we consider how the cases vary both in respect to the opacity and to vascularization, we comprehend why parenchymatous keratitis should present a very varying clinical picture, and hence often offer difficulties in the way of diagnosis for the beginner. But we shall generally be able to make the diagnosis with certainty if we hold fast to those symptoms which are common to all the cases—namely, the deep situation of the opacity and of the vessels, the typical increase in the infiltration up to a certain, usually considerable, degree, and finally the absence of purulent disintegration, so that the *formation of ulcers* [*practically*] *never takes place.*

The *vessels* in parenchymatous keratitis generally lie in the deep layers. Yet it often enough happens that we find, especially with the aid of the magnifying glass, a few vessels also which evidently arise from the network of marginal loops or from larger conjunctival blood-vessels, and which therefore lie superficially in the cornea. All vessels tend toward the center of the cornea, but do not generally reach it, so that here a roundish spot of the size of a millet seed or more remains unvascularized. The vascular portions of the cornea look red, and if the vessels are abundant, rise above the level of the non-vascular center; the latter, accordingly—which, in consequence of the marked infiltration, is gray or even yellowish gray—is depressed. We must not on this account allow ourselves to be misled and consider the depressed gray spot as an ulcer, for parenchymatous keratitis does not, as a general thing, lead to ulceration. Exceptions to this rule, though rare, do, however, occur. I have seen, in fact, two cases in which perforation had taken place in the center of the cornea.

238. Symptoms, and Complications.—Parenchymatous keratitis is accompanied by *irritative symptoms* of inflammation, such as pain, photophobia, and lachrymation. These are sometimes very slight, sometimes violent; in general we may say that they are more pronounced, the greater the amount of vascularization with which the keratitis is associated. Furthermore, parenchymatous keratitis is almost always complicated with *inflammation of the uveal tract.* In the lightest cases there is merely hyperæmia of the iris, which makes itself apparent by the failure, complete or nearly so, of the pupil to dilate under atropine. In severe cases there is irido-cyclitis, which may lead to the formation of posterior synechiæ, the formation of deposits upon the posterior surface of the cornea, and seclusion and occlusion of the pupil. In particularly bad cases a plastic irido-cyclitis develops, which terminates in flattening of the cornea, or even in atrophy of the eyeball.

Parenchymatous keratitis generally attacks both eyes, and more frequently both in succession than both at once. Sometimes there is even an interval of several years between the involvement of the two eyes. Recurrences of the disease take place, but are not common.

The uvea is practically always implicated in parenchymatous keratitis. To be sure, the participation of the uvea in the inflammation is not by any means equally pronounced in all cases. Minute deposits, which are discovered in making a careful examination of the cornea with the magnifying glass at the time when the opacity is resolving, are scarcely ever wanting. Besides these, the most frequently occurring complications are posterior synechiæ and also chorioiditic foci; hypopyon, on the other hand, is extremely rare. In many cases the part that the uvea takes is so slight as not to be clinically demonstrable; in other cases, on the contrary, it is so very prominent, as compared with the process in the cornea, that what we have before us is really an irido-cyclitis—the implication of the cornea being evidenced only by the presence of a few spots of opacity in its deep layers. Thus there is a continuous series of intermediate forms between typical parenchymatous keratitis and irido-cyclitis e lue hereditaria. [When both uvea and cornea are markedly involved the condition is often called *uveitis anterior.* This disease has the characters of a chronic iritis (see § 374) combined with a marked keratitis and sometimes with a deep scleritis as well. It is extremely chronic, and is

undoubtedly of constitutional origin, probably often being due to tuberculosis, although in many cases the cause is obscure.—D.]

The *anterior chamber* in parenchymatous keratitis is often found to be deeper than usual, a circumstance which should not, however, be referred without further consideration to an ectasis of the cornea, a thing which occurs quite rarely. On the contrary, the cause of it ordinarily is a recession of the iris due to the increased accumulation of the aqueous—an accumulation to which both the irritated state of the uvea and the altered conditions of filtration may contribute.

Often it is impossible to obtain an adequate dilatation of the pupil with atropine, even though no evident signs of iritis are present. It seems that in these cases the atropine does not diffuse through the inflamed cornea to the same extent as it does through a sound one, so that it does not in fact get into the aqueous in sufficient quantity to dilate the pupil.

The *intra-ocular pressure* not infrequently shows an alteration in parenchymatous keratitis. Generally, it is diminished so that the eye appears softer, although we need not therefore imagine that an atrophy of the eyeball is beginning. An increase of tension is but rarely observed, occurring for the most part only when the keratitis has induced ectasis of the cornea. In that case sometimes it does not occur until years after the inflammation has run its course. I have seen this even in those cases in which no ectasis of the cornea has been left.

Many cases of parenchymatous keratitis are accompanied by *chorioiditis*. This is localized in the most anterior segment of the chorioid (chorioiditis anterior), which is covered with numerous—in most cases black—spots. This variety of chorioiditis would probably be set down among the most frequent symptoms accompanying parenchymatous keratitis if the examination with the ophthalmoscope, and hence the determination of the presence of chorioiditis, were not rendered impossible while the inflammation lasted by the cloudiness of the cornea. The making of this examination and diagnosis can be done only when the cornea has cleared up once more after the inflammation has run its course. It is also often possible to make out the existence of peripheral chorioiditis in the other, as yet uninflamed eye.

Another and rarer complication of parenchymatous keratitis is a diffuse scleritis in the region surrounding the cornea. This may subsequently give rise to ectasiæ of the sclera.

239. The *prognosis* of the disease, from what has been said, is unfavorable as regards its duration, inasmuch as it drags on for months and years, especially if the two eyes are successively attacked. On the other hand, the prognosis in regard to the ultimate outcome must be put down as good, because in by far the greater number of the cases a good, or at least serviceable, degree of sight comes back. By holding up this prospect of recovery the physician must keep up the courage of his patient, who, because of the slow progress of the disease, is very apt to lose all hope of a restoration of his sight.

240. Etiology.—Parenchymatous keratitis is a disease of youth, appearing, as a rule, between the sixth and the twentieth year of life. It is only the exception that persons before or after this age (sometimes even after the thirtieth year) are attacked. The female sex suffers from it more frequently than the male. The ordinary cause of the disease is syphilis, and especially hereditary syphilis. To prove the existence of hereditary syphilis

from the history of the case directly—i.e., by getting the father or mother to confess to syphilis—is often a difficult thing to do. Besides, in most cases it is not at all necessary, since hereditary syphilis can generally be recognized with sufficient certainty from a series of symptoms. In that case, we abstain from questioning the parents in regard to this matter, the more so since it would be a severe reproach to them to have to recognize in their own persons the cause of their children's illness. On the other hand, it is a good thing to determine by questioning whether many children have died in the family (the mortality of the children of syphilitic parents amounts, on an average, to 50 per cent), whether premature labors, and especially those in which the fœtus was dead or putrefied, have not occurred, etc.

The *symptoms of hereditary syphilis* which patients with parenchymatous keratitis frequently exhibit are as follows:

1. A peculiar formation of the face and cranium. The upper jaws are markedly flat, and the bridge of the nose low or actually sunken in. Not infrequently there is ozæna or disease of the lachrymal sac, the latter in consequence of the changes in the nose. The frontal eminences are very prominent. The cornea, which is ordinarily a horizontal ellipse, has often the

Fig. 109.—The Upper Incisor Teeth in Hereditary Syphilis. (After Hutchinson.)

A. The interspace between the middle incisor teeth is abnormally broad. The teeth themselves have a semilunar notch on their cutting edge. The two lateral incisor teeth have somewhat convex lateral borders which converge downwards.

B. The incisor teeth are all stunted, and hence the interspaces are enlarged. The middle incisor teeth are most undeveloped and show the notching of the cutting edge. The lateral incisor teeth have convex sides which converge downwards.

shape of a vertical ellipse. The intelligence of these patients is often abnormal, they being either precocious, or, on the other hand, backward in mental development.

2. The incisor teeth are abnormally shaped (Hutchinson), so that, instead of a straight edge, they show a semilunar notch (Fig. 109). This change is present only in the teeth of the second dentition, and in them most frequently in the upper central incisor teeth. Often the incisor teeth are all stunted, so that they are either too small or some are altogether wanting.

3. At the angles of the mouth we may find fine cicatrices, the relics of former rhagades; so also cicatrices in the buccal and pharyngeal cavities (especially on the hard and soft palate) point to the existence of antecedent syphilitic ulcerations.

4. Numerous enlarged lymphatic glands can be made out, especially upon the neck. These are small, hard, painless, and with no tendency toward ulceration, by which characters they are distinguished from the

lymph glands of scrofulous subjects, which are large and soft, and readily undergo caseation.

5. Swellings of the periosteum (tophi), which are hard and are but little or not at all painful, occur on the long bones. They are most frequently and most easily found upon the anterior border of the tibia.

6. Often there is inflammation of the knee-joint, either occurring simultaneously with the keratitis or, more often, antedating it by some years. The inflammation is ordinarily bilateral, pretty torpid, and of several months' duration, but benign, in that it gets well without leaving lasting sequelæ.

7. We frequently find hardness of hearing, which, with the outbreak of the keratitis, sometimes increases to absolute deafness.

It is important to look for all of these symptoms, for any one of them by itself is not to be looked upon as conclusive evidence of the existence of hereditary syphilis; and, on the other hand, we ought not to expect to find all the changes above given distinctly marked in the same individual at once. If, nevertheless, a case remains doubtful, we make a Wassermann [or luetin] test, which in cases of hereditary syphilis usually turns out positive. The more carefully we make our investigation, the more do we arrive at the conviction that by far the greatest number of cases of parenchymatous keratitis are to be referred to hereditary syphilis. In a few cases this form of keratitis is also observed in acquired syphilis, and we must not forget that the latter may be acquired even in childhood (e.g., through a nurse). A few cases depend upon tuberculosis, while, in some, no cause at all that we can be sure of is discoverable to account for the eye disease.

The typical course of parenchymatous keratitis, and the participation of both eyes in it, early suggested the idea of its being due to a *constitutional cause*. Thus Mackenzie gave an excellent description of this disease under the name of corneitis scrophulosa, and at the same time gave a number of the accompanying symptoms which he looked upon as signs of scrofula. Hutchinson has the credit of having completed this series of symptoms, and of having at the same time furnished the proof that they belong not to scrofula, but to hereditary syphilis. This novel view was slow in making its way. Many at first would only allow that it was true for a limited number of cases, and hence divided parenchymatous keratitis into two forms, which they called keratitis scrofulosa and keratitis syphilitica. But the more precise our knowledge becomes in regard to the symptoms of hereditary syphilis, the more surely we arrive at the conviction that this disease lies at the root of parenchymatous keratitis, whatever form the latter may exhibit. Some have succeeded in producing in monkeys and rabbits by inoculating them with syphilitic material an inflammation of the cornea which resembles the parenchymatous keratitis of man and in which, too, spirochætæ could be demonstrated in the tissue of the cornea (Scherber, Greeff).

[Positive evidence of syphilis is afforded in nearly all cases of parenchymatous keratitis by the *Wassermann* or *luetin tests* (in 56 out of 63 cases in Glasgow, in 37 out of 38 examined by Lesser and Carsten. According to the last-named, in nearly 80 per cent of the cases syphilis can be demonstrated in at least one of the parents, and in 70 per cent it can be demonstrated in both).—D.]

[It has been suggested that parenchymatous keratitis may be an *anaphylactic phenomenon*, the cornea being sensitized at some early period of life (perhaps even in intra-uterine life) by the syphilitic virus, so that later it is readily attacked even by the attenuated virus (Derby and Walker), this second attack occurring at some time when the resistance of the system is reduced in some way. Some think that this *reduced general resistance* is itself the essential factor, and that because of it the cornea and uvea succumb to an attack that they would otherwise withstand. It is on this principle that Dunn explains the genesis of uveitis in keratitis parenchymatosa. He says that a vicious circle is established, by (*a*) the development of an autotoxæmia, originating probably in the intestinal tract; (*b*) excessive action of the thyroid gland to combat the toxæmia; (*c*) failure of the thyroid resulting in hypothyroidism and pervading uncontrolled toxæmia. According to this view, which resembles that held by Elschnig, the uveitis accompanying keratitis parenchymatosa is often due not so much to the syphilis as to metabolic toxæmia and general malnutrition due to hypothyroidism. The theory seems to find some corroboration in the occasionally successful results of treatment with the thyroid extract (see page 299).—D.]

Some believe that at times in an heredito-syphilitic individual trauma may occasion the outbreak of a parenchymatous keratitis.

Parenchymatous keratitis is one of the latest of the forms under which hereditary syphilis shows itself and is therefore rightly regarded as one of the most important and most frequent symptoms of lues hereditaria tarda. Many believe that there is no lues hereditaria tarda if we use the term in the sense that the symptoms of lues which appear in later life are really the first symptoms of the disease; they think that, on the contrary, luetic symptoms must have been present in every such case either at the time of birth or soon after, but that either these symptoms were overlooked or else the physician had failed to encounter any signs of them because he had taken charge of the case after the symptoms had gone. Thus it is supposed that parenchymatous keratitis is never really the first symptom of hereditary syphilis. But positive observations like the following contradict this view: A physician brought to me his six-year-old boy who a short time before had become affected with a typical parenchymatous keratitis. In other respects the child was healthy, well developed, and had a good appearance. The father had acquired syphilis twelve years before, that is, six years before the birth of this child. Some years afterwards he married. The first child his wife brought into the world was still-born, the second was the little patient brought to me. This one, the father, conscious of his lues, had watched anxiously and carefully from birth, but had never been able to discover in him the slightest signs of syphilis, until the parenchymatous keratitis showed itself. The latter, therefore, in this case must be regarded as being the first demonstrable sign of the inherited syphilis.

The following history may serve to show how from different symptoms we get at the diagnosis of hereditary syphilis. A twelve-year-old girl with parenchymatous keratitis of both eyes was brought into the clinic by her mother. The latter declared that she had no knowledge of syphilis as affecting either herself or her deceased husband. She only admitted that the latter had led an irregular life. The woman went on to say that she had been pregnant by this man ten times in all. From the first four pregnancies came four children (the eldest at present about twenty-two years old), who are all healthy. The fifth child died at the end of one year, the sixth pregnancy ended in an abortion, the seventh child is the little patient who is brought into the clinic, the eighth child died at the age of nineteen months, the ninth child is living but is always sickly, and the tenth died at the age of six weeks. Then the husband died in consequence of an accident; the woman married a second time, and has had by her second husband two perfectly healthy children. The woman's daughter who was brought into the clinic was deaf; she presented, in addition to the parenchymatous keratitis of both eyes, the

characteristic formation of the cranium belonging to syphilitic children. The teeth showed the form described by Hutchinson; numerous small, hard lymphatic glands were found upon the neck. I made the younger sister (the woman's ninth child) come too. She, who was a feeble girl, was not, to be sure, absolutely deaf, but heard very badly, and had the characteristic formation of the face and swollen lymphatic glands upon the neck; and the teeth—they were the milk teeth—were markedly small and separated by wide interspaces. Externally the eyes looked healthy, but in both the periphery of the fundus was found by the ophthalmoscope to be covered with spots, black as ink, lying in the chorioid. The correct interpretation of this history, without doubt, is that the woman's first husband had acquired syphilis after the fourth pregnancy. While, therefore, the woman's first four children enjoy very good health, only two of the children of the six following births are living and both are sickly, both marked with evident symptoms of hereditary syphilis. When the woman had become pregnant by her second healthy husband, she had healthy children once more.

From the history just submitted, it can be deduced that the examination of the little patient's brothers and sisters may often contribute to the clearing up of the case, inasmuch as we may find in them, too, symptoms of hereditary syphilis, and thus may still further confirm the diagnosis. Moreover, it is not at all rare for two or even three of a set of brothers and sisters to be affected with parenchymatous keratitis. Furthermore, from this history we may deduce the important fact that a woman may bear syphilitic children begotten by her diseased husband, without at the same time acquiring syphilis herself; to the syphilis of her own children she is immune.

241. Treatment.—*Local* treatment during the period of progression consists in combating the inflammation by protecting the eyes from light and by instilling atropine, which latter counteracts the complications arising from the iris. Moist hot compresses often ameliorate the symptoms of irritation and accelerate somewhat the progress of the disease. Dionin acts favorably on the photophobia and the pain. [Many think subconjunctival injections (page 65) very effective.—D.]

In the regressive period, the thing to do is to secure as thoroughgoing a clearing up of the cornea as possible. For this purpose dionin again and the well-known irritant remedies—like calomel, tincture of opium, yellow-precipitate ointment, steam, hot air, etc.—are indicated (see page 266). These, however, should be brought into use only when, on making cautious tests, the eye is found to bear them well—i.e., does not through their use fall into a state of renewed irritation. It is advisable to keep on with these remedies, interchanging them frequently, for a very long time—for months or years. If ectasis of the cornea threatens, it is to be combated by a pressure bandage, which, if necessary, can be combined with repeated paracentesis of the cornea.

General treatment in those cases in which syphilis is the cause must be directed against the latter. Salvarsan has not proved effective. [Yet, according to many, it is helpful in shortening the process, especially if it is given repeatedly. It should be combined with mercurial treatment.—D.] The latter alone is ordinarily not very efficient [though it is more so in cases due to acquired syphilis]. In adults we use mercury by inunction or better still by intramuscular injection. [We give at periodical intervals a series

of injections, using the bichloride (up to gm. 0.01) in solution every other day, or the salicylate (up to gm. 0.06), or calomel suspended in albolene or oil every ten days.—D.] In children the internal administration of corrosive sublimate is preferable. We give pills of 1 mg., beginning with one a day, increasing the dose to from six to ten pills a day, according to the age, and being mindful to pay careful attention to the condition of the mouth, in order to avoid salivation. [In general, the best results are obtained if careful and persistent hygienic measures (fresh air and exercise, good diet, etc.) are combined with the specific treatment (Derby and Walker).—D.] In the lighter cases of parenchymatous keratitis, indeed, we may confine ourselves to a simple tonic treatment with the simultaneous employment of remedies containing iodine (cod-liver oil with iodine, iodide of iron, and mineral waters containing iodine, the latter especially as a form of after treatment). [Thyroid extract, gm. 0.20 to 0.30, according to age, may be used with advantage in some cases (Radcliffe, Dunn).—D.] Unfortunately, we must say that, in many cases, parenchymatous keratitis, even under the most careful treatment, runs a course that is not essentially different from what would have been the case without any treatment at all. Not infrequently we see the disease while under treatment break out in the other eye, without our being able to prevent the opacity from spreading gradually over the entire cornea in this eye also. [Yet, though we may not be able to arrest the process, we can in many cases shorten it, and we can render important service by combating the complications arising from the uvea, and also in securing a more rapid and more perfect clearing up of the corneal opacities during the period of regression.—D.]

4. *Keratitis Profunda*[16]

242. In this a gray opacity develops very gradually in the cornea—ordinarily in its center—an opacity which is situated in the middle and deep layers of the cornea, and over which the corneal surface is gray and punctate, but not depressed. Seen with the naked eye, the opacity looks uniformly gray, while with the magnifying glass it may be resolved into dots and maculæ, or into gray interlacing striæ. After the opacity has remained for some time (several weeks) at its acme it begins slowly to abate, without ulceration having taken place. The development of new vessels is either entirely absent or is very inconsiderable. The accompanying symptoms of inflammatory irritation are sometimes slight, sometimes pretty violent. The participation of the iris is mostly limited to hyperæmia.

The disease attacks adults only. It lasts from four to eight weeks or more. In the lighter cases it terminates in a complete restoration of the transparency of the cornea, while in other cases diffuse opacities remain permanently in the center of the cornea.

[16]Synonyms: central parenchymatous infiltration of the cornea, keratitis parenchymatosa circumscripta.

The *causes* of keratitis profunda are in the great majority of cases unknown. For individual cases the following causes have been given: 1. The effect of cold. Arlt has characterized such cases as keratitis rheumatica. These ordinarily run their course with marked inflammatory symptoms, especially with violent pain and photophobia. 2. Herpes zoster ophthalmicus (§588). 3. Intermittent fever in its chronic form of malarial cachexia (Arlt). In this sometimes a keratitis profunda develops which is characterized by the absence of marked symptoms of irritation, and also by an unusually chronic course (Arlt). 4. After injuries, especially contusions, a keratitis profunda not infrequently develops, the peculiarity of which is its comparatively rapid course and the speedy restoration of the transparency of the cornea.

Treatment consists, locally, in the application of a bandage or of protective glasses, and the use of atropine. Moist warm compresses, dionin, and subconjunctival injections of salt [or mercury] solutions may accelerate the progress of the case provided these are well borne. After the inflammatory symptoms have run their course, irritant remedies for clearing up the opacity are indicated. The general treatment depends upon the cause that we are able to discover for the keratitis.

5. *Sclerosing Keratitis*

243. This has retained its name given it by Von Graefe, because at the spot where the infiltration is present in the cornea, dense and white opacities are left which often look like sclera. Sclerosing keratitis occurs either alone or more frequently as an accompanying symptom of scleritis (page 337). If a scleritic nodule is situated near the margin of the cornea, there develops in the adjacent portion of the latter an opacity which is situated in its deeper layers (Fig. 128). It has approximately the shape of a triangle, the base of which is situated at the corneal margin, while the rounded apex looks toward the center of the cornea and gradually merges into the transparent cornea. The opacity thus occupies a sector of the cornea, the base of which corresponds to the scleritic nodule. In many cases other dense opacities of rounded or irregular shape also develop at a distance from the corneal border, and even in the center of the cornea. The opacities are gray or grayish-yellow, and gradually increase in density until the cornea at the affected spot has become completely opaque. The surface of the cornea over the opacity is stippled but not depressed; vascularization is either altogether absent or is very slight, and, when it is present, is in the deep layers of the cornea. After the opacity has reached its maximum density, a gradual retrogressive process sets in, without ulceration having taken place at any time. The process of clearing affects the thin edge of the opacity and its apex, which looks toward the center of the cornea; the greater portion of the opacity remains permanently and becomes ultimately bluish-

white like the adjacent sclera, into which it passes without any sharp line of demarcation. At the spot where the opacity is found, therefore, it looks as if the sclera had pushed its way into the space occupied by the cornea.

Like scleritis itself, this keratitis that accompanies it shows repeated recurrences, and it may happen that in severe cases the entire cornea is sclerosed with the exception of a small area in the center.

The disease usually attacks young persons, especially females, and often occurs in both eyes. It sometimes runs a torpid course, but more often is associated with marked irritation which accompanies each recurring attack. The inflammatory symptoms are in part also caused by the complicating scleritis, and iritis. The etiology is ordinarily the same as that of scleritis, namely anæmia, chlorosis, scrofulosis, tuberculosis, hereditary syphilis, and in older patients gout and rheumatism. Often no certain cause can be found for the disease.

Locally the same remedies are indicated that have been suggested for keratitis profunda. The general treatment is directed to the cause of the disease, in case such a cause can be ascertained.

6. *Keratitis Springing from the Posterior Surface of the Cornea*

244. When the posterior surface of the cornea is not washed by the aqueous humor, as it is in the normal eye, but an exudate or tissue is brought into apposition with it, the substance of the cornea becomes cloudy. To produce this effect, however, it is necessary that the apposition should be kept up for a pretty long time. Hence this sort of opacity is not generally found in ordinary hypopyon, because the latter disappears too quickly, but is found in those more solid gray exudates which appear in the anterior chamber, particularly in scrofulous and syphilitic irido-cyclitis. Large deposits also on the posterior surface of the cornea, if they remain for a long time, usually leave behind them gray spots in the cornea. Another thing besides exudates that gives rise to this form of keratitis is the apposition of tissue to the back of the cornea, as occurs with protrusion of the iris, with cysts or other tumors of the iris that reach to the cornea, and with lenses that have prolapsed into the anterior chamber. The opacity of the cornea develops at a point corresponding to that spot at which apposition takes place, occurring, therefore, in the case of exudates, most frequently below. The surface of the cornea at this spot is dull, sometimes slightly uneven, and apparently gelatinous. The opacity is gray, and, after lasting a long time, becomes pretty dense, and is permeated by vessels which lie in the deep layers of the cornea. It never completely disappears, even after the causal lesion has been remedied.

The mode of origin of this form of keratitis is probably to be conceived of thus: Contact of the cornea with foreign tissue alters the endothelium of Descemet's membrane. This alone, according to Leber's researches,

protects the cornea from the aqueous humor. Now, if the endothelium becomes deficient, aqueous humor can penetrate into the tissue of the cornea, which consequently becomes cloudy. [See page 7.]

245. Other Forms of Non-Suppurative Keratitis.—The clinical pictures under which non-suppurative keratitis makes it appearance are exceedingly manifold. Only a certain number of them can be marshaled under fixed types, as has been done in the foregoing pages. Some, occasionally very peculiar, forms come under observation too rarely for us to be able to build up from them a typical disease picture; they cannot at present be utilized except for purposes of record as individual cases. A few rather more frequently occurring forms may be enumerated here as an appendix to those before described.

7. **Keratitis Pustuliformis Profunda.**—This is a rare condition occurring mainly in old people and chiefly in men. It is marked by the presence of yellow, puriform deposits, placed very deeply in the cornea. Sometimes a single, large, usually central, deposit occurs; more often there are several, the size of a pin's head and variously disposed. The deposits are generally surrounded by a grayish, hazy, often punctate opacity. The condition is associated with a severe iritis, which, indeed, probably constitutes its real starting point. It is also accompanied by hypopyon, deposits on Descemet's membrane and, at times, vitreous opacity. There is great pain. The deposits never break down to form ulcers. Occasionally they undergo resolution, but generally they are transformed into permanent opacities, and in bad cases flattening of the cornea may ensue. Usually but one eye is affected. The disease runs a chronic course, and treatment has very little effect. In some cases syphilis seems to be the cause; in other cases the origin is unknown; but in any event the immediate cause must be a toxin, derived from the inflamed iris and acting on the cornea from behind. This is inferred mainly from the pathological changes in the cornea, which decrease in intensity from behind forward and which in the lightest cases consist in destruction of the posterior endothelium and proliferative inflammation of the hindmost layers of the cornea. In the severer cases, Descemet's membrane and the deeper layers of the cornea are also destroyed. The condition seems frequently to result from acquired syphilis.

8. **Deep Scrofulous Infiltrates** occur in conjunctivitis eczematosa under the form of extensive gray, subsequently yellow, opacities in the middle and deep layers of the cornea. They may either proceed to suppurate, or they may go on to resorption, in which case the cornea clears up—sometimes in a surprising manner. For a more detailed account, see page 195.

Fig. 110.—Keratitis Marginalis Profunda.

The finely striate marginal zone represents the limbus, adjoining which above and on the outer and inner sides is the arc-shaped infiltrate drawn in darker shading.

9. **Keratitis Marginalis Profunda.**—This rare disease generally affects old people, and occurs for the most part in one eye only, rarely in both. There forms upon the margin of the cornea, with moderate symptoms of irritation, a gray, later grayish-yellow, or even purulent-yellow opacity, which directly adjoins the sclera and hence extends under the limbus, while on the other hand it reaches for a distance of 1 or 2 mm. into the transparent cornea (Fig. 110). This marginal zone of opacity generally embraces from one-third to one-half of the circumference of the cornea (most frequently the upper part), or in rare instances surrounds the entire cornea. The surface of the cornea over the opacity is somewhat dull, but shows no loss

of substance, and never any exfoliation of epithelium. The limbus soon pushes forward so far as to cover the opacity with its vessels. The irritative symptoms disappear in from one to two weeks, while the marginal infiltrate is transformed into a permanent gray opacity. This opacity bears a great resemblance to the arcus senilis, from which it is chiefly distinguished by its not being separated by a transparent zone from the scleral margin, but passing into the latter without any clear line of demarcation. Iritis does not occur with this affection of the cornea, nor does ulceration of the cornea, as a rule; only twice have I seen small superficial ulcers develop upon the cornea. On account of the marginal situation of the residual opacity, this form of keratitis is without danger to the sight.

10. Striate Opacity of the Cornea.—When we examine carefully an inflamed cornea with a magnifying glass, we often discover gray striæ in it. These may be short and irregular and run in the most diverse directions. This is commonly the case in keratitis profunda. At other times we see a system of parallel striæ—e. g., in the dense opacities that occur in parenchymatous keratitis jutting out from the margin of the cornea and extending toward its center. Again, striæ may occur that take a radiating direction, all emanating from a single point—e. g., from a corneal ulcer. The anatomical changes that give origin to the striæ are not always the same. It may be that cells, or fluid, thrust the fibers of the cornea apart and so pass on between them, taking a linear course. Such a thing can be effected artificially by injecting liquid through a puncture made in the cornea. In doing this we fill a system of parallel chinks (Bowman's tubes) which cross the successive corneal lamellæ at right angles.

Isolated, long, very delicate gray lines appear to be produced by turbid fluid filling one of the channels that lie in the substance of the cornea and lodge the nerves passing from the margin of the cornea to its center.

Very frequently, however, striæ are produced not by exudation, but by wrinkling. This is pre-eminently the case with *traumatic striate opacity*. This is observed after incised wounds of the cornea, and most beautifully after the cataract operation. Within the first twenty-four hours after the operation gray striæ make their appearance in the cornea, which, starting from the wound, extend sometimes as far as the opposite margin of the cornea, and are always disposed perpendicularly to the length of the wound (Fig. 111). These striæ are particularly observable in those cases in which the lips of the wound have been somewhat contused, as, for example, those in which the delivery of the lens has been difficult. They generally pass off within the first eight days, and it is only when specially pronounced that they take several weeks to disappear. Such an opacity causes no symptoms of irritation, and does not cause the least disturbance of the healing of the wound. This proves that here we are not dealing with a real inflammation. Anatomical investigation has, in fact, shown that in these cases cellular infiltration is altogether wanting, and that there is simply a dilatation of the lymph spaces of the cornea, which are distended with fluid (Becker, Laqueur, Recklinghausen). The striate opacities themselves are referable to wrinkling of Descemet's membrane, which as a result of the incision near the corneal margin has its tension relaxed in one direction but not in others (cf. Fig. 134).

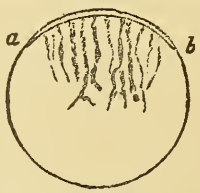

FIG. 111.—STRIATE OPACITY OF THE CORNEA AFTER A CATARACT EXTRACTION.

The cicatrix, *a b*, left by the section, lies at the upper margin of the cornea.

A wrinkling of this sort may be the cause of some of the striate opacities that are observed in a true keratitis—e. g., the radiating streaks which are often seen in ulcus serpens extending out into the transparent cornea (Hess, Schirmer).

A similar striate opacity of the cornea is sometimes observed in cases of detachment of the retina which has been treated with the pressure bandage. The eye becomes

suddenly very soft and the anterior chamber remarkably deep, and in the cornea fine gray striæ show themselves, which cross in different directions, so that the opacity looks like creased tissue paper. Here also, without doubt, folds in the cornea take part in producing this effect (Deutschmann, Nuel).

II. Injuries of the Cornea

246. Foreign Bodies in the Cornea.—The penetration of foreign bodies into the superficial layers of the cornea is among the most common of accidents. Obviously the interpalpebral area of the cornea is the part that suffers most from injuries due to foreign bodies as from injuries in general. What is most frequently observed are small particles of iron in the cornea, particularly among mechanics of a certain sort, like locksmiths, blacksmiths, iron founders, etc. These particles do not look like metallic iron, but vary from dark brown to black; for the particles of iron, which, for example, fly off when iron is being hammered, are heated by the force of the blow so that they are thrown out as sparks. Thus they become oxidized into ferroso-ferric oxide (so-called iron scale), and under this form are found in the cornea. If the fragment of iron remains sticking in the cornea, it becomes surrounded very soon by a brown ring, because it impregnates the portions of the cornea in its immediate vicinity with iron (ferric hydrate) and so turns them brown. Fragments of coal are also frequently found in the cornea—for instance, in firemen or in people after a railroad journey—and fragments of stone in stonecutters, stonebreakers, etc.

Much more infrequent, but also much more serious, are those cases in which a small foreign body has penetrated into the deep layers of the cornea.

Foreign bodies penetrating into the cornea should be removed as soon as possible (see § 846). If the foreign body is not removed in season, its expulsion by suppuration follows. An inflammatory infiltration forms about it, surrounding it in the form of a gray ring. Then the tissue of the cornea in this place breaks down, so that the foreign body becomes loose and ultimately falls out. The resulting ulcer generally becomes rapidly cleansed and heals, leaving a small opacity after it. This process of elimination takes place with marked symptoms of irritation, and especially with hyperæmia of the iris, or even with iritis, which latter makes itself evident by the formation of a hypopyon and of synechiæ. [These symptoms may persist for several days after the foreign body has been eliminated or removed. Both the symptoms and the resulting damage to the cornea are obviously greater if the foreign body has penetrated into the deeper layers, and in this case, too, there is more danger of purulent infection being carried to the deeper parts.—D.] Foreign bodies which are chemically indifferent, like powder grains or particles of stone, are often tolerated by the cornea without producing inflammation and may become permanently incorporated in it.

[For the irritation remaining after the removal or elimination of a for-

eign body, we use hot or cold applications, dionin etc., and if photophobia or iritis is present, atropine. Holocaine or acoine in oily solution or in ointment may be required to relieve pain, especially at night. Sometimes plain vaselin answers the same purpose. In many cases the patient is more comfortable if the eye is bandaged. For the treatment in cases of perforation or of deep infection, see § 291.—D.]

[*Oyster-shuckers' keratitis* is a rather rare condition caused by the impact of fine bits of oyster shell upon the eye. It is marked by its acute onset, a small, circumscribed, very white ulcer developing in the cornea (usually in its center) within twenty-four hours. This is associated with considerable photophobia and irritation. The ulcer usually shows no tendency to spread, and, when small, heals without bad result. If a large area is involved or the cornea is perforated, the eye is usually lost by infection (Randolph). —D.]

247. Solution of Continuity of the Cornea.—Superficial excoriations of the cornea, which simply produce a loss of substance in the epithelial covering, are known as *erosions*. These are among the most frequent of injuries, such as one gives himself by scratching the eye with the finger nail, with a rough cloth, a stiff leaf or twig, etc. Such an injury is commonly accompanied by pretty marked symptoms of irritation, such as photophobia, lachrymation, and especially by violent pain, Examination of the eye shows, besides the ciliary injection, a defect in the epithelium, forming an ulcer, the floor of which is perfectly transparent, so that it is only by taking the corneal reflex [or using fluorescein] that the loss of substance can be discovered. Healing generally takes place within a few days by a complete regeneration of the epithelium, starting from the edges of the epithelial defect; a permanent opacity does not remain. Quite a good deal of significance attaches to these traumatic erosions, from the fact that not infrequently they are the starting-point of an ordinary ulcer of the cornea or an ulcus serpens, especially if an opportunity is given for the production of infection. This latter is particularly apt to occur if there is present either a conjunctival trouble associated with abnormal conjunctival secretion, or a disease of the lachrymal sac.

It is worth remarking that sometimes *recurrences* of corneal erosion take place without any new injury having preceded them (Arlt). After the lesion has been to all appearances fully healed, marked symptoms of irritation set in suddenly several weeks or months afterward without known cause; and a loss of substance is again found upon the cornea in the epithelium at the site of the former injury. Such relapses may occur repeatedly. They have their cause probably in the fact that the regenerated epithelium is no longer firmly adherent to its bed (Szili), so that under the action of any insignificant cause it is again separated and cast off. This separation of the epithelium generally takes place in the form of a vesicle, which, however, ruptures so quickly that we do not get a sight of it, but only of the consequent loss of substance in the epithelium.

In this category also are to be reckoned those cases in which for months or even years after an erosion of the cornea, pain occurs when the eye is suddenly opened either in the night or on awakening in the morning. The pain passes off in a few minutes.

Then if we see the patient during the day, there is either nothing at all to be discovered in the cornea objectively or at most there is found an extremely faint cloudiness which is situated in the corneal epithelium on the spot that was injured. Yet in this case also there are probably constantly recurring, very insignificant lesions of the corneal epithelium, which are produced, at the site of the former injury, by sudden opening of the lids.

Erosions are best *treated* by applying a simple protective bandage which should be continued until the epithelium is completely regenerated. If there are marked accompanying symptoms of inflammation, which are not relieved by the bandage alone, we may instill atropine [or homatropine—the latter in 2-per-cent solution]. The recurrences require the same treatment, as long as a defect in the epithelium can be made out to exist. [In obstinate recurrent cases the bandage may have to be applied to both eyes and kept on, day and night, for from one to two weeks.—D.] After complete restoration of the epithelial covering, it is advisable, in order to prevent further recurrences, to have the cornea massaged for some time with the yellow-precipitate ointment. For pain which recurs at night we direct the patient to introduce fatty substance (e. g., boric-acid ointment) into the conjunctival sac before retiring, and, besides, advise him in case he wakes during the night to open the eyes very cautiously—possibly not until he has first wet the margin of the lids. If, in spite of this, relapses recur we obtain solid union of the epithelium to the cornea and hence also a cure, if we scrape off the epithelium wherever it is but loosely adherent to the cornea, and then paint this area with tincture of iodine.

248. *The deeper wounds* of the cornea are usually either incised or lacerated wounds. Their margins soon after the infliction of the injury become cloudy and swollen through imbibition of fluid (tears or aqueous). In irregular, lacerated wounds, this may occur over a very great area. As the wounds heal this cloudiness in large part disappears, although a dense opacity always remains along the line corresponding to the solution of continuity, and this opacity is very frequently associated with an irregular bulging of the whole cornea (giving rise to irregular astigmatism). Corneal wounds are particularly dangerous under two circumstances—i.e., when they are infected and when they perforate the cornea. In the former case a purulent keratitis develops which may give rise to extensive destruction of the cornea. In the latter case prolapse of the iris occurs, provided the wound is large enough. Moreover, the iris or the lens may be injured at the same time, and lastly there exists, as in all perforating lesions of the eyeball, the danger of an inflammation of the deep parts of the eye, produced by infection and very frequently ending in the destruction of the organ.

The *treatment* of recent wounds of the cornea demands as its prime requisite that after dropping in atropine to combat any iritis that may exist, we apply a protective bandage. If we are dealing with a perforating wound of the cornea, the greatest possible quiet on the part of the patient (rest in bed) is requisite in order to bring about a speedy and solid closure of the wound. If the iris is prolapsed, it should be so excised, after carefully separating it from the lips of the wound, that no iris remains any longer incarcerated in the wound; according to just the same principle that holds good

for prolapses of the iris of spontaneous origin. (For more precise particulars in regard to perforating wounds of the cornea, see § 291.)

The healing of *incised wounds* of the cornea takes place quickly when, as is pre-eminently the case in operation wounds, the edges are smooth and are closely applied to each other. The edges of the wound then soon become agglutinated by a mass which consists of fibrin and round cells, and which later organize into a delicate cicatrix binding the corneal lamellæ together. Bowman's and Descemet's membranes do not reunite. During the first few days the epithelium of the anterior surface of the cornea grows rapidly over the lips of the wound and down between them, this *involution of the epithelium* sometimes extending as far as the posterior layers of the cornea (Fig. 112). By reason of the permanent adhesion of the lips of the wound that takes place later, the intruding epithelium is gradually squeezed back from below up toward the surface, and the epithelial involution disappears. Sometimes, however, it is persistent. In

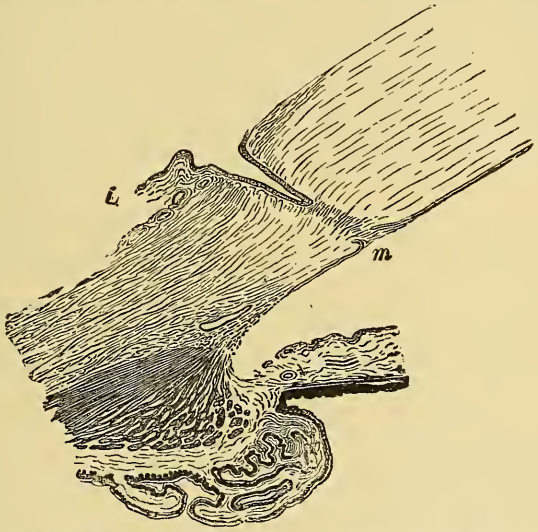

FIG. 112.—SCAR OF THE CORNEA THIRTEEN DAYS AFTER CATARACT OPERATION. Magnified 22 × 1.

The section passed obliquely through the cornea and the margin of the limbus *L*. From both edges of the wound, peripheral (limbus) and central (cornea), the epithelium has grown down into the depth of the wound, which, therefore, in the living eye appeared under the form of a smooth chink, running along the margin of the cornea. It is only in the deepest layers of the cornea that the lamellæ of the latter are united by recently formed scar tissue, which also pushes its way in between the cut edges of Descemet's membrane, *m*, which are curled forward. Since the operation was made with an iridectomy, there remains of the iris nothing but a stump, whose cut surface shows no trace of reaction.

that case the wound externally looks as though it had healed smoothly, while really only the posterior layers of the cornea are united by a firm cicatrix. Such scars may be ruptured by moderate pressure upon them; and this explains why it is that the scar left by a cataract operation sometimes—it may be years afterward—splits asunder on very slight provocation.

[To facilitate the healing of corneal wounds, Ramsay recommends that they be covered with a wafer of gelatin containing 10 per cent of collargol.] Perforating wounds, if fairly large, sometimes gape a good deal. This is especially true of flap wounds, since the flap soaks up the aqueous and hence becomes greatly swollen, so that it projects above the rest of the cornea, and may even be bent double. In such cases we may try to unite the edges of the wound in the cornea with fine sutures [painting the sutured lips with 5-per-cent tincture of iodine or with Whitehead's varnish (a solution of 1 part

of iodoform and 6 parts of compound tincture of benzoin in ether) (De Schweinitz)—D.J. Unfortunately it sometimes happens that a purulent infiltration of the cornea or, in case the sutures have extended into the anterior chamber, an irido-cyclitis is started by the sutures. More favorable are those cases in which the gaping wound in the cornea is so placed that we can pass a suture through adjacent parts of the limbus and by drawing them tightly together can approximate the lips of the wound. We may also cover in wounds of the cornea with conjunctival flaps (see § 823).

When the cornea is much lacerated the practical value of all these attempts is very questionable, even supposing that the lips of the wound heal well. The cornea remains permanently flattened and so opaque that only an inconsiderable residue of sight is left. At the same time the eye is for a long time prone to inflammation, and, besides, the danger of sympathetic ophthalmia is not altogether excluded, so that in these cases we should have done our patient a better service by enucleating at once.

249. *Contusions of the cornea,* produced by force applied to the latter either directly or through the lids, often result in an extensive opacity which occupies the central portions of the cornea, and under the magnifying glass can be resolved into delicate gray striæ interlacing in different directions. As this opacity is situated in the intermediate and the deep layers of the cornea, it has been considered under the head of keratitis profunda (page 300).

The striæ may in part be referable to wrinkling of Descemet's membrane, and be dependent upon the reduction in the intra-ocular pressure that often occurs after contusions of the cornea.

Severe contusions of the cornea may cause its *rupture* (ruptura corneæ). Such ruptures are much less commonly observed than are those of the sclera. Moreover, while ruptures of the sclera are generally pretty much alike in respect to position and direction, ruptures of the cornea follow no rule with regard to the way they run. This is due to the fact that ruptures of the sclera are ordinarily indirect, those of the cornea are direct (for explanation of these terms see page 342). In most cases the wounds are nearly rectilinear, but sometimes they look jagged, and are flap-shaped.

In new-born children there is sometimes found a uniformly dense, bluish-white opacity of the cornea which is produced by contusion of the cornea *during birth* (usually by the forceps). The opacity usually disappears altogether in the course of a few weeks.

250. Injuries of the Cornea by Caustic Agents and by Burns.—These occur simultaneously with the analogous injuries of the conjunctiva, and are produced by the same causes that these are (see page 211). In fact, in the case of such injuries of the eyeball, it is precisely the part which the cornea takes in the process that is a criterion for the prognosis—for the most harmful consequences of these injuries are the opacities which are left in the cornea. The corroded or burned cornea looks dull and opaque. The extent of the opacity depends upon the extent of the burn, but the intensity of the opacity depends upon the depth to which the corneal tissue has been destroyed. In light cases the color of the opaque cornea is gray, but in severe cases whitish. In the worst cases the cornea is all as white as porcelain, dry upon its surface, and quite insensitive; such a cornea is completely necrotic. In general it is not always easy to estimate from the character of

the injury alone how deeply the destructive process has penetrated into the cornea, and hence caution is advisable in giving the prognosis.

The injury is generally followed by sharp pain. [In some cases a temporary rise of tension may occur (see page 212).] It heals by the extrusion of the dead tissue. In the lightest cases, in which the injury has affected the epithelium only, the processes of extrusion and of healing occur very rapidly (as in the frequent cases of burns of the cornea produced by a curling iron). If the destructive process has made its way into the parenchyma of the cornea, a delimiting suppuration sets in, which leads to the separation of the eschar; the loss of substance thus produced heals, leaving a permanent opacity. If the destructive process has at any point gone through the entire thickness of the cornea, perforation of the latter develops after the separation of the eschar. The iris then prolapses, and the resulting cicatrization unites the iris to the cornea (cicatrix of the cornea with anterior synechia). In a similar way adhesions often develop between the cornea and the conjunctiva of the lids (symblepharon), provided that a loss of substance is present in the latter also. The treatment of injuries produced by burns and caustics has already received mention under the head of the analogous injuries of the conjunctiva.

Corrosion of the cornea is most frequently produced by lime, which usually gets into the eye under the form of mortar. Corrosion with lime gives a bad prognosis inasmuch as the corneal opacity produced by it has no tendency to clear up. This behavior is explained by the microscopical findings in a corneal opacity of this sort, the latter being caused not, like ordinary opacities, by scar tissue, but by the deposition of small opaque particles which consist of calcium carbonate. For this reason this form of opacity has been called a *calcareous incrustation*. To clear the cornea by dissolving the calcium carbonate, Zur Nedden advises bathing the eye, after cocainization, several times a day for half an hour at a time in a 10-per-cent solution of neutral ammonium tartrate. The more recent the case, the better the outlook for a good result is said to be. Zur Nedden advises the same treatment for clearing up lead incrustations (page 147).

[Corrosion of the cornea may also be produced by bituminous dust, which causes corneal ulcers with iritis; dimethyl sulphate fumes, which cause cloudiness of the cornea; and substances (e. g., bits of indelible pencils) containing the basic aniline dyes, which cause ulceration of the cornea, cataract and even panophthalmitis (Stephenson). The baneful effects of the aniline preparations can be averted, if a 5- or 10-per-cent solution of tannin is at once instilled (Vogt).—D.]

III. Dystrophies of the Cornea

251. The dystrophies are chronic disturbances of nutrition in the cornea which find their expression in the fact that very gradually an opacity develops in the cornea, which opacity steadily increases in the course of years. From inflammations in the cornea the dystrophies are distinguished —(1) by the absence of external evidences of inflammation, such as injection, pain, etc.; (2) by the continuously progressive course while an inflammation after reaching its acme recedes; (3) by the anatomical finding which

does not show an invasion of leucocytes into the cornea, but degenerative processes such as fatty degeneration (arcus senilis), the deposition of calcareous matter (zonular opacity of the cornea), hyaline degeneration (nodular opacity of the cornea), etc. From opacities in the narrower sense of the word (page 313), i.e., opaque spots which remain after inflammations or injuries and which are stationary, the dystrophies are distinguished by their progressive course; in many cases, also, the epithelium over the opaque spot is dull, proving that a progressive process is present.

In the case of dystrophies we have to do with degenerative processes, whose cause is either old age (arcus senilis), or disordered nutrition of the eye (zonular opacity of the cornea, alteration in old scars—page 317), or an hereditary tendency (nodular opacity of the cornea). In many cases the cause of the degeneration remains unknown.

252. Arcus Senilis.—The physiological type of dystrophies is the arcus senilis, in which fatty and hyaline degeneration is found in the cornea (page 231).

In rare cases the arcus senilis grows so broad, especially above, that it extends into the pupillary area and thus can actually cause an impairment of sight. Another pathological change which occurs very rarely in the arcus senilis is:

Senile Marginal Atrophy.[17]—This is a very indolent process occurring in old persons. The arcus senilis becomes steadily broader, while the cornea in the area of the arcus gets thinner, so as to form a gutter-shaped depression, which ultimately, yielding to the intra-ocular pressure, becomes ectatic. The peripheral margin of the furrow is sloping, the central margin forms a steep wall. Along this wall some of the corneal lamellæ are cut off short, others become fibrillated and are continuous with the lax connective tissue which covers the floor of the furrow. The lamellæ of both the floor and wall of the furrow contain droplets of fat. Bowman's membrane ceases at some distance from the central wall. Thickenings and fissures are found in Descemet's membrane, but not till after the furrowed cornea has begun to bulge.

A similar, perhaps not identical condition, associated with slight inflammatory symptoms, occurs at times in younger people.

Opacities resembling the arcus senilis are found in conjunction with affections of the limbus—e. g., with the growths of spring catarrh, with small neoplasms, and sometimes with a large pinguecula.

Fig. 113.—Zonular Opacity of the Cornea.

253. Zonular Opacity[18]—This is the most frequent of the pathological type of dystrophies. It forms a gray stripe from 3 to 5 mm. broad, which passes straight across the cornea, a little below its center. It develops with extreme slowness, occupying years in its progress, the first parts to appear being the two terminal points of the opaque stripe—that is, the portions of the opacity lying nearest the outer and inner margins of the cornea. These points are always separated from the margin of the cornea by a narrow, transparent zone. Starting from them the opacity gradually pushes its way toward the middle line, where the two parts of it unite, and thus close in the opaque zone which covers the lower half of the cornea. This zone is, accordingly, broadest and most opaque at its two extremities, these being its oldest parts (Fig. 113). On examining it pretty closely, especially with a magnifying glass, we ascertain that the opacity, which has a sharply defined outline on all sides, is composed of minute white

[17] Synonyms: Peripheral furrowing keratitis (Schmidt-Rimpler), dystrophie marginale symétrique.
[18] Synonym: Ribbon-shaped opacity of the cornea, calcareous band of the cornea.

or gray dots which lie quite superficially in the epithelium or directly beneath it; hence we usually find the surface of the cornea over the opacity roughened like shagreen or covered with minute prominences. Often in the midst of the opacity we see rounded or irregular areas which are quite clear (as shown in Fig. 113), or the opacity is traversed by fissures and chinks; and portions of the opacity may actually drop off altogether.

Zonular opacity of the cornea generally develops in eyes which have nearly or quite lost their sight in consequence of some intra-ocular affection (irido-cyclitis, glaucoma), and in this case it is practically of little significance. It is only very rarely (and then only in elderly people) that we encounter it in eyes which are otherwise perfectly sound, so that here the corneal opacity itself is the sole cause of the disturbance of vision (senile zonular opacity).

The anatomical changes which underlie zonular opacity of the cornea consist in the deposition of lime in the form of very minute granules in Bowman's membrane (Fig. 105, *b*, Fig. 114), which thus becomes opaque, white, rigid, and brittle. In the spots where the calcification is far advanced we ordinarily find new-formed connective tissue (Fig. 114) on Bowman's membrane, between the latter and the epithelium. Owing to the presence of this tissue the surface of the epithelium, *e*, becomes irregular,

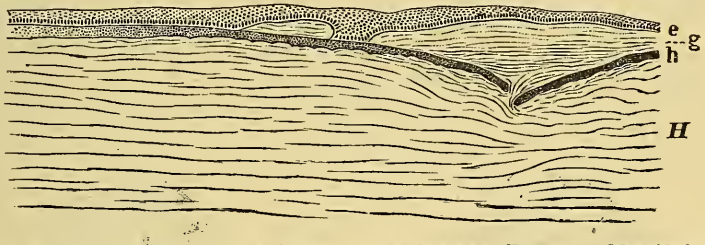

Fig. 114.—Zonular Opacity of the Cornea in an Atrophic Eyeball. Magnified 68 × 1.

Bowman's membrane, *b*, stands out because of its dark coloration which it owes to minute granules of lime deposited in it. At the left end of the drawing the deposit is slight, but increases to the right to such an extent that the individual granules can no longer be distinguished. At the same time the membrane becomes thinner and on its posterior surface uneven. Where the calcification of the membrane is the greatest, a layer of compact connective tissue, *g*, is found lying upon the latter. At the point where this connective tissue is most pronounced it pushes Bowman's membrane backward against the parenchyma of the cornea, *H*, and has caused a rupture of the brittle membrane. Through the point of rupture, connective tissue goes down into the deeper parts. The epithelium, *e*, is normal only at the spot where the calcification of Bowman's membrane is as yet slight; further along it is raised by the connective tissue and is of unequal thickness. Cf. Fig. 105.

a thing which explains the granular character of the corneal surface over the opacity. Acting in the opposite direction the connective tissue pushes Bowman's membrane backward, causing breaks in it and displacement of the broken portions. In advanced cases minute granules of lime also make their appearance in the lamellæ of the cornea lying beneath Bowman's membrane.

Zonular opacity of the cornea depends upon a disturbance of nutrition, caused by a lessened ability on the part of the cornea to withstand external injurious influences. The position and extent of the opacity correspond to the palpebral zone of the cornea—i. e., that part of it which lies exposed in the palpebral fissure even when the latter is but slightly opened. Since this form of opacity affects corneæ which have generally been made insensitive already and often opaque, too, by some antecedent disease, it must be assumed that it occurs because these corneæ are unable any longer to withstand properly the external injurious influences to which they are subjected in the region of the palpebral fissure. If such influences exert their effect for a very long time, even healthy corneæ may react to them by the production of a zonular opacity. Thus Topalanski has seen this opacity in three hat makers, into whose eyes particles of hair were constantly flying from the hare skins that they were engaged

in cutting. I myself found zonular opacities of both eyes in a physician who had blown calomel into them every day for twelve years. According to Leber, one of the injurious influences that affect the palpebral region of the cornea is evaporation: the nutrient fluid of the cornea being supposed to be richer in lime salts in cases of zonular opacity, and these salts being precipitated under the influences of evaporation.

The zonular opacity being superficially placed can readily be removed by scraping off the epithelium and the cloudy corneal layers immediately subjacent (abrasio corneæ). There is, of course, no object in doing this except in the cases where, as in the senile form, we have to do with an eye that but for the opacity would be serviceable for vision.

254. *Other forms* of dystrophies have been described, mostly under the title of chronic keratitis. But, taking it all in all, the cases are so rare that hitherto only a few typical pictures of diseases of this sort have been constructed. One such is:

Nodular Opacity of the Cornea (Groenouw). In the cornea are gray spots of rounded or irregular shape. The largest occupy the area of the pupil and sometimes coalesce to form quite large irregular figures (Figs. 115 and 116). The smaller spots lie in the periphery of the cornea and are sometimes arranged in the form of an irregular circle. The spots are quite superficial, and the cornea shows elevations corresponding to them. The unevenness of the corneal surface thus produced contributes not a little to the impairment of the sight. Between the spots the cornea shows a faint uniform cloudiness. The spots are produced by the deposition of substances insoluble in the body fluids (an acidophile, often laminated substance beneath the epithelium and a basophile granular substance in the cornea itself). The latter is the more characteristic and constant phenomenon.

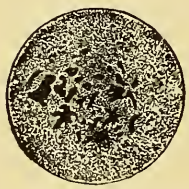

Fig. 115. Nodular Opacity of the Cornea. Fig. 116.

In the allied **Lattice-shaped Opacity** of the cornea there is present together with the spots a network of minute lines in the cornea (Haab, Dimmer). Both forms of keratitis usually begin in youth and last for life, slight inflammatory symptoms making their appearance now and then, while the spots themselves very gradually increase. They frequently occur in several members of the same family. Their etiology is unknown.

Another form is the **Dystrophia Epithelialis Corneæ.** There is found in the cornea a diffuse opacity which is most intense in the pupillary area, and which without any sharp line of demarcation fades off into the transparent margin of the cornea. The most pronounced change affects the epithelium, which is coarsely roughened and looks swollen, and in advanced cases also regularly shows vesicular elevations. These last, because they are transparent, appear when seen against the pupil as black spots, standing out upon the opacity. This proves that the opacity is situated for the most part in the epithelium; but if we scrape the latter away, we find in the uppermost layers of the corneal parenchyma a faint gray opacity which can be resolved by means of a strong glass into extremely minute gray dots. The diffuse appearance of the opacity, the marked alteration of the epithelium, and lastly, the simultaneously present insensitiveness of the cornea, correspond to the picture of a marked glaucomatous opacity of the cornea.

However, all other evidences of glaucoma are wanting, except in those not infrequent cases in which a secondary increase of tension is superadded to the change in the cornea. The disease attacks elderly people, and sometimes only one, sometimes both eyes. It begins with slight symptoms of irritation, or without any irritation at all, so that in the latter case the disturbance of vision is the only thing that calls the patient's attention to his trouble. The opacity steadily increases in the course of years, so that ultimately even counting the fingers is scarcely possible. The cause of the disease and also an effective treatment of it are unknown. [In one case of the translator's, arsenic seemed to help.]

Degenerations in Corneal Opacities.—Analogous to the dystrophies are the degenerative processes occurring in corneal opacities. Such are the amyloid, hyaline, and calcareous concretions which are deposited in old opacities and which often lead to softening, disintegration, and even perforation of the cornea (see page 316).

255. Dystrophies of the cornea which can undergo retrogression are produced by the action of chemical sources of injury; occurring, for example, in workmen who are engaged in the manufacture of aniline dyes and of naphthaline, or in dyeing with aniline colors, and who are exposed for years to the emanations from these substances. In this case there develops gradually a pretty deep, smoky-gray or brownish opacity which occupies chiefly that portion of the cornea which lies in the palpebral fissure; the surface of the cornea over the opaque parts is coarsely roughened. Inflammatory symptoms are absent. The opacity is situated in the epithelium and the most superficial layers of the cornea itself and, provided work in the manufactory is given up, disappear after rather a long time—sometimes after over a year—has elapsed.

[For other conditions of abnormal pigmentation of the cornea, either congenital or acquired (blood-staining, etc.), see page 316.]

IV. Opacities of the Cornea

256. Opacity of the cornea is a constant accompaniment of every inflammation of the latter. This recent inflammatory opacity is of a changeable nature, increasing or diminishing according to the course of the inflammation. From this variety we must distinguish those opacities which are permanent, and represent either the residua of an inflammation that has run its course, or the results of an injury. These permanent opacities, of which alone we shall treat here, we call opacities of the cornea in the narrower sense of the word. They are by far the most frequent cause of poor sight, and hence have a particular claim upon the interest of the physician.

Permanent opacities of the cornea are in most cases the consequence of a keratitis, either suppurative or non-suppurative. In the first case the tissue of the cornea, which has been destroyed by suppuration, is replaced by cicatricial tissue, and the opacities thereby produced are corneal cicatrices in the proper sense of the word. In this category also are to be counted most opacities which remain after injuries. The cornea may also have opacities remaining after a non-suppurative keratitis, either because its tissue has been so altered by the deposition of a former exudate that it does not regain its physiological transparency even after the exudate has vanished, or because the exudate itself in part becomes organized and thus

remains as new tissue in the cornea (an example is a pannus which has been transformed into connective tissue). Opacities that are situated in the epithelium only are comparatively rare, occurring, for instance, in those cases in which the epithelium as a result of constant mechanical irritation—in trichiasis—becomes thickened and hence opaque. [For non-inflammatory opacities, see page 315.]

257. The *appearance* of opacities of the cornea varies according to their degree of intensity and their age. Faint opacities appear as translucent bluish-white spots with outlines altogether hazy—*maculæ* or *nubeculæ corneæ*. [Some opacities are so faint as to be quite invisible, even by oblique illumination, and yet, on account of their diffuseness, may cause considerable reduction in sight. These can be seen with the ophthalmoscope, if we examine them by the direct method, using a + 20 D lens. The cornea then looks like ground glass, permeated with striæ, which represent the remains of former vascularization (Butler). Cf. page 316.—D.] Dense opacities are grayish-white or pure white, and are usually pretty sharply outlined; moreover, in the beginning they are apt to be traversed by vessels which afterward become fewer or disappear altogether. The surface of the opacity generally lies on a level with the adjacent healthy cornea, especially, if the opacities are small, although elevation or depression of the surface of the cornea at the site of the scar is also observed. Elevation of the surface is commonly the result of an *ectasis* of the scar. More rarely it is caused by excessive development of cicatricial tissue, or by thickening of the epithelium upon the surface of the scar. Depression of the surface of the cornea at the site of the scar occurs most frequently in the case of small scars from the incomplete filling up of the ulcer with cicatricial tissue (*facet of the cornea*). In the case of larger scars that have developed in consequence of extensive perforation of the cornea, or more rarely as a result of a severe non-purulent keratitis, a flattening of the entire cornea may follow from the retraction of the cicatricial tissue (*applanatio corneæ*). This is especially apt to occur if a plastic irido-cyclitis had been present simultaneously with the inflammation of the cornea; for, on account of this irido-cyclitis, extensive membranous exudates are deposited in the interior of the eye, which by their contraction diminish the intra-ocular pressure, and so favor flattening of the cornea.

258. Varieties of Inflammatory Opacities.—From the form and position of corneal opacities we may often gather an impression as to the variety of keratitis to which they owe their *origin*. Thus:

(*a*) Maculæ of the cornea originate from small corneal ulcers. They most frequently develop in childhood as a consequence of conjunctivitis eczematosa, and in that case are often distinguished by being situated on the margin of the cornea. Quite characteristic opacities are the elongated ones that are left by a vascular fasciculus. These after they have lasted a long time clear up in the portion that adjoins the margin of the cornea.

(*b*) Opacities which are faint and diffused, but which are nevertheless spread over the greater part of the cornea, are mostly the result of pannus or of parenchymatous keratitis. Opacities resulting from pannus are situated superficially, while those due to parenchymatous keratitis are situated in the depth of the cornea, and when examined with the magnifying glass disclose, even years after the inflammation has ceased, the presence of deep-seated vessels (Hirschberg).

(*c*) Extensive, tendinous-looking opacities, without incarceration of the iris, in which chalky-white dots are often visible, are observed after particularly severe cases of parenchymatous keratitis. Similar white dots also occur sometimes in the opacities due to pannus (see page 171); also in those resulting from corrosion by lime, in this case depending upon imbedded calcareous particles. Finally, scars with incrustation of lead are also distinguished by a sharply circumscribed, extremely white opacity (page 147).

(*d*) Marginal, crescentic or arcuate opacities are the consequence of catarrhal ulcers or of keratitis marginalis profunda; they should not be confounded with an arcus senilis.

(*e*) Marginal scars with incarceration of the iris form after perforating ulcers in conjunctivitis eczematosa. They are round, often consisting of a thinner, dark center (the incarcerated iris) surrounded by a white cicatricial ring (Fig. 118). They lie so far peripherally as often to extend into the limbus, and on account of this peripheral situation are associated with a particularly marked displacement of the pupil (Fig. 92).

(*f*) Large, dense scars with inclusion of the iris, which often occupy the whole cornea except a narrow rim about the margin, are most frequently produced by an ulcus serpens or by gonorrhœal conjunctivitis. The same sort of extensive cicatrices also occur after keratomalacia, diphtheria, and burns; in the last two cases scars upon the conjunctiva are never wanting, and conduce to the correct diagnosis.

(*g*) Sharply defined punctate or striate scars are the result of traumatism, whether effected by accident or by design (operation).

(*h*) Dense, white scars, which occupy the lowermost part of the cornea and terminate above in an almost horizontal border, are caused by keratitis e lagophthalmo. Sometimes we see men in whom such scars are present in both eyes. In this case the scars are usually the consequence of some severe disease, in which a condition of somnolence and a resulting imperfect closure of the lids were present and lasted for some time.

(*i*) Opacities in the lowermost part of the cornea having the shape of a triangle with its apex directed upward are the result of a parenchymatous keratitis which, contrary to rule, has become localized in the lower half of the cornea, or they are due to the deposition of an exudate upon the posterior corneal surface.

(*j*) Small, bluish-white opacities which are situated at the margin of the cornea and project into the transparent part of it under the form of obtuse-angled triangles, are the residua of a sclerosing keratitis.

Sclerosis of the Cornea.—By this is denoted a dense opacification and milk-white coloration of the cornea, developing without any precedent ulceration and therefore not consisting of scar tissue. It may be produced by a peculiar transformation of a pannus (see page 170) or follow deep inflammation of the cornea such as parenchymatous or sclerosing keratitis. Sclerosis, however, also occurs as a sclerotic degeneration of the cornea and then most frequently as a result of glaucoma. It is then caused either by the deposition of new-formed tissue on the surface of the cornea or by a considerable swelling and disintegration of the corneal lamellæ themselves. Sclerosis of the cornea is incurable.

259. Non-Inflammatory Opacities.—*Pressure opacity* of the cornea is the form of opacity which develops in connection with an elevation of the intra-ocular tension. It is a diffuse, smoky opacity, which is most marked in the center of the cornea and

gradually diminishes toward its margin. That it is not of inflammatory nature is proved by the fact that, after the disappearance of the rise in tension, it very soon—often in less than an hour—vanishes completely, which would not be possible if it depended upon an inflammatory infiltration of the cornea. In fact, the pressure opacity is simply an œdema of the cornea, which is situated mainly in the epithelium, and which is capable of rapid subsidence (see Fig. 217).

Likewise of non-inflammatory origin is the traumatic striate opacity of the cornea (page 303).

Congenital Opacities of the cornea, although rare, do occur, some being of inflammatory, some of non-inflammatory, origin. The former are caused by a fetal keratitis or by crushing of the cornea during birth. The latter are relatively more frequent, and are found along with other congenital eye anomalies. *Embryontoxon* is the name of a congenital opacity which in shape and appearance is like a gerontoxon. [Melanosis corneæ (see below) is another form of congenital opacity.]

Pigmentation of the Cornea.—A congenital form of corneal pigmentation is *melanosis corneæ*. This occupies the central portion of the cornea, and lies in its deepest layers. It is usually present in both eyes and symmetrically in the two (Krukenberg, Stock, and others). Differing from this is the acquired pigmentation of the most posterior layers of the cornea, which occasionally develops when the anterior chamber has been obliterated for some time and meanwhile pigment has got into the cornea from the apposed iris. Again, by a way analogous to that in which pigmented precipitates are formed, iris pigment may be cast on the posterior surface of the cornea in iridocyclitis and when operations are performed in the anterior chamber. Pigmentation of the cornea is also produced by aniline dyes, etc. (see page 313).

A peculiar sort of opacity and coloration (*blood-staining*) develops when the anterior chamber remains for some time filled with blood. Then the cornea, because it takes up the coloring matter of the blood, assumes an intense reddish, brownish, or brownish-green color, and at the same time becomes so opaque that the parts beneath can no longer be distinguished through it. It gradually clears again, beginning at the edge, but only in rare cases becomes once more perfectly transparent, and then only after months or years have elapsed. In that stage in which the central brown portion is surrounded by a narrow marginal zone which has regained its transparency, it looks as if a brown-colored crystalline lens had prolapsed into the anterior chamber and was lying behind the cornea (Vossius, Treacher Collins, etc.).

260. Metamorphoses of Corneal Opacities.—It often happens that faint scars dating from childhood no longer appear in adult life under the guise of a continuous opacity, but are traversed by clear striæ, interlacing in all directions and thus dividing the opacity into small separate areas; in other cases the transparent striæ follow the branching of vessels which had been present in the scar when it was still recent (Fig. 117). This peculiar aspect of an opacity always indicates that the latter has lasted a very long time. The explanation of it probably is that in the interstitial growth of the cornea new-formed transparent fibers develop between the old opaque ones. It may also happen that scars, originally flat, later become ectatic. Markedly ectatic scars, the most prominent point of which is but incompletely covered by the lids, not infrequently display at this point a xerotic condition of the epithelium, which looks dry and epidermoid. In old, dense scars, yellow spots sometimes develop, caused by the deposition of concretions of a hyaline or amyloid substance. Small, chalky-white specks imbedded in the scars are to be referred to a deposition of lime. In fact, small calcareous plates are frequently thus formed, which, when they become loose, can be picked off with a

Fig. 117.—Scar of the Cornea with Striate Clearing.

forceps. In these cases we have to do with different varieties of retrograde metamorphosis, which are referable to an insufficiency of nutrition of the dense cicatricial tissue (dystrophies, see page 309). Such processes may give rise to the softening and ulcerative disintegration of old scars—so-called atheromatous ulcers, which not infrequently induce perforation (page 261).

261. With many scars *incarceration of the iris* occurs. This is a proof that there has been an antecedent perforation of the cornea; hence such scars are always very opaque. It is important to determine in any special case whether a scar of the cornea is or is not connected with the iris, since an incarceration of the iris may entail serious consequences. We recognize the presence of such an anterior synechia by the displacement of the pupil toward the site of the incarceration, and furthermore by the unequal depth of the anterior chamber, which is always shallower near the place where the iris is adherent. In many cases, also, the dark color of the cicatrix gives evidence of the incarcerated iris, the pigment of which shows through the cicatricial tissue (Fig. 118).

Fig. 118.

Fig. 119.

Fig. 118.—Scar of the Cornea with Anterior Synechia. Produced by an ulcer in the course of a conjunctivitis eczematosa. The dark central portion of the scar is surrounded by a white ring. The pupil is displaced toward the scar.

Fig. 119.—Section Through a Scar with Incarceration of the Iris. The iris runs from the ciliary body to the scar, and in this part of its course is greatly thinned and is adherent to the posterior surface of the cornea, so that the sinus of the chamber is obliterated, a condition which may subsequently lead to increase of tension. In the scar itself the iris is thick and puckered. On the posterior surface it is covered with the retinal pigment layer, on its anterior merely with epithelium, so that if the latter were removed, the tissue of the iris would be directly exposed. Scar tissue, *n n*1, is found only between the iris and the edge of what was formerly the perforation opening. It fills up the space between the two which is triangular in cross section. It is this scar tissue that corresponds to the white ring which in Fig. 118 surrounds the dark incarcerated iris.

The union between iris and cicatrix is often confined to quite a small spot, so small, sometimes, that only a very fine filament rises from the iris and passes over to the scar in the cornea. At other times, on the contrary, broad adhesions exist, and there may even be an incarceration of the entire pupillary margin of the iris in the cicatrix (Figs. 122 and 123).

Opacities that are produced by the deposition of an exudate sometimes exhibit an adhesion of the iris to the cornea, and hence belong to those rare cases in which an *anterior synechia exists without a preliminary perforation* of the cornea. The iris is drawn up to the posterior surface of the cornea by the exudate, while the latter is contracting and undergoing organization, and becomes fixed there. In a similar way anterior synechia without preliminary perforation of the cornea is observed in those cases in which the iris has been pushed forward as far as the posterior surface of the cornea and kept there for some time. The iris in such cases becomes agglutinated to the cornea

in spots, and if it afterward, either spontaneously or as the result of an iridectomy, returns to its normal position, these agglutinated parts of it remain attached to the cornea. We then either find the iris extensively adherent to the cornea, or one or two tags arise from it, whose apices are inserted into the posterior corneal surface. The same thing may take place if the anterior chamber has been effaced for some length of time, so that iris and cornea have been directly in contact with each other.

262. The question whether a scar of the cornea is or is not connected with the iris is of the greatest significance for the future of the eye. For incarceration of the iris entails two dangers—increase of tension and purulent inflammation of the interior of the eye.

With regard to *increase of tension* we may stand by the rules set forth in the following plan:

- Scars without incarceration of the iris
  - Flat: no increase of tension.
  - Ectatic (keratectasia): increase of tension frequent.
- Scars with incarceration of the iris
  - Flat
    - Pupillary margin partly free: no increase of tension.
    - Pupillary margin totally incarcerated: increase of tension always occurs.
  - Ectatic staphyloma: increase of tension always occurs.

In the two cases last given, increase of tension is absent if there is a counteracting influence afforded by other changes which act to diminish the tension (e.g., fistula of the cornea, shrinking exudates in the vitreous). Unless aid is soon given by means of operation an eye affected with increase of tension is lost.[19]

Purulent inflammation often sets in quite suddenly and unexpectedly in old scars with incarceration of the iris. If an irido-cyclitis with hypopyon is present an actual panophthalmitis may develop. This inflammation is referable to a slight lesion of the epithelium which covers the scar. Any defect in epithelium makes it possible for bacteria to enter a tissue. If the epithelial defect is found in a normal cornea or in a scar of the cornea which is not connected with the iris, an ulcer in the cornea or in the scar is produced by the infection. If, however, in the case of an incarceration of the iris the imbedded iris lies beneath the epithelium (Fig. 119) the bacteria as they enter get into the iris directly, and pass back in this to the ciliary body and set up a purulent irido-cyclitis. [According to some, however, it is not necessary to have incarceration of the iris in order to produce this result. If the scar is associated with a defect of Descemet's membrane even a superficial purulent infiltration in the tissue over this defect may give rise to a peculiarly rapid destructive inflammation of the eye.—D.] The danger of this "late infection" of old scars is the greater in proportion as the layer of scar tissue which covers the incarcerated iris is thinner, a thing which can be recognized from the way in which the dark color of the pigmented iris shows through. The danger is greater in ectatic than in flat scars, because

[19] An understanding of the different ways in which the tension of the eye behaves is obtained from the teachings on the subject of seclusio pupillæ (§ 348) and the anatomical causes of glaucoma (§§ 453 and 461).

the former are generally thinner and because owing to their prominence the epithelium on their summit is more exposed to the effect of slight injuries.

263. Disturbance of Vision Produced by Opacities of the Cornea.—Every opacity which falls wholly or in part within the pupillary region of the cornea results in disturbance of vision; for the sum of the incident rays at the site of the opacity, instead of being all permitted to pass through the cornea, is divided into two parts; one part is absorbed by the scar or is reflected off from it; the other part penetrates through it into the eye. The relation between these two parts depends upon the density of the opacity; the denser it is, the more numerous are the reflected, the less numerous the transmitted, rays. Hence the cicatrix does harm by cutting off light. To be sure, this factor becomes a matter of serious consideration only in very dense opacities since we are able to see with very much less light than we usually get. Thus we see through a stenopæic slit scarcely less clearly—and if affected with an error of refraction even more clearly (§ 757)—than with the naked eye, although the slit allows but little light to pass. So, also, people with abnormally contracted pupils are still able to see with perfect distinctness. The real cause of the disturbance of vision produced by corneal opacities is not, therefore, the cutting off of light, but rather the *scattering* (*diffusion*) of light (see page 21). For, rays passing through a turbid medium, such as a cloudy cornea, are not regularly refracted, but are scattered in all directions, just as if they emanated from the turbid stratum itself. Thus, when a physicist requires a uniform illumination, he makes the light from a luminous body pass through a ground glass plate or through oiled paper, substances which may then be considered to act as self-luminous bodies themselves. The diffusion of the rays is the more complete the denser the opacity.

With respect to the density and the extent of the opacity, the following cases are possible: 1. A *dense* opacity occupies the whole pupillary region of the cornea. Then all the light that issues through the scar is diffused; no image at all of external objects is formed upon the retina, and therefore there cannot be qualitative but only quantitative vision. 2. A *faint* opacity covers the whole pupillary area. In this case the diffusion of light is not complete. A part of the rays is refracted, although not quite regularly; another part is diffused. Hence there are retinal images formed, which are, however, indistinct; and, besides, there is much diffused light. 3. *Only a part* of the pupillary region is taken up by the opacity, while the remaining part is normally transparent. Then distinct retinal images are produced by means of the latter clear portion, but at the same time much diffused light is thrown into the interior of the eye by means of the clouded portion. Hence in this case, also, vision is disturbed, and that by the dazzling which the diffused light causes.

To the disturbance of vision produced by diffusion there is often added that caused by the irregular curvature of the corneal surface, which is so

frequently present at the site of opacity. There is thus produced that refractive condition which is designated by the name of irregular astigmatism (see § 790). If the opacity of the cornea corresponds to a flattening of the surface, as in facets of the cornea, this spot refracts less strongly and is hypermetropic; if the cornea is bulged forward at the site of the opacity, as in the case of ectasiæ, excessive refraction, and hence myopia, are produced. In ectatic cicatrices of the cornea the abnormality of curvature is not confined to the scar, but extends to the neighboring transparent portion of the cornea also, so that, as a rule, no portion of the cornea retains its normal curvature. In consequence of the irregular astigmatism, objects appear indistinct, distorted, and often also double or multiple.

The disturbance of vision produced by an opacity of the cornea often entails still other *indirect results.* Among these are strabismus, nystagmus, and myopia. The last named is in many cases only apparent. The patient with corneal opacities brings minute objects unusually close to his eye, in order to make their retinal images as large as possible, and so in a measure compensate for their indistinctness. Nevertheless, elongation of the axis of the eye—i.e., true myopia—may ultimately develop in consequence of the great accommodation and convergence necessitated through such an excessive approximation of objects. [In regard to this see § 773.]

264. Treatment.—The chief task that this has to attend to is to improve the sight. The means employed for this purpose are as follows:

(*a*) *Clearing up of the Opacity.*—In the case of every opacity of recent date we must first try to clear it up as much as possible by the application of irritants (see page 266). [As Pyle has pointed out, it is particularly important to do this in the case of young children—first, because we are much more likely to succeed in clearing up opacities in them than in adults (see page 256); second, because the indirect results of the opacities, such as myopia (see above), are particularly apt to develop and advance in childhood; and, third, because if such indirect results have already begun, it is much easier to check them at the outset than later.—D.]

In older opacities, which cannot be cleared up any further by medicinal means, the next step apparently would be to render the cornea transparent once more by excising the opaque layers with a knife. Such attempts, however, have resulted unsuccessfully, for the loss of substance resulting from the excision of the opacity heals again with the formation of cicatricial tissue—that is, with the formation of an opacity, just as before. Removal of opacities by operation is indicated only when they are situated in the epithelium, since losses of epithelium are made good by normal transparent epithelium. Cases in which removal of the epithelium—*abrasio corneæ*—is indicated, are those in which the epithelium has been thickened by mechanical irritation, as in trichiasis; also in those in which lead, lime, or

grains of powder are imbedded in the epithelium, and finally in some cases of zonular opacity of the cornea.

(*b*) *The optical aids* that may be employed for improving the sight are glasses and the stenopæic aperture. The object of the latter is to bring nothing but the transparent part of the cornea into use for vision, and to exclude the portion bearing the opacity, by which means the dazzling due to diffusion is prevented. Glasses may sometimes be of advantage when the opacity is complicated with changes in the curvature of the cornea.

(*c*) *Displacement of the pupil* by means of iridectomy (after the method of Beer) is generally the only means of restoring sight in the case of dense opacities which entirely conceal the pupil. Iridectomy is also indicated when it is not a question of producing an optical improvement, but of checking the injurious consequences that many scars cause, such as increase of tension and late infection. (For the indications and the method of performing this operation, see §§ 853–857.)

In large and very white scars of the cornea it is often desirable to do away with the disfigurement that they produce. For accomplishing this purpose, *tattooing of the cornea* is of service (see § 852 for method of performing and indications).

In some cases, especially for removing protruding or fistulous scars, transplantation of the cornea (§ 851) may be tried.

For clearing up old opacities, especially those produced by parenchymatous keratitis, *electricity* has done me good service in some cases. The positive pole of a constant-current battery is placed on the temple or the neck, while the negative pole is applied to the previously cocainized cornea. The negative pole consists of a solid cylinder of silver, 7 mm. in diameter. This is surrounded by an insulating envelope of caoutchouc, the only portion exposed being the surface at its end, which is concave so as to fit the surface of the cornea. Contact between the electrode and the cornea is effected by a drop of mercury, which readily adheres to the concave surface of the silver. The current intensity employed is 0.2 to 0.5 milliampères (Alleman).

V. Ectasiæ of the Cornea

265. Just as in the case of opacities, so also in the case of ectasiæ of the cornea, we must first of all distinguish whether they have been produced by inflammation or not. On the basis of this distinction we make the following subdivision of ectasiæ of the cornea:

Ectasiæ of inflammatory origin . . . . { Staphyloma, Keratectasia.

Ectasiæ of non-inflammatory origin { Keratoconus, Keratoglobus.

By many authors the expression staphyloma corneæ is employed in a broader sense, and all ectasiæ of the cornea are designated under this name. In that case a further division of the term is made by distinguishing the transparent ectasiæ of the cornea, keratoconus and keratoglobus, under the name of staphyloma pellucidum, from cicatricial staphylomata and from keratectasiæ.

1. *Staphyloma of the Cornea*

266. Symptoms.—A staphyloma is a protuberant scar originating in a prolapse of the iris, and wholly or in part replacing the cornea. We distinguish accordingly between total and partial staphylomata. In *total staphyloma* there is found in place of the cornea an opaque, protuberant cicatrix, the base of which is encircled by the margin of the sclera or by the very outermost rim of the cornea, which may still be preserved. In one series of cases the protuberant cornea has the form of a cone (staphyloma totale conicum). In conical staphyloma the protuberance starting from the margin of the sclera slopes gradually up to its apex (Fig. 124). In other cases, however, the protuberance is hemispherical (staphyloma totale sphæricum), and its walls, rising abruptly from the sclera or even overhanging it, are sharply demarcated from the latter (Fig. 121). The spherical is more frequent than the conical form in total staphyloma. Many spherical staphylomata, above all those of recent date, have such a very thin wall that the layer of black pigment (Fig. 121, *i*) on its posterior surface is seen through it, shining with a bluish luster. Such staphylomata accordingly form a slate-colored or bluish-black hemisphere, which in form and color has a certain resemblance to a blue grape, whence the name staphyloma (σταφυλή, a bunch of grapes). Afterward thickening of the wall of the staphyloma occurs. If this takes place first under the form of separate, stout bands, by which the surface of the staphyloma is constricted in spots somewhat after the fashion of a blackberry, what is called staphyloma racemosum is produced. Old staphylomata have for the most part a thick white wall, in which usually one or two dark spots may be observed, resulting either from a deposition of pigment

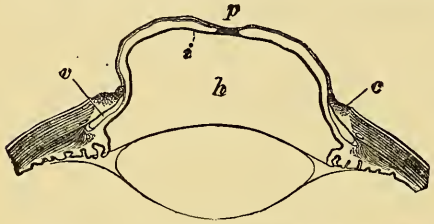

Fig. 120.—Total Prolapse of the Iris.

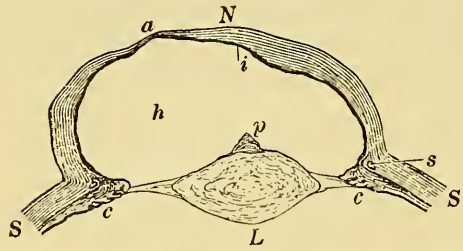

Fig. 121.—Total Spherical Staphyloma of the Cornea with Consecutive Elevation of Tension, Originating from the Total Prolapse of the Iris Represented in Fig. 120.

Out of the iris, *i* (Fig. 120), which has been thinned out in consequence of its protrusion, a thick cicatrix, *N*, has grown, in which a thin spot, *a*, corresponds to what was once the pupil (*p*, Fig. 120), while on the posterior surface of the cicatrix the retinal pigment of the iris remains as a black coating, *i*. The staphyloma is apparently directly continuous with the sclera, *S*, from which it is separated only by Schlemm's canal, *s*. For, on account of the elevation of tension, the periphery of the iris has been pressed against the cornea so that the anterior chamber (*v*, Fig. 120) has disappeared, and the iris and the marginal portions of the cornea are fused into a single mass which has been incorporated in the staphyloma. As a comparison of the two figures shows, the corneo-scleral margin has become distended. Because of this, and also because of the shrinking of the lens, *L*, the zonula has become tightly stretched, and has drawn the atrophic ciliary processes, *c*, inward. The lens is cataractous, shrunken, and has at its anterior pole a pyramidal cataract, *p*.

or from localized thinning (Fig. 121, *a*). Staphylomata are generally traversed by one or two pretty large vessels originating from the conjunctiva. On account of the opaqueness of the staphyloma, nothing is to be seen of the deeper parts of the eye. The iris is all taken up into the staphyloma—that is, all of it except its extreme periphery, which is so closely applied to the posterior surface of what remains of the marginal portion of the cornea that there is no longer any anterior chamber.

A *partial staphyloma* occupies only a portion of the cornea. It rises as a white prominence, usually in the form of a cone (staphyloma partiale conicum); a spherical protuberance (staphyloma partiale sphæricum) is pretty rare in the case of partial staphylomata. The relation here, therefore, is the reverse of what it is in the case of total staphyloma. A partial staphyloma usually extends in one direction as far as the margin of the cornea, while in the other direction there is a portion of the cornea of varying extent which is still left, and which, moreover, is generally transparent, so that the iris can be recognized behind it. The iris is drawn forward to the staphyloma, so that the pupil is displaced toward the latter and often partly concealed by it. Indeed, the pupil may be closed up altogether if the whole pupillary margin of the iris is incorporated in the staphyloma (as is the rule in the case of total staphyloma).

267. Anatomy of Staphyloma of the Cornea.—The wall of a staphyloma consists of a dense, tough cicatricial tissue, which is traversed by a few vessels, and often contains pigment imbedded in it. The thickness of the wall differs greatly; it varies from the thickness of a sheet of paper to a thickness three times as great as that of the normal cornea, and more. Very thick staphylomata are often as hard as cartilage, and when ablation is performed can scarcely be cut through. Thick and thin spots frequently occur in the wall of the same staphyloma (Figs. 121 and 124). The anterior surface of the staphyloma is covered by a thick, irregular layer of epithelium, sometimes containing epithelial pearls. The posterior surface is frequently uneven, on account of the inequality in thickness of the wall. It is covered by a coating of black pigment (Fig. 121, *i*, and Fig. 124), which is nothing but the retinal pigment layer of the iris. As, however, this has to be distributed over such a large surface, it is rarefied, so that the epithelial coating shows numerous gaps, and in the center of the staphyloma, corresponding to what was once the pupil, it is often entirely wanting. Through staphylomata with thin walls light can be passed by means of focal illumination, and in this way the pigment layer may be demonstrated in the living eye. Why Bowman's membrane should be wanting on the anterior surface of the staphyloma, and Descemet's membrane on its posterior surface, is self-evident since the staphyloma is not cicatrized cornea, but is iris, and represents a spot at which the cornea has been destroyed. It is only on the sloping sides and the edges of the staphyloma, which are formed of the remains of the cornea, that both these membranes can still be demonstrated. This is more the case in a conical than in a spherical staphyloma. A spherical staphyloma, in fact, is produced in cases where the sides of the perforation shelve off abruptly, so that the cornea even in the immediate vicinity of the opening has its normal thickness. In this case, when the prolapsed iris is driven out into the opening, the adjoining corneal tissue takes little or no part in it. The sides of the prolapse rise at right angles to the adjoining corneal surface, and thus a spherical staphyloma is formed. A staphyloma

of the sort readily develops when, as in Fig. 120, only a narrow marginal rim of cornea is left. This rim then is afterward carried forward under the influence of the intraocular pressure, while the sclera being firm offers resistance, so that now there is a furrow formed at the corneo-scleral junction (Fig. 121). The latter is particularly apt to develop such a furrow, since even under normal conditions it forms a re-entrant angle. A conical staphyloma, on the other hand, is the result of a less extensive perforation (Fig. 122), in which the portions of cornea forming the walls of the aperture taper down toward the latter, and on account of their thinness are driven forward at the same time with the prolapsed iris. The fully developed ectasis, therefore, consists only in its

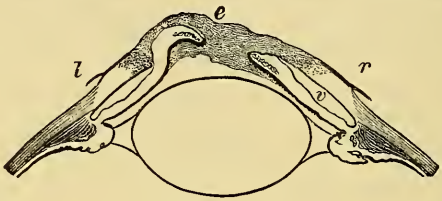

Fig. 122.

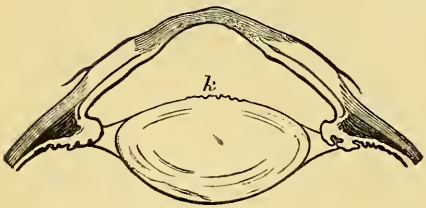

Fig. 123.

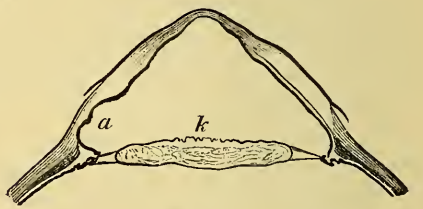

Fig. 124.

Figs. 122–124.—Development of a Total Conical Staphyloma. Magnified 3 × 1.

Fig. 122.—A large ulcer has destroyed the central portions of the cornea. Into the aperture left by the perforation projects the iris; on the right side, *r*, presenting only by its pupillary border, while on the left side, *l*, it forms a real prolapse. A gray mass, *e*, composed of coagulated fibrin, pus corpuscles, and detritus, fills the perforation and covers the exposed iris. The anterior chamber, *v*, is very shallow, and nevertheless the posterior chamber has become shallower as well, because the lens is pushed forward. Moreover, the latter, owing to the relaxation of the zonula, has an increased curvature.

Fig. 123.—In place of the prolapsed iris a somewhat protuberant scar has developed, which is thinner than the cornea, and in which is included the entire pupillary margin of the iris. In consequence of this seclusio pupillæ the whole iris has been pushed forward right up against the cornea, so that the anterior chamber has disappeared and the posterior chamber has become correspondingly deeper. The lens has re-acquired its normal shape, and shows beginning opacification of its cortical layers and wrinkling of the anterior capsule in the region, *k*, formerly occupied by the pupil.

Fig. 124.—The increase of tension that has set in as a result of the seclusio pupillæ has produced a uniform protrusion of the marginal portions of the cornea, which now form the sloping sides of the conical protuberance, and which are not separated by any sharp line of demarcation from the central cicatrix. On the right side of the drawing the iris can still be made out, although fast adherent to the cornea and greatly atrophied. On the left side nothing is left of the iris but the retinal pigment layer coating the posterior wall of the staphyloma. The wall of the staphyloma shows various protuberances caused by the constant increase in tension. The largest of these, *a*, situated directly in front of the ciliary body, represents a beginning intercalary staphyloma. The posterior chamber is very deep, the ciliary body, owing to atrophy, is flattened out, and the lens is shrunken, flat like a cake, and presents a capsular thickening, *k*.

central part of old prolapsed iris, its lateral slopes being in large part formed of the tapering, protruded cornea (Fig. 124). For this reason the ectasis does not rise abruptly from the cornea, but presents a gradual slope from its margin up, so that a conical shape is thus given it.

In staphyloma the *iris* persists as an independent structure only in cases in which at least a part of the pupil has remained free, so that the anterior and posterior chambers can communicate through it. If, on the other hand, the entire pupillary border has been incorporated in the cicatrix (seclusio pupillæ), increase of tension sets in, and as the iris is consequently pushed forward right up to the cornea (Fig. 123), the shallow anterior chamber, which was present at the outset (Figs. 120 and 122, *v*), disappears.

Then the iris becomes more and more intimately adherent to the cornea, and becomes constantly thinner and thinner as a result of atrophy, so that at last scarcely anything of it but the pigment layer remains to cover the posterior surface of the staphyloma (Fig. 124). In such cases, even when the marginal portions of the cornea are still somewhat transparent, the performance of an iridectomy has become technically impossible.

As the anterior chamber becomes shallower the posterior becomes deeper; in total staphylomata, the whole large space between the posterior surface of the staphyloma and the lens is to be looked upon as the posterior chamber (Figs. 121 and 124).

The *ciliary body* suffers mainly on account of the increase in tension, which causes it to atrophy, especially if an ectasis of the sclera develops in the ciliary region (staphyloma ciliare). Furthermore, the ciliary processes are sometimes very strongly pulled upon by the fibers of the zonula, and are thus elongated (*c*, Fig. 121).

The *lens* very frequently suffers changes in the case of staphylomata. In total staphylomata it is often entirely wanting, because it has been discharged from the eye through the pupil at the time when a large perforation existed. If the lens is still present it frequently shows alterations of position, being tilted in consequence of the unequal bulging of the staphyloma. Sometimes we find it partially adherent to the staphyloma, or it vibrates with the movements of the eye, because of the atrophy of the stretched zonula of Zinn. These alterations in the lens favor the development of an increase in tension; for this reason, after performing incision or ablation of the staphyloma, we remove the lens from the eye. Very frequently the lens is rendered opaque either in toto or only at its anterior pole (anterior polar cataract, *p*, Fig. 121). In some few cases we may find the lens greatly diminished in size, or even shrunken into a mere membrane (Fig. 124).

The *deeper parts* of the eye also suffer from the increase in tension. Excavation of the optic nerve, atrophy of the retina and chorioid, and fluidity of the vitreous thus develop.

268. Etiology.—Staphyloma constitutes the final outcome of the corneal ulcer with perforation, and is nothing but the prolapsed iris which has become protruded and transformed into cicatricial tissue. The protrusion may be primary or secondary in its development.

(*a*) *A primary protrusion* is produced in the following way: After perforation of the cornea has occurred, the iris becomes prolapsed and bulges forward because it is too weak to sustain even the normal intra-ocular pressure. The cicatrization which follows, and which in favorable cases produces flattening of the prolapse, cannot in unfavorable cases do away with the protrusion. On the contrary, the prolapsed iris remains protruded at the same time that it is gradually converted into cicatricial tissue; it becomes consolidated while still in a position of protrusion (thus from the prolapse of iris in Fig. 120 is formed the staphyloma represented in Fig. 121). A total or partial staphyloma develops according as a total or partial prolapse of the iris has existed. The causes which oppose the conversion of a prolapse of the iris into a flat scar and which favor the formation of a staphyloma are chiefly two: The first is large size of the perforation. In very small perforations there is no development whatever of staphyloma; and the larger the perforation, the more likely is it that a staphyloma will

develop. The second cause is improper behavior on the part of the patient. In this regard the chief factors to be considered are, in adults, great physical exertion; in children, crying, and also squeezing together of the lids; and, in both, great straining at stool. The temporary increase of tension induced by this means distends the newly formed and yielding cicatricial tissue; but as the latter has no elasticity, it does not return to its former dimensions after the elevation of tension has disappeared, but remains permanently protruded. In that case, however, a permanent protrusion leads sooner or later to permanent increase of tension which now does not have its cause in external influences but in the obliteration of the sinus of the chamber produced by the incarceration of the iris.

(*b*) We speak of *a secondary protrusion* when a prolapse of the iris first heals with the formation of a flat scar, which latter afterwards bulges out again. The cause of this is frequently supplied by the same injurious influences that have been enumerated above—e. g., by the too early resumption of work by a patient with a recently cicatrized corneal ulcer. The recent cicatrix is still too yielding to offer a proper resistance to the repeated though transitory elevations of intra-ocular pressure, and so becomes gradually distended. But in any case a protrusion is sure to occur whenever the inclusion of the iris is of such a kind that by it there are produced a blocking of the channels of outflow (page 15) and consequently a permanent increase of intra-ocular tension. This is very often the case in partial inclusion of the iris and very regularly so in inclusion of the whole pupillary margin (seclusio pupillæ, Figs. 122–124).

In individual cases a primary and secondary protrusion cannot always be sharply differentiated. A very frequent case is that in which the iris, being extensively prolapsed, is protruded from the start; the intra-ocular pressure in this case being at first normal and afterwards gradually rising, so that the protrusion then increases still more.

A staphyloma, accordingly, in its origin is not a bulging of the corneal tissue, but of the iris. It develops from a prolapse of the iris, which is converted into cicatricial tissue—that is, it develops just at the spot where the cornea no longer exists. It would therefore be more correct to speak of staphyloma iridis. In fact, the transition from prolapse of iris to staphyloma is altogether gradual, so that at a certain stage of its development the protrusion in the eye may be equally well denoted as an old prolapse of the iris or as a recent staphyloma.

269. Consequences of Staphyloma of the Cornea.—The *sight* is always diminished. In total staphyloma it is reduced to the mere ability to distinguish between light and darkness. In partial staphyloma the degree of sight depends upon the character of the part of the cornea that is still preserved, and also upon the position of the pupil. Even in the most favorable case,

in which a part of the pupil happens still to lie behind perfectly transparent cornea, the sight is considerably reduced by the irregular curvature which is present not only at the site of the staphyloma itself, but to a less degree over the whole cornea. Large staphylomata produce a very conspicuous disfigurement. They also cause trouble by giving rise through mechanical irritation to catarrhal conditions of the conjunctiva, with increased secretion, lachrymation, etc. Closure of the lids is rendered difficult in the case of large staphylomata by the great size of the protrusion; the apex of the latter, being but incompletely covered by the lids, becomes dry (xerotic), or becomes the site of ulcers (atheromatous ulcers). Sometimes the lids are forced so much apart by the staphyloma that ectropion develops.

Staphyloma of the cornea is almost invariably accompanied by *elevation of tension.* With regard to the relation between this and the staphyloma, two sets of cases exist—i. e., the increase of tension may be the cause or the result of the staphyloma.

But when a staphyloma has once developed, elevation of tension is always present. Besides causing the increase of intra-ocular pressure that is perceptible to the touch, this elevation of tension finds its chief expression in a diminution of sight, which finally ends in complete blindness. Pain, too, is sometimes associated with the elevation of tension. Moreover, as soon as the increase of tension has set in, it gives rise to further changes both in the staphyloma and also in the whole eyeball. Thin-walled staphylomata are made to protrude farther and farther by the heightened pressure, and thus suffer an increasing attenuation of their wall, until the latter, from almost any trifling cause, ruptures at some particularly yielding spot. Opening of the staphyloma may also occur from perforation of one of those ulcers which so frequently develop at the apex of the staphyloma, either as a result of mechanical injury or because of the inadequate nutrition of the scar tissue. In either case when perforation takes place the aqueous humor is discharged in great abundance; and the staphyloma collapses, becomes smaller, and remains so for some time. But perforation of the staphyloma may also be followed by profuse intra-ocular hæmorrhage, or by severe iridocyclitis or panophthalmitis. Then the eyeball undergoes atrophy, and by this means a sort of spontaneous cure of the staphyloma takes place.

The scleral portion of the eyeball, too, when the elevation of tension has lasted a pretty long time, gives way and becomes distended, especially in young people in whom the sclera is more extensible. In this case we observe both total and partial ectasia of the sclera. In the former, the sclera becomes uniformly distended, the entire eyeball grows larger, and the sclera becomes so thin as to look bluish owing to the way in which the chorioidal pigment shines through it. In the second case, the sclera in the vicinity of the cornea bulges forward under the form of a circumscribed swelling, which appears dark from the pigment shining through it; intercalary and

ciliary staphylomata (see page 359) are developed. Very frequently general and partial ectasiæ of the sclera are found simultaneously in the same eye, which may thus grow to an enormous size.

As a rule, the *increase of tension* resulting from staphyloma develops quite gradually. But sometimes protrusion of the cicatrix and increase of tension take place simultaneously and in a sudden fashion, as the following example may illustrate: A person has had an ulcus serpens which has destroyed the cornea in its central part. Under suitable treatment the prolapsed iris is in process of transformation into a flat cicatrix. Then, one morning the patient complains of violent pain which has suddenly developed in the eye. After removing the bandage we find the eye, which the day before was almost free from irritation, now the seat of a dusky ciliary injection. The cicatrix is bulged forward in the form of a cone, and the anterior chamber is very shallow, or is altogether abolished, because the iris is pressed against the cornea. The latter looks dull, and small hæmorrhages are visible in the cicatrix or in the anterior chamber. The eye is hard, and very sensitive to the touch. An external cause for this sudden change in the course of healing is generally not discoverable.

270. Treatment.—Stress is chiefly to be laid upon *prophylaxis*. The physician who has to treat an eye with prolapse of the iris must make every endeavor to secure the production of a flat cicatrix. He should not suffer a staphyloma to develop before his very eyes. In this connection, what has been said in regard to prolapse of the iris (page 264) may be consulted. When we have succeeded in effecting the formation of a flat cicatrix we must, while the latter is still recent, take measures to keep it from bulging out again. With this end in view we should refrain from discharging the patient too soon from treatment, and particularly we must advise him to abstain for a long time from all severe physical exertion. It is often advisable before discharging the patient to perform an iridectomy, whenever possible, as by this means the subsequent development of an ectasis is most effectually counteracted.

If we have to do with a staphyloma which has already developed, our treatment must have a different object in view, according as the case in hand is a total or a partial staphyloma. With the former the sight is irreparably lost, since there is no transparent cornea left; we must hence confine ourselves to the relief of the symptoms and of the disfigurement produced by the staphyloma. With partial staphylomata, our first aim is to improve whatever sight may be left, or at least to preserve it from injury (as would be produced by increase of tension). For the methods which are employed for the cure of staphyloma and which are all of an operative character see §§ 848, 849.

It cannot be denied that, in spite of the therapeutic means at our command, the cicatrix in many cases keeps constantly bulging anew, the increase in tension returns every time and thus the eye slowly but inevitably goes blind.

2. *Keratectasia*

271. By keratectasia we understand a protrusion of the cornea, which makes its appearance after inflammation of the latter, without, however, any perforation having taken place. The protrusion, therefore, in this case consists of corneal tissue, in contradistinction to staphylomata, in which it is formed of the tissue of the iris. From ectasiæ of the cornea of non-inflammatory origin—i. e., keratoconus and keratoglobus—the inflammatory ectasia of the cornea is distinguished by the fact that the bulging portion of the cornea, in consequence of the inflammation, is opaque.

Inflammation produces protrusion of the cornea by thinning it or by softening it. It produces protrusion by *thinning* in those cases in which an ulcer of the cornea has destroyed the superficial lamellæ of the latter to such an extent that the posterior lamellæ are no longer able by themselves to offer resistance to the intra-ocular pressure (*keratectasia ex ulcere*, Fig. 125). If all the layers as far as the membrane of Descemet have been destroyed,

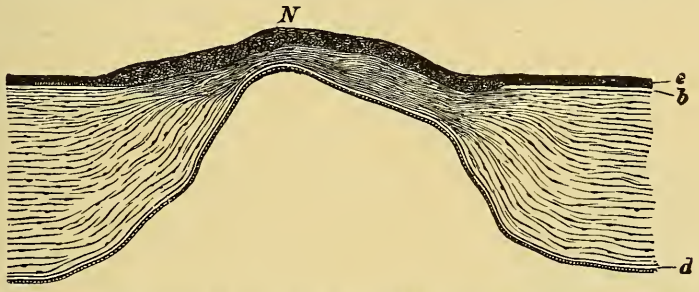

Fig. 125.—Keratectasia ex Ulcere. Magnified 25 × 1.

The thinned and bulging cicatrix, *N*, is distinguished from the surrounding normal cornea by its denser structure. The epithelium, *e*, over it is thickened, while Bowman's membrane, *b*, is wanting in this spot. On the other hand, Descemet's membrane, *d*, along with its epithelium is everywhere present—a proof that the ulcer has not perforated.

a hernia of this membrane (keratocele) is produced, which may cicatrize in this ectatic form. In this case this hernia persists as a perfectly transparent vesicle which projects above the surface of the cornea, and which is surrounded by an opaque cicatricial ring.

Ectasis of the cornea may also take place because of a *softening* that results from inflammation. Under this head belong the *keratectasia e panno*, which develops when a thick pannus penetrates pretty deeply into the cornea proper, also the keratectasia following parenchymatous keratitis. In these cases the cornea protrudes as a whole and uniformly, while in keratectasia ex ulcere it is generally only a local protrusion that is present.

A consequence common to all inflammatory ectasiæ of the cornea is that the protruding portions have very little power of regaining their transparency. Accordingly, the prognosis for vision in pannus, keratitis parenchymatosa, etc., must be regarded as essentially more unfavorable as soon as protrusion of the cornea shows itself. And in considering the prognosis

we must take into account, besides the opacity of the bulging cornea, its alteration in curvature and the resulting alteration of refraction of the eye. Sometimes keratectasia is followed by increase of tension.

Treatment is powerless against a fully developed keratectasia; it is attended with success only when there is a question of combating a protrusion that is in the process of development. The means suited for this latter purpose are repeated punctures of the cornea with the subsequent application of a pressure bandage, and iridectomy. Very small ectasiæ, as, for example, small keratoceles, we may perforate with a cautery point and then induce the formation of a flat cicatrix by the long-continued application of a pressure bandage.

Keratectasia corresponds to applanatio corneæ, staphyloma to phthisis corneæ. In the two former cases the cornea itself is protruded or flattened in consequence of inflammation. In staphyloma and in phthisis, on the other hand, the cornea is either partially or wholly destroyed, and it is the prolapsed iris that forms the ectatic or flattened cicatrix.

Marginal ectasia with obliquity of the cornea or with a general protrusion of it may occur as a result of catarrhal ulcers (page 258) or senile marginal atrophy (page 310).

3. *Keratoconus*

272. Symptoms and Course.—In keratoconus, the central part of the cornea very gradually and without inflammatory symptoms begins to bulge forward in the form of a cone. At first the cornea is perfectly transparent, and its peripheral portions keep their normal curvature. Accordingly, the bulging forward of the center of the cornea, as long as it has not advanced too far, is recognized only by the diminution in size which the corneal reflex presents in the central part of the cornea. In the subsequent course of the disease the bulging of the center constantly increases, and the peripheral parts of the cornea are also involved in the conical projection, so that we can perceive the conical shape of the cornea at a glance, especially when we look at the eye from the side (Fig. 126). Finally, the apex of the cone becomes opaque and its surface uneven.

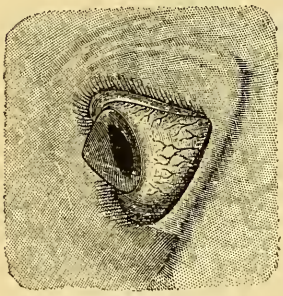

Fig. 126.—Keratoconus. (After Elschnig.)

To the patient the disease makes itself apparent only by the disturbance of vision. The eye becomes apparently myopic, so that all objects have to be brought up very close. Nevertheless, perfectly sharp vision is not attainable by means of spherical concave glasses, because the bulging of the cornea is not spherical, but conical (hyperbolic). By the development of the opacity at the apex of the cone the visual power is, of course, still further reduced.

Keratoconus is a rare disease, which, as a rule, affects both eyes. It begins for the most part between the twelfth and the twentieth year, devel-

ops very gradually in the course of years to the height above described, and ultimately, sooner or later, comes to a standstill. A subsidence of the ectasis is not observed; but ulceration or rupture of the cornea is not observed either. Nor does increase in tension, which so frequently develops in inflammatory ectasiæ of the cornea, occur.

The *cause* of the protrusion lies in a progressive thinning of the central portion of the cornea, which consequently gives way before the intra-ocular pressure [i.e., the tension is relatively too high (see page 85)—D.]. How this thinning is produced is unknown.

In keratoconus the ectasia of the cornea is associated with a corresponding thinning of the latter within the area of the conical protrusion. This *thinning of the cornea* within the area of the conical protrusion can be demonstrated by the ease with which the apex of the cone can be dimpled by means of a sound. Anatomical examinations also have demonstrated that the cornea may be reduced in its center to a third of its normal thickness (Wagner, Hulke).

Owing to the considerable degree of stretching of the cornea, lacerations are produced in Descemet's membrane. The *opacities* which are found at the apex of the keratoconus are partly the result of the lacerations, partly the result of mechanical injuries to which the superficial layers of the cornea are exposed at the point of greatest prominence. In particularly severe cases, ulceration at the apex of the cone may be produced in this way.

Besides opacities which are lasting and which constantly increase, there are sometimes discovered opacities that are present for a time only. These form very minute parallel and generally vertical gray lines at the apex of the cone, and probably represent wrinkles on the posterior surface of the cornea.

A keratoconus of slight degree may be *readily overlooked*, since the cornea is perfectly transparent. The diagnosis, however, can be made even in the earlier stages by examining the corneal reflex, particularly if we use Placido's keratoscope (§ 788) for the purpose. [See also Fig. 399.] The latter instrument at the same time shows most clearly that the apex of the cone is not ordinarily situated in the center of the cornea, but somewhere below it. Again, if we illuminate the pupil with the ophthalmoscope, we see in the red field of the former an annular shadow which is particularly dark at one spot; this spot shifts with the movements of the mirror.

In high degrees of keratoconus, in which the apex is already opaque, the distinction from a keratectasia following a central ulcer of the cornea is often very difficult. We must then take into consideration the condition of the other eye. In keratoconus we almost always find the second eye diseased as well, although not ordinarily to the same extent; while a central keratectasia could only by a rare accident be present at the same time in both eyes.

Keratoconus affects the female more frequently than the male sex. In some cases the statement has been made to me by female patients that in the course of repeated pregnancies the trouble has made particularly rapid progress each time that pregnancy has occurred.

When we see a keratoconus at the outset we can never foretell to what *extent* it will develop, since in some instances it remains stationary after having attained only a moderate pitch, at other times it reaches the very highest point, and moreover ordinarily acts in a different sort of way in the two eyes of the same patient.

273. *Treatment* can point to but slight results in this disease. If we are dealing with cases of moderate intensity, and particularly if after being

under observation for a long time they prove to be stationary, it is best to content ourselves with an optical correction of the refractive error. Usually a satisfactory improvement in the sight can be secured by concave spherical glasses, either alone or combined with cylinders. [In selected cases marked improvement of sight is secured by the use of disks with stenopæic holes or slits, the size and shape of these being determined by careful experiment (Mackay, Hensen, Snellen, Fox).—D.] When the protrusion has become so great that serviceable vision cannot be obtained even with glasses, operation is indicated (see § 850).

Attempts to arrest keratoconus by non-operative means have consisted in the long-continued instillation of a miotic, to diminish the pressure in the anterior chamber, or in the employment of compression. Neither procedure has any success to point to.

4. *Keratoglobus*

274. In keratoglobus the cornea as a whole is larger than normal. Keratoglobus is but one of the symptoms of the general enlargement of the eyeball that constitutes buphthalmus, and for this reason reference must be made to the latter disease (§ 447) for its description.

[Though keratoglobus is thus regarded as one of the forms of glaucoma, cases have been described in which no evidences of glaucoma were present (Stähli).—D.]

In keratoglobus we sometimes see in the otherwise transparent cornea faint, gray, sharply defined opacities of striate or band-like form. These are the result of isolated lacerations or detachments of Descemet's membrane, produced, as in the case of a keratoconus, by the excessive stretching of the cornea.

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275. Tumors of the Cornea.—Tumors that develop primarily in the cornea are among the greatest of rarities. Isolated instances of primary papilloma, fibroma, myxoma, sarcoma, and epithelioma of the cornea have been published. The carcinomata and sarcomata which not infrequently are observed upon the cornea do not originate there, but in the adjacent conjunctiva, and in fact generally in the limbus. These tumors have been already considered under diseases of the conjunctiva; so also has been the dermoid, a congenital form of tumor, situated partly upon the cornea, partly in the conjunctiva.

CHAPTER III

DISEASES OF THE SCLERA

Anatomy

276. The sclera,[1] together with the cornea, forms the fibrous envelope of the eye, the shape of which is nearly that of a sphere having a constriction corresponding to the basis of the cornea. The mean diameter of this sphere (length of the axis of the eye) amounts to 24 mm. The sclera is thickest in the posterior segment of the eyeball, where it has a thickness of about 1 mm. It gradually diminishes in thickness anteriorly, becoming, however, somewhat thicker in the most anterior segment, because here the tendons of the recti muscles become fused with and reinforce it.

The sclera consists of fine fibrillæ of connective tissue, which are united into bundles. These run, generally speaking, in two directions—from before backward (meridional fibers), and in a direction concentric with the margin of the cornea (circular or equatorial fibers). Between the bundles are found a few flat cells. The tissue of the sclera and of the cornea are very much alike, and, moreover, at the corneal margin pass into each other without any sharp line of demarcation. They are chiefly distinguished by the arrangement of the bundles of fibers, which is much more regular in the cornea than in the sclera.

The sclera also contains branched pigment cells, which for the most part are met with only in its deep layers and also along the vessels and nerves that traverse it. In the living eye we often perceive the pigment in the spots where the anterior ciliary veins emerge from the sclera, these spots appearing as small brown dots upon the white membrane. Sometimes we find in the human eye larger, slate-colored or light violet spots upon the sclera due to an abnormal pigmentation. Such pigmentation as this is the rule in many animals. If the sclera is thin, the pigment of the subjacent chorioid is seen as a dark substance shining through it. In this case, which occurs especially in children, the white of the eye has a bluish tint, like thin white porcelain. In some families great thinning and consequent bluish coloration of the sclera occurs as a congenital anomaly. It usually is associated with abnormal fragility of the bones.

The sclera is traversed by vessels and nerves which penetrate into the interior of the eye, but has itself very few vessels. On the other hand, there are numerous vessels contained in the so-called episcleral tissue, that loose connective tissue which envelops the sclera and in the anterior segment of the eye attaches the conjunctiva to it. In the posterior segment of the eye

[1] From σκληρός, hard.

the optic nerve passes through the sclera, which here apparently has an aperture for the passage of the nerve (foramen scleræ). In reality, however, the inner layers of the sclera are continued as the lamina cribrosa through the foramen scleræ (Fig. 24; for more precise particulars, see § 540).

I. Inflammation of the Sclera

277. Inflammation of the sclera (scleritis), which belongs among the rarer affections of the eye, attacks the anterior segment of the sclera, lying between the equator of the eyeball and the margin of the cornea. It sometimes affects only the superficial layers of the sclera, sometimes the deep layers also. In the first case the disease runs its course without entailing any injury upon the eye; but in the second case it is dangerous to the sight, inasmuch as the inflammation spreads from the sclera to the other membranes of the eye. It is hence of practical importance to distinguish between a superficial and a deep form of the disease (episcleritis and scleritis of authors).

(*a*) *Superficial Form of Scleritis* (*Episcleritis*)

278. This form makes its appearance as a focal inflammation, a circumscribed inflammatory nodule forming in the sclera. At the affected spot the sclera, owing to the deposition of exudate, bulges out in the form of a boss, so that a prominence which is sometimes flat, sometimes more acute, and which may reach or surpass the size of a lentil, is found here (Fig. 127). This is traversed by vessels which, because deeply situated (episcleral), are violet in color; and it is immovably attached to the sclera, while the conjunctiva, though, to be sure, injected, can be moved about freely. The nodule feels hard, and is sometimes very sensitive to the touch. Except at the site of the nodule the eye may be perfectly free from injection. The subjective disturbances vary greatly; frequently the disease is associated with but slight discomfort for the patient, while in other cases very violent pain is present, which for a long time deprives the patient of sleep.

In the subsequent *course* of the disease disintegration and ulceration of the nodules never occur; on the contrary, they always disappear by resorption. After the inflammation has remained at its acme for some weeks, the nodule gradually flattens, becomes paler, and at length disappears completely, after lasting altogether from four to eight weeks. Sometimes it leaves no trace behind it; more frequently, however, at the spot where it was situated a slate-colored patch is left, and in the same place the sclera appears somewhat depressed and the conjunctiva is closely adherent to it (cicatrix in the sclera). In other respects the eye suffers no after-injury from the inflammation.

Scleritis is peculiarly prone to recur. The disease may, to be sure, stop with one or two attacks, or years may intervene between the attacks; but in other cases, scarcely has the first nodule disappeared—nay, even the first

one may not have disappeared—when a second one appears upon another portion of the sclera. Sometimes the disease does not cease until nodules have developed, one after another, in the entire circumcorneal space, and at length a zone of gray discoloration is visible entirely surrounding the cornea. By this time the disease has exhausted itself, since a new nodule does not generally develop in the spot where another was situated before. But, before it has gone as far as this, several years may have elapsed, during which the patient with but brief interruptions is annoyed by attacks of inflammation. Moreover, the disease very frequently attacks both eyes. The *prognosis* of the superficial form of scleritis is hence unfavorable in respect to the duration of the disease; while with regard to the final outcome it must be stated as favorable, because the usefulness of the eye for vision suffers no impairment, even if the process lasts a long time.

Superficial scleritis occurs, as a rule, only in adults, and especially in elderly people. In some cases it appears to be connected with rheumatic or gouty affections; in others its origin is obscure. *Treatment* avails but little against it. We are able to ameliorate the symptoms and accelerate somewhat the subsidence of the nodules, without having it in our power to prevent the recurrences. Sodium salicylate or aspirin and sometimes also colchicine are given internally; furthermore, diaphoretic methods of treatment, derivative remedies in the shape of mildly purgative mineral waters, iodide of potassium, etc., may be recommended. As regards local remedies, we may try to produce more rapid subsidence of the nodule by massaging it. Fat, either without addition or under the form of the yellow-precipitate ointment, is introduced into the conjunctival sac, and the nodule, which can be felt through the lid, is then rubbed and squeezed through the lid by means of the fingers. If the disease is associated with violent pain, we may employ hot, moist compresses, dionin, atropine, and local blood-letting (six to ten leeches upon the temple) in addition to the massage: the latter procedure, indeed, in these cases frequently cannot be performed because the nodule is too painful. In this event the application of the constant current to the nodule by means of a small electrode (Reuss) has been recommended. [Orthoform ointment (10 per cent) or acoine oil (1 per cent) may also be tried.—D.]

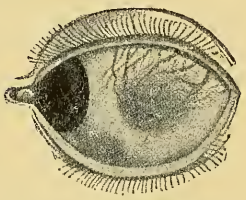


Fig. 127.—Scleritis.

The recent focus is on the outer side of the sclera; from the upper and outer side of the eye dilated conjunctival vessels run down to it. To the outer side and below, and somewhat closer to the cornea, lies a grayish spot at the point where half a year before a scleritic eminence had been situated.

Scleritis in the well-marked cases is a very characteristic and easily recognizable disease. In light and abortive cases the diagnosis is sometimes difficult. For instance, a scleritic nodule which is small and situated particularly close to the margin of the cornea, might be taken for an efflorescence of conjunctivitis eczematosa. The following characters may be regarded as distinctive: The scleritic nodule never actually lies

in the limbus, and besides is not situated in the conjunctiva, but beneath it, so that the latter can be moved about over it. Lastly, the subsequent course will soon clear up the diagnosis, as the eczematous nodule is converted by superficial disintegration into a conjunctival ulcer—a thing which never occurs with a scleritic nodule.

Under the name of *episcleritis periodica fugax* (subconjunctivitis of von Graefe) is denoted an inflammation of the vascular episcleral tissue, distinguished by its transient character and by its tendency to recur. The recurrences often take place with a considerable regularity at intervals of some weeks or months, and may keep on being repeated for years. The separate attacks affect sometimes one, sometimes both eyes. The eye attacked shows marked redness and œdematous swelling of the episcleral tissue and of the over-lying conjunctiva. Often the inflammation is partial in the sense that it is confined to one portion of the anterior segment of the eyeball, or begins in one quadrant of it and travels from this to another. In severe cases contraction of the pupil and spasm of the ciliary muscle (temporary myopia) are present. The pain is sometimes slight, sometimes pretty violent. Ordinarily the inflammation runs its course and the eye becomes normal again in a few days. The disease is, therefore, free from danger, but owing to its frequent recurrences is very troublesome. On the whole, it is a rare affection, and commonly attacks persons in middle life, sometimes without known cause, sometimes as the result of a rheumatic or gouty tendency. Quinine, sodium salicylate, and treatment directed against a uratic diathesis prove advantageous; but many cases defy all treatment.

(*b*) *Deep Form of Scleritis*

279. In this form, too, a swelling of the sclera exists which may make its appearance under the form of separate prominences, but which more frequently is not so sharply circumscribed. In the latter case the sclera shows an extensive bluish-red injection, sometimes covering the whole circumcorneal region, and a more uniform swelling not composed of isolated prominences. Later the sclera in this situation often takes on a peculiar pale-violet color and a transparent look, that make it resemble fine porcelain. But the deep is distinguished from the superficial form most of all by the course of the inflammation and by its being communicated to other parts of the eye.

The inflammation in the sclera in this case as in the superficial form leads not to disintegration of the inflammatory products, but to their disappearance by resorption with the formation of a residual dark-colored cicatrix. In the meantime, however, the sclera has been undergoing considerable attenuation at the site of the cicatrix, so that it is no longer able to offer resistance to the intra-ocular pressure, even though this does not exceed its normal amount. Hence *ectasis* of the diseased spot occurs. This makes its appearance under two forms—either as an expansion of the whole surface, or as a gibbous protrusion of the sclera. In the former case the entire circumcorneal zone of the sclera, which has been colored gray by the scleritis, becomes constantly more and more dilated. Consequently, the cornea, together with the adjacent portions of the sclera, is projected forward, so that the eyeball is elongated in a sagittal direction and becomes pear-shaped. In the second case, on the contrary, a circumscribed protrusion of the

thinned-out spots, raising them above the level of the healthy sclera, is produced, so that there are seen rising about the cornea a number of humps, which, because of their thin walls, show the dark pigment shining through. As these belong to the region of the ciliary body, they are called ciliary staphylomata (see page 359).

Complications affecting other portions of the eye are likewise a characteristic feature of the deep form of scleritis. They affect both cornea and uvea. In the cornea, infiltrates, deeply situated, develop, which do not break down into pus but become absorbed again, leaving a permanent opacity (sclerosing keratitis, see page 300). In the iris we find the signs of iritis, mainly under the guise of posterior synechiæ and even of occlusio pupillæ, but never of hypopyon. In the chorioid, the inflammation affects chiefly its most anterior portion, and causes injury to vision chiefly through accompanying opacities in the vitreous. So in this form of scleritis almost all parts of the eye suffer; and for this reason it is much more dangerous than the superficial form.

The superficial and the deep variety of scleritis are not by any means sharply distinguished from each other, but show many transition forms. We cannot in fact, see in the living eye how far the inflammation penetrates into the sclera. When we make the assumption that in the deep form the inflammation does extend more deeply, we have no direct proof of it; we can only infer this indirectly from the subsequent thinning of the sclera and from the spread of the inflammation to the subjacent uvea. In fact, many authors assume that the real starting point of the inflammation in this form lies in the uvea, and they call it, therefore, sclero-chorioiditis or uveo-scleritis. [Cf. remarks on Uveitis Anterior, page 293.]

In parenchymatous keratitis we sometimes find a coincident, slight but widely diffused, circumcorneal scleritis. Isolated nodules are sometimes also observed in the sclera as a result of syphilis, but a typical recurrent scleritis does not ordinarily arise from this cause.

In many cases of diffuse deep scleritis there develop in the inflamed zone hard whitish nodules of the size of a pin's head, which lie beneath the conjunctiva, and all are pretty much the same distance from the margin of the cornea. They might be taken for tuberculous nodules, which, however, they are not. In fact, they disappear again without undergoing disintegration. [Real tuberculous nodules do occur. See page 338.]

Scleritis Posterior.—In exceptional cases it happens that the inflammation is situated not in the anterior segment, but further back, either in the equatorial region, or actually in the posterior segment of the eyeball. Such a *scleritis posterior* is hard to diagnosticate, because the inflammatory swelling of the sclera, being in a place which it is impossible to get at, can neither be seen nor felt. The symptoms are pain and disturbance of vision. The ophthalmoscope shows the cause of the latter to be a gray cloudiness and swelling or even a detachment of the retina over the scleritic focus. In severe cases there are even some exophthalmus and limitation of the mobility of the eyeball. As the inflammation in the sclera abates the changes in the retina and the associated disturbance of sight likewise disappear.

280. Morbid Anatomy.—Anatomical examinations up to the present time have been confined almost exclusively to cases of deep scleritis, since it is only in these cases—and but occasionally then—that enucleation of the eye is performed and thus material for the examination obtained. In such cases the sclera is permeated in its middle and

deep layers with uninuclear leucocytes (Fig. 128). In particularly severe cases in which clinically the whole tissue appears to contain a gelatinous infiltration, the sclera at the diseased spot is transformed throughout its entire thickness into a tissue which contains an excessive abundance of cells and in which necrosis has set in here and there. Sometimes even whole lamellæ of the sclera have become necrotic en masse and form a

FIG. 128.—DEEP SCLERITIS. SECTION THROUGH THE REGION OF THE CILIARY BODY, TAKEN FROM THE EYEBALL OF A TWENTY-FOUR-YEAR-OLD GIRL. Magnified 24 × 1.

The conjunctiva, *B*, which in the enucleation had been cut off close to the limbus, is pretty strongly infiltrated. In the subjacent sclera, *S*, are numerous narrow files of round cells, which show a special disposition to accompany the blood-vessels. The scleritic nodule, that was visible on clinical examination, corresponds to a large, irregularly shaped mass of infiltration, occupying almost the entire thickness of the sclera. The posterior section of this mass is the part that presents the most marked accumulation of cells; in the anterior section the cells are most densely placed near the margin that is directed toward the cornea—looking as if the infiltrate were making an attempt to push its way into the latter. The anterior layers of the cornea, *H*, are occupied by quite a large infiltrate, *i* (sclerosing keratitis). In its posterior layers can be made out files of cells, which accompany the deep-seated, new-formed vessels, and which show a tendency to pass up from the deep to the middle layers of the cornea. The ciliary body, *C*, and the iris, *I*, are atrophic. In the former the ciliary processes in particular are greatly diminished in size. At the anterior border of the ciliary muscle can be seen the cross section, *c*, of the circulus arteriosus iridis major. The iris also is thinned, and chiefly at its peripheral portion, which over the space, *a b*, is closely applied to the posterior surface of the cornea, and has become adherent to it. Descemet's membrane, *d*, and the lumen of Schlemm's canal are consequently no longer to be made out in this situation. This peripheral adhesion of the iris is in consonance with the increase of tension that existed in the living eye.

sequestrum. Sometimes in the cellular tissue there are scattered nodules containing giant cells—this without there being any tuberculosis present. In exceptional cases, however, there are real tuberculous nodules.

The more intensely the sclera is diseased the surer we are to find inflammatory changes not only in the adjacent cornea, but also in the uvea and especially in the chorioid.

281. Deep scleritis almost always affects both eyes, and, as treatment is unable to arrest it, is prolonged over a course of years. It leads to the formation of dense corneal opacities, to seclusio pupillæ with its baneful consequences, to opacities of the lens and vitreous, to myopia of high degree due to the elongation of the axis of the eye, and finally to elevation of tension due to the ectasiæ in the sclera. Hence the disease always ends by producing great impairment or even complete loss of sight.

The deep form, in opposition to the superficial variety, affects mainly young people (but not children). It is often found in conjunction with the signs of scrofula, tuberculosis, or hereditary syphilis. In the female sex, which is more frequently attacked than the male by this disease, disturbances of menstruation appear to furnish the exciting cause.

Treatment has very little power over deep scleritis. It has first of all to combat by dietetic and medicinal remedies any constitutional affection that may lie at the root of the disease, and for this purpose the preparations of iodine (iodide of potassium, iodide of iron, iodureted mineral waters), or, in case of the disturbances of menstruation, the preparations of iron are employed. As concerns the eye itself, the inflammation of the cornea and iris must be treated in the appropriate way. In the subsequent course of the disease iridectomy is frequently required, either for optical reasons, to place the pupil behind that part of the cornea that is still transparent, or to prevent the elevation of tension which may be excited by the seclusio pupillæ or by the ectasiæ of the sclera. Iridectomy, however, should, if possible, not be performed until after the subsidence of all inflammatory symptoms.

II. Injuries of the Sclera

282. Perforating Wounds of the Eyeball.—The same varieties of traumatism that we have discovered as occurring in the cornea and conjunctiva are also met with in the sclera. The first question that we have to ask in considering any such injury is, whether a perforation of the tunics of the eyeball has or has not been produced by it, and the second question is whether in case a perforation is present, there is a foreign body in the eye.

Every *perforating injury* is to be regarded as intrinsically serious, because, given a perforation, there is a possibility of a coincident infection of the interior of the eye; but this almost always leads to a severe form of inflammation that is very destructive to the eye. This is true for perforating wounds of the cornea as well as of the sclera, for which reason the statements made in the following lines may be applied to both.

Perforating injuries of the eye are very frequent in the working class; and furnish a large contingent of the blind. This is particularly the case in regions where many industries are carried on. The following report of Cohn gives a good idea of the frequency of the injuries to which the eyes of many workmen are exposed: Among twelve hundred and eighty-three workers in metals employed in six factories, each man received

on an average from two to three injuries in a year. Of course, the great majority of these injuries were of a slight character; most, indeed, consisted merely in the penetration of small metallic particles into the surface of the cornea, which were, for the most part, removed at the factory itself. About half of the workmen were compelled to seek medical aid; and out of every thousand 28 suffered a partial impairment of sight, and 16 had lost one eye altogether. [Every year 50 per cent of the stone workers in Aberdeen have to resort to treatment for eye injuries. From extended statistics it appears that in England and America eye injuries constitute something over 5 per cent of all injuries occurring in mines, quarries, factories, and workshops (Collis). The majority of these injuries are slight; yet even of these slight injuries many, especially in stone-masons and coal miners, give rise to dangerous infection and particularly to ulcus serpens (Shufflebotham, Collis). From 2.5 to 3.0 per cent of these industrial eye injuries result in blindness of one or both eyes (Collis).—D.]

283. The most important *symptoms of the presence of a perforation* are:

(*a*) Reduction of the intra-ocular tension. This symptom is particularly valuable in the case of small wounds in the sclera, which are concealed by the ecchymosed conjunctiva, and are hence not directly accessible to inspection. The diminution of tension, of course, lasts only as long as the wound is open. [It must be borne in mind that a reduction of tension may also occur in injuries of the eye without perforation (see § 463).—D.]

(*b*) If the perforation has occurred in the region of the anterior chamber, the latter is shallower or altogether obliterated, as long as the wound remains open.

(*c*) In the case of somewhat larger wounds the prolapse of the subjacent structures gives evidence of the presence of a perforation. Most frequently it is the uvea which protrudes from the wound under the form of a darkly pigmented mass. According to the situation of the wound, the prolapsed portion belongs to the iris, to the ciliary body, or to the chorioid. If the uvea is ruptured, some vitreous is often found hanging out of the wound. Very frequently, too, there are extravasations of the blood in the interior of the eye, which, to be sure, often likewise occur in the case of non-perforating contusions of the eye. Blood extravasated into the anterior chamber generally sinks to the bottom of it soon after the occurrence of the injury, so that, like a hypopyon, it fills up the lowermost part of the chamber and is bounded above by a horizontal line (*hyphæma*[2]). Blood in the vitreous often makes itself evident by a reddish reflex from the pupil (*hæmophthalmus*[3]).

Hæmorrhage into the anterior chamber or the vitreous occurs not only from injury, but also as the result of inflammation or even without any known cause. In the *anterior chamber*, blood sinks to the bottom and is reabsorbed. In otherwise healthy eyes small quantities of blood may often disappear completely within twenty-four hours. The process of resorption lasts longer when there is much blood in the anterior chamber, and especially when the eye is diseased in other ways as well and has not normal metabolism. The longer the blood remains in the anterior chamber the darker does its color become. So, in cases in which a hæmorrhage into the anterior chamber has been repeated

[2] From ὑπό, beneath, and αἷμα, blood. [3] From αἷμα, blood, and ὀφθαλμός, eye.

after the lapse of some time, we see a hyphæma which is composed of two strata of different color; the lower dark stratum represents the first hæmorrhage, the upper bright one belongs to the recently extravasated blood. Very old extravasations of blood sometimes acquire a brown or dirty-green color, and the cornea, too, may take on a similar coloration (see page 316). In the iris likewise a greenish or brownish discoloration has been observed as a result of hæmorrhage into the aqueous or vitreous chamber.

If the blood remains a long time in the anterior chamber it may—especially if there is a coexistent inflammation—serve as the substratum for the formation of new tissue. In this way the good result of operations, such as iridectomy and iridotomy, designed for the restoration of a clear pupil, is often rendered of no effect, as the blood extravasated during the operation covers up the opening that has been made and subsequently causes its reocclusion by means of a membrane.

[Blood in the anterior chamber lying in the pupil often remains long after blood on the iris has been absorbed (see page 16).—D.]

Blood extravasated into the *vitreous* is found there under the form of flocculi or larger masses. When observed with the ophthalmoscope these either look simply black or show a faint reddish gleam. If they occupy the anterior section of the vitreous, they may even be recognized with lateral illumination (provided the pupil is widely enough dilated) through the dark-red reflex emitted from the depths of the eye. Blood present in the vitreous always requires a long time for its complete resorption, and if much blood has been extravasated opacities of the vitreous of considerable size always remain and cause great impairment of vision. [In rare cases almost complete restoration of sight occurs even when the whole vitreous has been filled with blood.—D.] In some cases of traumatic hæmorrhages into the vitreous I have observed that, some time after the injury, the coloring matter of the blood all at once became dissolved in the ocular fluids and then was immediately diffused all through the eye. The aqueous humor, too, in such cases was colored red, so that the iris looked as though seen through ruby glass. [Unabsorbed hæmorrhage may also cause siderosis bulbi (see page 350).—D.]

[To absorb vitreous hæmorrhages we use potassium iodide, subconjunctival injections of salt (see page 65), and dionin either by instillation or subconjunctival injection.—D.]

If vitreous has been prolapsed through a wound it gradually becomes clouded wherever it lies outside of the eye, so that it gets to look like a shred of mucus adhering to the region of the wound. It takes often several weeks before such a shred is finally cast off. [Such a shred affords a ready pathway for infection to the interior of the eye.

Other consequences of eye injuries, especially blows on the eye, are dislocation of the lens, rupture of the chorioid, and concussion and detachment of the retina.—D.]

284. Perforating wounds of the sclera are distinguished into incised, punctured, and lacerated wounds. To the two former belong, besides wounds produced by operation, those caused by the penetration of sharp-pointed or sharp-edged foreign bodies. Lacerated wounds are most frequently the consequence of a *rupture* of the sclera. This is produced by the action of a blunt instrument upon the eye (contusion)—e. g., by a blow with the fist or with a cane, by a stone, by the impact of large flying fragments of wood or metal, by a thrust from a cow's horn (not infrequent among farmers), by striking the eye against a projecting corner, etc. The rupture of the sclera is generally pretty long, is curved, and lies near the corneal margin and concentric with it. Most ruptures are observed along the upper and

inner margin of the cornea (Fig. 129). As a rule, the process does not stop at laceration of the sclera, but a part of the contents of the eye, most generally the lens, is expelled, and the vitreous is sometimes seen hanging out of the wound. The iris, at a point corresponding to the site of the rupture, appears to be wanting, and the eye often looks as if the portion of the iris in question had been removed by iridectomy (see § 392). Frequently the conjunctiva over the lacerated sclera, thanks to its great extensibility, remains uninjured. In this case the extruded lens is sometimes found lying beneath the conjunctiva (Fig. 134).

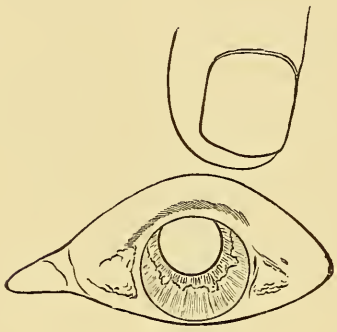


Fig. 129.

Fig. 130.

Fig. 129.—Rupture of the Sclera. Injury produced by the eye being hit with a stone thirty years before. Rupture healed with retention of the visual power. The site of the rupture can be made out along the convex gray line (scar) which runs concentric with the upper margin of the cornea. The cornea is clear, the anterior chamber rather deeper than normal. The pupil is displaced bodily upward (the lower margin of the pupil lies approximately behind the center of the cornea) and extends to the upper margin of the cornea. The lesser circle of the iris can be followed on both sides of the coloboma, to a point behind the limbus. On both sides of the cornea is a pinguecula.

Fig. 130.—Same Case in Section. Magnified 3 × 1. The rupture runs from Schlemm's canal, *s*, obliquely upward so that that portion of the scar which is visible externally lies in the sclera 2 mm. back of the limbus. The lips of the wound are separated; between them lies scar tissue, and imbedded in this is the iris. The latter is torn away from its insertion in the ciliary body and is rolled up; the section has happened to strike it in such a way that the iris here looks like a closed ring on the inner circumference of which the retinal pigment layer is visible. The ciliary body is somewhat lacerated at its insertion and the scar tissue extends into the chink that is thus produced.

In rupture of the sclera the blow may act directly upon the eye itself or a blunt body—e. g., the tip of a cow's horn—may enter between the eyeball and one wall of the orbit and squeeze the eye against the opposite wall.

We distinguish between direct and indirect rupture. The former is the kind that originates at the point of impact of the foreign body by which the blow is inflicted; the latter, that which occurs at a spot remote from the point of impact. In *indirect* ruptures the only efficient factor is the compression of the eyeball, so that these ruptures almost always have the same typical situation. In *direct* ruptures, there is added to this compression a local indentation of the eyeball, by which the situation of the rupture is determined. This situation, therefore, varies greatly and often is not in the sclera but in the cornea. When compression is the determining factor the direct result of it is that the contents of the compressed eye are put suddenly in a condition of increased tension and thus cause rupture of the capsule of the eyeball. The rupture, therefore, takes place, like an explosion, from within outward, and begins in the region of Schlemm's canal, because here the tough inner layers of the sclera pass over into the

delicate lamellæ of the ligamentum pectinatum, and thus the resistance of the sclera is diminished at this point (Fig. 131). The fact that most scleral ruptures start from a point situated upward and inward is due to the trochlea, which forms a bony prominence at the upper and inner angle of the orbit (Figs. 301 and 302, *T*). When the eyeball is forced against either the inner or the upper wall of the orbit by a blow coming from below or from without, the trochlea presses into the sclera and thus causes the rupture to begin in this meridian (Müller).

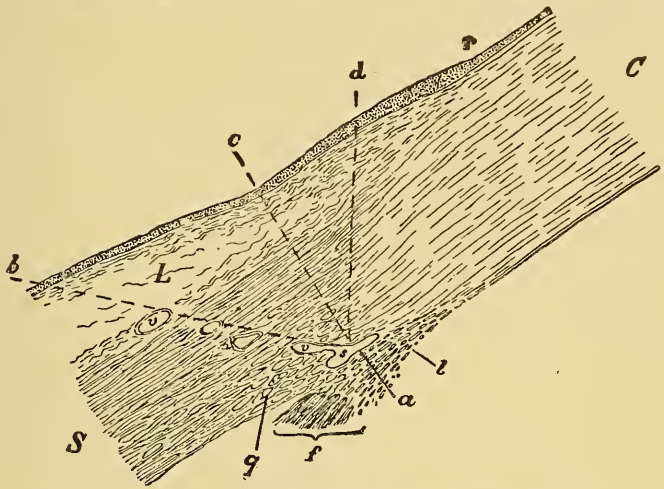


Fig. 131.—Course of a Scleral Rupture. Magnified 22 × 1.

C, cornea; *S*, sclera; *r*, end of Bowman's membrane, and beginning of the limbus *L*. *f*, anterior end of the longitudinal fibers of the ciliary muscle, which pass over into the lamellæ of the ligamentum pectinatum, *l*. The capsule of the eyeball is less firm at the corneo-scleral margin, first, because here a larger number of the fibers of the sclera (those namely which in the figure are seen in cross section at *q*) take a circular course; second, because in front of these fibers Schlemm's canal, *s*, is imbedded in the scleral tissue, which consequently has its inner layers interrupted at this spot. From Schlemm's canal to a point right beneath the conjunctiva, the anterior ciliary veins, *v*, *v*, run continuously through the sclera. Most scleral ruptures follow these cavities in the direction *ab*, so that the external orifice of the rupture lies some millimetres behind the limbus. More rarely the ruptures of the sclera pass perpendicularly in the direction *ac*, or even run obliquely forward in the direction *ad*. Ruptures of the latter sort have usually but little length so that nothing but the iris prolapses and this only to a slight extent (Fig. 132), and the rupture itself does not become visible until the iris is excised (Fig. 133). These small ruptures almost always afford a good prognosis.

Fig. 132.

Fig. 133.

Fig. 132.—Small Scleral Rupture Situated in the Limbus. Magnified 2 × 1. The iris has prolapsed through the rupture and hence the pupil is displaced in the direction of the latter. The iris at the opposite side is broadened and hence the contraction furrows are separated from each other, as comparison with Fig. 133 shows.

Fig. 133.—The Same Case after Excision of the Iris. Magnified 2 × 1. The short rupture which is situated in the limbus has now become visible. *a* a_1, the angles of the sphincter which are now properly placed.

Sometimes *very small* ruptures occur right in the limbus itself or even in the transparent cornea (Figs. 131, *ac* and *ad*). In these, owing to the close attachment of the conjunctiva, the latter is necessarily ruptured and the iris almost always prolapses.

The prognosis of these small ruptures is good, provided the prolapsing iris is excised. They occur comparatively often in young people (even in children), while the more extensive ruptures occur in the old.

Very rarely we meet with *incomplete* rupture of the sclera. We then find the conjunctiva and sclera ecchymosed and swollen, and a few days after the injury a bluish-black line appears near the limbus and concentric with it. This dark-area may become ectatic later, and increase of tension may set in. There may be displacement of the pupil.

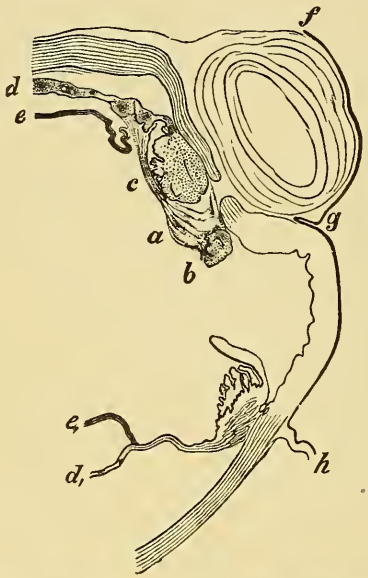


FIG. 134.—RUPTURE OF THE SCLERA AND LUXATION OF THE LENS BENEATH THE CONJUNCTIVA. Magnified 3 × 1.

Vertical section through an eyeball which had been injured by a calf's horn. Since seven weeks later symptoms of sympathetic inflammation of the eye set in, the eye was enucleated. The rupture in the sclera lies close to the upper margin of the cornea, not quite one millimetre behind the corneo-scleral junction, so that the lower lip of the wound contains a narrow rim of sclera. The tear in the sclera gapes to the extent of nearly a millimetre, and is filled with a delicate cicatricial tissue, *a*, which extends back from this point into the interior of the eye, passing between the ciliary body, *c*, and the iris, *b*. The latter at a point corresponding to the scleral rupture is torn off from the ciliary body (iridodialysis) and is rolled into a ball just as in Fig. 130. The ciliary body, *c*, likewise greatly altered, is found behind the upper edge of the rupture. It is continued into the chorioid, *d*, which presents marked inflammatory infiltration about the large blood-vessels (inflammation of the kind that gives rise to sympathetic trouble). The retina, *e*, is detached up to the ora serrata, and at the latter point is torn loose and is much puckered. Below the scleral rupture is seen the cornea in section, compressed vertically and as a result of the compression puckered on its posterior surface. At the lower portion of the eyeball the ciliary body is swollen, and the iris is carried backward so as to be in part applied to the surface of the ciliary body. The detachment of the chorioid, d_1, and of the retina, e_1, seen here in the cut, did not exist during life, but is a result of the way the specimen was made. The scleral rupture is covered in front by a mass which from its concentric striation is recognized to be the lens. The conjunctiva, *f*, overlying the lens, is detached by it from the sclera as far as the limbus, *g*. *h*, remains of the conjunctiva at the lower margin of the cornea.

Ruptures of the sclera are generally such serious injuries, because a force that is strong enough to break an eye open always causes *lesions elsewhere*, in the interior of the eye. The iris (Figs. 130 and 134, *b*) is almost always torn away from its insertion at a point corresponding to the extent of the scleral rupture (iridodialysis), and is either incarcerated in the wound or is extruded through the latter beneath the conjunctiva, to which it becomes adherent. The eye then presents a coloboma over an area corresponding to the rip in the sclera. The portion of the iris remaining in the eye is commonly found to be considerably retracted (Fig. 134, lower part). The anterior chamber is

consequently unusually deep, and particularly so if, in addition, the lens and a part of the vitreous have been expelled. In many cases a piece of the iris, or even the entire iris, is torn altogether out of the eye. In only a very few cases does the lens remain in position and in place. Usually it is either expelled entirely from the eye, or it remains lying beneath the conjunctiva, provided the latter is unruptured (Fig. 134, *f*). The vitreous is often densely permeated with blood; and laceration or hæmorrhagic detachment of the retina or chorioid may be present. Since to the severity of such lesions there is added the danger of subsequent infection of the wound, it can be readily understood that most eyes which have suffered a rupture of the sclera undergo destruction. It is an exception, in fact, for such an injury to recover with the retention of serviceable vision. A farmer once presented himself at my clinic who had been gored first in one eye and then, some years afterward, in the other also, by a cow's horn. In both eyes there was a healed rupture of the sclera on the inner side, with what looked like a well-made coloboma of the iris. Both lenses were absent, but the fundus was healthy and with cataract glasses the sight was very good. This man, therefore, may be said to have had a double extraction performed by the cow, and that, too, with more success than many operators are accustomed to have with their operations.

For rupture of the cornea, see page 308.

285. The course after a perforating injury varies according as a foreign body has remained in the eye or not. Suppose, first, that no foreign body has remained in the eye. Then the only thing to be considered is the healing of the wound itself; and in regard to this the main thing to determine is whether the injury has been followed by *infection* of the wound or not, as upon this depends the question whether healing takes place with or without inflammation.

(*a*) *Healing without inflammation* is to be expected only in the case of clean, non-infected wounds. The most favorable conditions are presented by clean incised or punctured wounds, such as, for instance, are made by operations, where at the same time care is taken that no foreign tissue, like uvea or vitreous, is introduced into the wound. But even if this latter should be the case, healing without inflammation is still possible, although the edges of the wound cannot unite with each other directly, because they are not in contact. The tissue consisting of uvea or vitreous introduced between them is gradually converted into cicatricial tissue, and thus takes part in the definitive closure of the wound. It then, to be sure, always remains attached to the cicatrix, a thing which later on often entails evil consequences. In this way—i. e., by the interposition of a cicatricial tissue taking place between the edges of the wound—even lacerated wounds, such as develop, for example, after rupture of the sclera, may, if the case runs a fortunate course, heal, and heal without inflammation so that the eye remains serviceable for vision.

We often observe that the scleral wounds, which lie near the corneal margin and in which the iris or lens capsule has become lodged, close imperfectly, since while the conjunctiva does unite over the scleral wound, the latter remains open to a certain extent because the interposed tissues pre-

vent its lips from coming into direct contact. Through the gap aqueous humor constantly exudes beneath the conjunctiva, which either takes on a gelatinous character in the region of the cicatrix or is bulged out in the form of a circumscribed vesicle resembling a cyst (Fig. 135, *h*). Following Von Graefe, we give this condition the name of *cystoid cicatrization* (see page 222). It is chiefly observed after operations (cataract extraction and iridectomy).

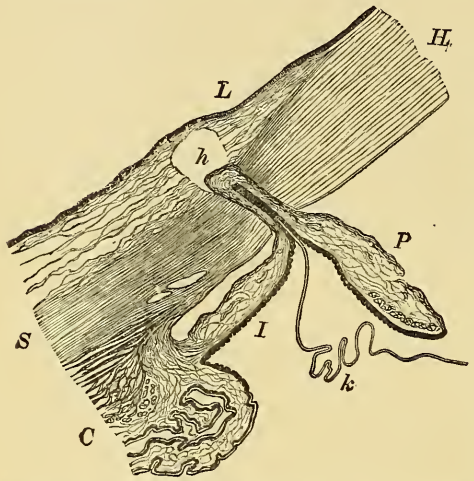


FIG. 135.—INCLUSION OF THE IRIS WITH CYSTOID CICATRIZATION AFTER THE EXTRACTION OF A SENILE CATARACT BY GRAEFE'S PERIPHERAL LINEAR SECTION. Magnified 13 × 1.

The section by which the extraction was made cuts in two the line of junction between the sclera, *S*, and the cornea, *H*, so that by its anterior half it lies in the sclera, by its posterior half in the cornea. On account of the interposition of the iris, *I*, the lips of the wound have not united; in fact, the tract of the wound extends under the form of a cavity, *h*, even into the tissue of the conjunctiva of the limbus, *L*, so that the wound is closed only by a very thin layer of conjunctiva which, on account of the cavity beneath it, looks like a vesicle. The iris by being jammed into the wound is folded upon itself, the point of flexion corresponding to the exterior surface of the sclera. Peripherally from the site of its incarceration the iris being drawn tense runs in a straight line to the ciliary body, *C*; consequently, the anterior chamber is here reduced to a narrow slit, although the sinus of the chamber remains pervious. The pupillary portion of the iris extends centrally from the scar into the anterior chamber, where it lies free. On its anterior surface may be seen the entrance of a crypt, near its posterior surface the cross section of the sphincter pupillæ. Along with the iris, the much-puckered capsule, *k*, of the lens is also, drawn up to the cicatrix, and has become adherent to it.

286. (*b*) Perforating wounds of the sclera are followed by violent *inflammation* whenever infection of the wound or the interior of the eye has occurred. Infection takes place either by means of the very body which causes the injury, it being covered with dirt and thus conveying infectious germs, or secondarily from the fact that the opening in the envelope of the eyeball affords a point of entrance for germs, especially from the conjunctival sac. The interior of the eye is uncommonly susceptible of infection, since it obviously affords a good medium for the cultivation of different kinds of schizomycetes. The inflammatory process mainly affects the uvea. In the acutest cases there is a purulent inflammation of the latter, which leads to the formation of abscess in the vitreous or to an actual suppuration of the whole eye (panophthalmitis). In the less violent cases there is a plastic iridocyclitis—i. e., the inflammation of the iris and ciliary body produces an exudate which subsequently becomes organized into a false membrane. In this case, too, the eye is generally lost, although it is not destroyed in such a violent fashion as in panophthalmitis, but by a process of protracted inflammation. The exudates which undergo organization shrink and thus gradually diminish the size of the eyeball (atrophy of the eyeball). This outcome of an injury is even more dangerous for the patient than panophthalmitis, since in the former

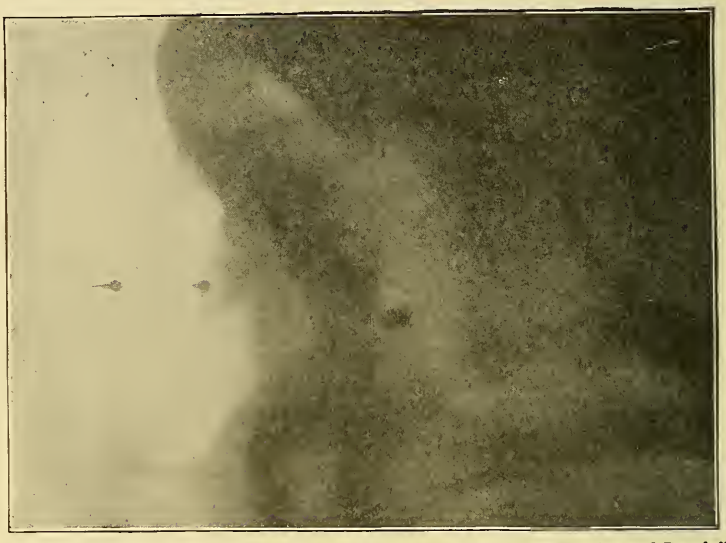
case sympathetic inflammation of the other eye very frequently sets in, which is not the case in panophthalmitis.

287. Foreign Bodies in the Eye.—The presence of a foreign body in the interior of the eye converts every injury, be it ever so insignificant otherwise, into a serious lesion, which, in most cases, entails the destruction of the eye. Hence in every injury attended with perforation we must at once propound the query whether or not there is a foreign body left in the eye. In most cases the history of the case itself supplies points important for the determination of this fact. If, for instance, a person has run a pair of scissors into his eye, we would naturally suppose that there was no foreign body there; conversely, in the case of a man who has had a perforating injury of the eye produced by the explosion of a percussion cap or while he was hammering iron, the presence of a foreign body in the eye is extremely probable. The character of the foreign bodies in question varies exceedingly. Most commonly we have to do with fine splinters, the points and sharp edges of which enable them to penetrate the sclera. In this category belong chiefly splinters of metal, splinters of glass, and fragments of stone—less commonly slivers of wood, etc. The foreign body may be situated in any part of the eye; indeed, if it has sufficient projectile force, it may even, after traversing the entire eyeball, perforate the sclera a second time on the opposite side, and penetrate into the tissue of the orbit. The precise determination of the place in which a foreign body is located within the eye is generally attended with great difficulties. As a rule, it is only during the time immediately succeeding the injury that it is possible to see the foreign body directly, although even then inspection of the interior of the eye is often rendered impossible by the presence of hæmorrhages. Subsequently, the difficulty of this inspection is still further heightened by the cloudiness which soon develops in the media and by the exudates which envelop the foreign body and render it unrecognizable. If we are dealing with metallic fragments of not too small dimensions, we may be able to make them out and localize them by means of the Röntgen rays; and for chips of iron we may also employ a sensitive magnetic needle (sideroscope). If these means are unavailing, we are often driven to conjectures with regard to the location of the foreign body—conjectures based upon the direction pursued by the body in its flight, the situation of the aperture by which it entered, the sensitiveness of certain portions of the eye to touch, the presence of a circumscribed obscuration (scotoma) in the field of vision, etc.

288. [The localization of foreign bodies in the eye by means of the X-rays has reached a high degree of accuracy, thanks to the instruments devised by Sweet, Mackenzie Davidson, Dixon, and others. In these instruments a marker or pair of markers placed close to the eye is used. Skiagrams of the eye are taken from two different positions. The differing relations existing on the two plates between the shadow of the foreign body and of the markers (see Fig. 136) determine the actual position of the

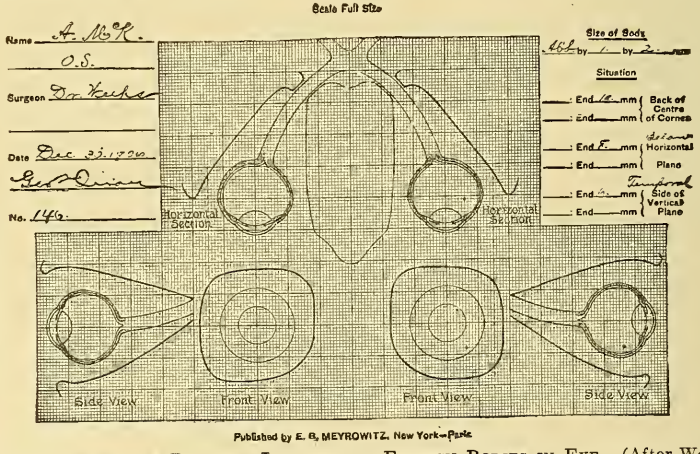
foreign body with relation to the markers and therefore its actual position in the eye. This position can then be plotted on a diagram (Fig. 137).—D.]

In injuries produced by chips of iron the *magnet* is employed. Forms of apparatus have been constructed to determine in doubtful cases whether a piece of iron is present



[Fig. 136.—Skiagram of Foreign Body in Eye. (After De Schweinitz and Randall.)

The picture is taken with the X-ray tube placed in the horizontal plane passing through the two markers. The shadow of the foreign body and of the two markers is seen on the plate. Another plate taken with the tube in a different position would show the shadows in a different position. A comparison of the two plates enables us to determine the position of the foreign body.—D.]



[Fig. 137.—Chart for Plotting Location of Foreign Bodies in Eye. (After Weeks.)

The chart modified from the one devised by Sweet is divided into mm. squares in order to facilitate measurement and plotting. The three views (front, side, and horizontal) enable the location of the foreign body to be indicated precisely.—D.]

in the eye at all. In these the injured eye is brought as close as possible to a very sensitive magnetic needle (astatic needle of Léon Gérard, sideroscope of Asmus) which undergoes deflection if there is a chip of iron present in the eye. By ascertaining at what

point of the surface of the eyeball this deflection is the greatest, we can determine approximately the situation of the iron. [It is a positive proof of the presence of iron in the eye if the application of a magnet, especially the large magnet of Haab, causes bulging of the iris or pain. The absence of this sign is no proof that iron is not present.—D.]

289. It is only in rare instances that a foreign body is tolerated for any length of time in the eye without setting up inflammation, the body itself either remaining free or becoming encapsulated in an organized exudate. But even such eyes as these are by no means secure from a sudden outbreak of inflammation—occurring sometimes years afterward—which causes their destruction. In the great majority of cases the inflammation follows close upon the heels of the injury. Such an inflammation is either a panophthalmitis, an abscess in the vitreous, or a plastic irido-cyclitis, just as in the case of simple perforating injuries.

Leber has determined, by a series of experiments upon animals, the reason why the presence of a foreign body in the eye regularly results in a severe inflammation. This inflammation is either excited by the presence of micro-organisms which make their way into the interior of the eye from the conjunctival sac, along with the foreign body or subsequently, or it is the consequence of a chemical irritation of the tissues produced by those foreign bodies which are not chemically indifferent. For example, purulent inflammation could be produced by bits of copper, and still more by particles of mercury, which were introduced aseptically into the anterior chamber. Hence, such inflammation does not necessarily presuppose the entrance of schizomycetes into the eye. Observations upon man agree in general with the facts obtained by experiment. Whether a body which penetrates into the eye is well borne by it or not depends upon the following circumstances: 1. First of all, upon the fact of its being *aseptic* or not. Small fragments of metal are commonly aseptic, if they fly off when the metal is being hammered or turned, because they are then strongly heated and thus sterilized. Large metal fragments are usually not heated to the point at which they become perfectly sterile, and hence give a bad prognosis even if they are removed very soon from the eye. 2. Upon its *chemical* character. Chemically indifferent bodies (such as, for instance, fragments of glass), if they get into the eye aseptically, are the ones most likely to remain there without producing any further ill effect. The contrary is true of the foreign bodies that are of most frequent occurrence—i.e., chips of metal. These almost always set up a severe inflammation, even when they are sterile, because they gradually dissolve in the tissues of the eye and become oxidized there, so as to act as chemical irritants. This is the case with iron, and still more with copper; metals which, like lead and the noble metals, are innocent in this regard, are of comparatively rare occurrence in the eye. 3. The *tolerance* of the separate tissues of the eye toward foreign bodies varies. The uvea, and especially the iris and ciliary body, exhibit the greatest reaction to injury of any kind. The lens, on the contrary, possibly on account of the sluggishness of its tissue metamorphosis, is the part of the eye in which foreign bodies are relatively the best borne. If, for example, a small chip of iron has become imbedded in the lens the latter, to be sure, becomes clouded, but inflammation ordinarily fails to take place. In such a case sometimes the lens is afterward colored brown by the oxide which is formed. A deep brown coloration is produced, mainly under the guise of rust-colored dots which lie beneath the anterior capsule of the lens and form a crown, corresponding nearly in situation to the margin of the pupil when dilated (Samelsohn). Later the coloration may extend to the iris, which, if previously gray or blue, assumes a rusty

brown hue. This impregnation with iron also occurs in the other tissues of the eye, particularly in the retina, which may in consequence become atrophic, so that blindness may ultimately ensue even when the fragment is tolerated without inflammation (Hippel, Jr.). The impregnation of the tissues with iron is called *siderosis bulbi* (from σίδηρος, iron). [Siderosis is a rare condition. It may also come from unabsorbed hæmorrhage (Kipp, Clegg). The condition may develop as early as two months after the injury (Natanson), but usually not till much later. The staining of the tissues may clear after removal of the foreign body (Clegg).—D.]

Small fragments of iron may in time be entirely dissolved by oxidation.

290. The *prognosis* of perforating wounds of the eyeball is deducible from the exposition given above. In every case it is grave, for even the minutest prick made with a fine needle may induce suppuration of the eyeball, if the needle was contaminated with septic substances. As we do not, for the most part, know whether the body causing the injury was aseptic or not, and as the consequences of an infection of the wound do not set in until several days have elapsed, we must be very cautious in stating the prognosis during the first few days after the injury. In general, the nature of the wound and the facts as to the presence of a foreign body in the eye serve to determine the prognosis. With regard to the former, we must take into consideration the situation and extent of the wound, and the condition of its edges; also whether or not the inner tunics of the eye have prolapsed into the wound, and how much, if any, vitreous has escaped. Large wounds with extensive prolapse of the inner tunics of the eye are always followed by inflammation and by shriveling of the eyeball. The question in regard to foreign bodies is often difficult to decide. If there is a foreign body in the eye and it cannot be removed at once, the eye is almost always lost. Again, in stating the prognosis, the danger which threatens the other eye, because of sympathetic inflammation, must not be forgotten.

[Wounds involving the ciliary region are held to be particularly dangerous both to the affected eye and to its fellow, since they often cause a plastic irido-cyclitis in the one and a sympathetic inflammation in the other.—D.]

The unfavorable prognosis which perforating wounds of the sclera generally offer holds good, at least in part, even for those cases in which, to begin with, there is a smooth healing of the wound. Such eyes, which often recover from the injury with the restoration of good sight, nevertheless not rarely become blind afterward because of secondary changes, which are the consequences of the cicatrix in the sclera. If the uvea is incorporated in the scar, this may give rise to inflammation or increase of tension. In scleral wounds which lie farther back, in the region of the retina, the latter may become attached to the cicatrix. By subsequent contraction of the cicatricial tissue the retina is drawn more and more into the cicatrix, and thus is loosened from its bed; the eye grows blind from detachment of the retina (Von Graefe). In this way, too, many eyes are destroyed which have been operated upon with apparently brilliant success by section of the sclera, as for the extraction of a foreign body or of a cysticercus. A further danger accrues to the eyes from the fact that the scleral cicatrices later on readily become ectatic, and lead to the formation of scleral staphylomata and also to increase of tension.

The prognosis must be stated as almost absolutely unfavorable when a *foreign body* has been left in the eye. A series of cases, to be sure, is known in which a foreign

body has been carried about in the eye for years without causing injury. But, in comparison with the extreme frequency of such injuries, the number of these cases is infinitesimally small; and even in these cases the safety of the eye is by no means to be considered as permanently assured. As an example, the following case that I observed may be adduced: A young lady of twenty-five years of age was injured by the percussion cap of a child's gun exploding near her eye. A piece of the copper case of the cap penetrated into the left eye through the cornea; it could be seen lying upon the lowermost part of the iris. The immediate consequence of the injury was an iritis, which, however, after some weeks got well, leaving several synechiæ. From that time on the eye remained free from inflammation and had good visual power. The piece of metal, which had a length of about 1 mm., could always be seen lying upon the iris, only it gradually assumed a black color. It was not until ten years after the injury that the sight began to diminish, and the patient was tormented by photopsiæ; she also complained that objects directly looked at seemed to move and look bent, so that straight lines, for example, appeared wavy to her. The eye was still free from inflammation, but, after rather prolonged examination, showed a slight ciliary injection. Examination with the ophthalmoscope could not demonstrate any changes except that the fundus in its lower half was not as beautifully red as it was above, but was of a light grayish color. It was hence taken for granted that here was a detachment of the retina in its very earliest stage. The traction thus produced upon the retina accounted for the photopsiæ, and the undulatory movement of the retina accounted for the apparent movement of objects, while the curved appearance of straight lines was referable to the difference of level existing in the detached retina [retinal metamorphopsia—see page 125]. Conjecturally the retinal detachment was produced by an old exudate which lay upon the ciliary body and the most anterior portion of the retina, and which by its gradual shrinking drew the retina farther and farther forward. Since then I have not had an opportunity of seeing the patient again, but the subsequent course of the case may be predicted with great probability. The retinal detachment will have become total; afterward probably an irido-cyclitis will have developed out of the condition of ciliary irritation of the eye, so that after some time the eye would become completely blind, would grow softer, and would be the site of frequently recurring attacks of pain and inflammation.

291. Treatment.—[In consideration of the great frequency of eye injuries (page 339), every attempt should be made to *prevent* them, especially by the use of suitable eye guards (page 53).—D.]

When we get a recent perforating wound to treat, which is so extensive that we have no prospect of retaining the eye in a serviceable condition, we advise the patient to have enucleation done at once (*primary enucleation*). By this he is saved from a protracted illness as well as from sympathetic disease of the other eye.

If the injury is such that there is a *prospect of retaining an eye* that will be able to see and there is no indication of the presence of a foreign body in the interior of the eye, we cleanse the wound from any adherent dirt with moistened pledgets of cotton. If the iris has prolapsed into the wound, which can be the case only in wounds situated in the cornea or in the most anterior portion of the sclera (Figs. 132, 133), it must be carefully excised. But if the ciliary body or chorioid projects into the wound, they should not be removed, because, if they are, the vitreous would prolapse. Small

wounds soon close of themselves by cicatrization; large gaping wounds should be united by sutures passed either through the edges of the sclera itself (only, however, through its superficial layers) or, better, through the conjunctiva overlying it. [They may also be covered in with conjunctival flaps (§ 823).] After we have thus treated the wound we instil atropine into the eye, put the patient to bed, and apply a bandage or, if there are signs of beginning inflammation, apply iced compresses. [These should be used without intermission. Mercury (especially calomel in broken doses) followed by or combined with large doses of the salicylates may be used; also dionin in solutions of ascending strength (De Schweinitz). If infection has begun, the use of a suitable vaccine occasionally succeeds in aborting it.—D.]

If in spite of the treatment the inflammation progresses, we do an enucleation (*secondary enucleation*), as soon as we see that the eye is lost so far as sight is concerned. How do we recognize this? The future of the eye depends not so much on the changes taking place in the anterior chamber as upon the exudation in the vitreous cavity, which leads to blindness and subsequently to shriveling of the eye. But exudation in the vitreous is just the thing that we cannot see directly, but can only infer, and we infer it from two facts, viz.: (1) Steadily diminishing function of the retina—this, because the latter is always implicated when there is exudation in the vitreous. In this case on repeatedly testing the light-perception and the visual field we shall find both suffering greater and greater impairment until finally the eye has lost all perception of light. (2) The gradually increasing softness of the eye, due to the shrinking of the vitreous exudate. When, therefore, these two symptoms have made their appearance the time for enucleation has arrived, and we should not delay too long about it either (not more than two or three weeks from the date of the injury), since otherwise we might be caught unawares by an outbreak of sympathetic inflammation in the other eye.

If there is a *foreign body* in the eye, we must in the very first place endeavor to remove it [either by instruments introduced into the eye or by a magnet (see §§ 846 and 847)]. For this it is requisite to know, at least approximately, the situation of the foreign body (except in the case of fragments of iron, which the magnet withdraws even from an unknown depth).

If there is no prospect of our being able to remove the foreign body, we may, as a matter of experiment, wait a while to see if possibly it will be tolerated by the eye without inflammation. This is especially apt to be the case when a foreign body is imbedded in the lens; the latter becomes cloudy, and consequently, when all the inflammatory symptoms have passed off, can be removed by a cataract operation along with the foreign body contained in it. As soon as plastic irido-cyclitis has set in, it is advisable not to make any more attempts at removing the foreign body; absolutely the only indication in this case is enucleation.

[The removal of a foreign body is often fraught with difficulty and may be associated with so much traumatism that the eye is lost at once. If not, it may be lost at a variable time later through detachment of the retina, irido-cyclitis or panophthalmitis. But many eyes retain their sight permanently or for a long time. The translator has seen a case in which, except for a traumatic cataract, the eye was normal 26 years after the extraction of a piece of iron from the retina.—D.]

292. Compensation for Injuries.—When the sight is permanently impaired as the result of an injury the injured man suffers an impairment of his earning capacity. In Germany and in Austria, a compensation has been fixed for such cases by the laws regulating accident insurance whenever the injury has happened in an industry in which insurance is obligatory. In order to determine the amount of indemnity the opinion of an expert physician is required. Such a physician has to decide: 1. Whether the disabilities alleged to exist by the injured man are actually present or not, and which of them are to be ascribed to the accident. 2. What impairment in earning power the person in question has suffered in consequence of them.

To decide the first point we must first determine by precise examination all the changes that are objectively perceptible, and then examine the function of the eye by the visual tests. If objective changes can be proved to exist, it is then to be determined which of them were produced by the alleged injury. With regard to this it must be remarked that the law awards compensation only for accidents that occur in the pursuit of an industry but not for injuries that are gradually produced by the industry, i.e., for what are called occupation diseases. For example, if a workman who is engaged in vulcanizing rubber gradually in the course of several months acquires an amblyopia through the inhalation of carbon disulphide (see § 561) there is no obligation to compensate him for the inability to work thus produced, although in such cases the law is often liberally interpreted.

The decision as to which of the changes that are found depend on the accident, is easy if one sees the injured man soon after the accident; if for instance, the physician who has to give the opinion is the same as the one who treated the injured man right after the accident. But very frequently the physician has to give an opinion on a case which he sees for the first time months after the injury. Then he must ask himself whether the changes present did not exist before the accident or, in case they followed it, whether they were not perhaps produced by disease which may have chanced to set in soon after the accident, but was independent of it. The *history* is often of no service; on the one hand, we must be prepared for statements which are intentionally wrong; on the other hand, the patients often act in good faith when they erroneously refer their eye disease to a preceding injury. Thus the entrance of dust into the eye is alleged as a cause of the most various eye diseases, even when the changes are old but had not been noticed by the patient until the occasion of his getting the dust in. The patient rubs his eye when the dust enters it and then discovers that when he closes one eye he sees little or nothing with the other. This, he thinks, has just occurred at the time and hence he regards it as the direct result of the slight accident. Once a young girl came to my clinic on account of some external affection of the eyes. The eyes were examined with the ophthalmoscope and in one of them was discovered an extensive old chorioiditis. When the tests of vision, which were thereupon made, showed that this eye was almost blind, the girl could not be dissuaded from believing that the blindness had developed as the result of the examination with the ophthalmoscope.

It may also happen, on the contrary, that the disease has been produced by an accident although the patient has no information to impart with regard to it. This happens especially in industries in which dust or small foreign bodies fly into the workmen's eyes so often that they no longer take any account of them. But the consequence of such an inconsiderable injury may be an ulcer; or a traumatic cataract may develop long after a small metallic particle has penetrated into the eye, and that, without any notice having been taken of it.

In some cases it is advantageous to take the statements of the fellow workmen.

Having due regard, then, to the unreliability of the history we have left to us the *objective findings*, from which we conclude, first, whether the changes present are of such a kind that they could be produced by an injury at all, and, second, whether the alleged accident was just the sort of one that would be apt to produce such changes. Scars in the cornea produced by incised wounds look different from those caused by ulcers; the results of a contusion of the eye (rupture of the sclera, iridodialysis, luxation of the lens, etc.), again, are different from those of incised or punctured wounds. However, it is not to be forgotten that the accident may also entail indirect consequences which produce objective changes quite different from those produced from the injury itself, as, for instance, when an ulcus serpens develops in consequence of an erosion of the cornea.

If we find changes which may have developed either as the result of injury or spontaneously, we shall still in most cases be able to judge about how long these changes have existed, and from this decide whether they are to be brought into relation with the accident, which has occurred at a determinate time in the past. Thus, old scars of the cornea look different from recent ones; if an eye has been affected with diminution of its visual power for quite a long while it often squints, etc.

Often not only careful examination but also great experience is required in order to judge accurately regarding changes when they are rather old, and this is still more so when we are dealing with those cases in which *no objective changes* can be discovered at all. These cases are divided into two groups. To the first group belong those cases in which anatomical changes are actually present but escape detection. Such changes may be so minute that they are not demonstrable with our present appliances, for example, in the case of an amblyopia produced by a lesion of the fovea centralis due to dazzling from strong light or produced by a small hæmorrhage which has already become absorbed. Or, the changes may lie behind the eye, for example, in the optic tracts as the result of fracture of the base of the skull. Careful examination of the function and prolonged observation of the case will often suffice to clear the matter up. But the real test of an expert are those cases in which it is a question of purely *functional disorders*. Such are the cases in which the function of the eyes is affected simply by shock, or the psychic commotion that accompanies the injury. These cases are known by the name of traumatic neurosis, and, so far as their essential nature is concerned, belong to hysteria; hence the disturbances of sight that are present must be characterized as hysterical amblyopia and hysterical asthenopia, produced by an injury. The symptoms of these conditions are described in § 579. If we find them in a workman who has been injured only a little while and has not yet had any medical examination of his eyes, we shall be able to make the above diagnosis. But it is otherwise when we have to do with patients, especially those who have been through a railroad accident, who make claims for big damages and have already been examined a number of times, particularly so if they belong to the cultivated classes. Then there is the danger that the symptoms have been suggested to the patients by the repeated examinations or that they become more and more conversant with the symptoms, about which they have been questioned, and then intentionally simulate them. Since

in default of objective finding we are altogether dependent on the patient's statements, it is in many cases absolutely impossible to detect a clever simulant. Cases of this sort are constantly accumulating and require the greatest precaution.

293. The second thing that the expert has to do is to answer the question as to how far the *earning power* of the injured man has been affected. For this purpose the function of both eyes must be carefully determined by visual tests. In doing this we must have in mind the possibility of exaggeration. With some experience it is not difficult to estimate the degree of visual disturbance that ought to correspond to the objective changes.

Both eyes may have been injured by the accident, or in case only one was affected by it, the other may have been already bad. In both cases the total visual power is diminished, and it will be easy to determine for what kind of work the visual power that is left should still about suffice. Much more difficult is it to gauge the working capacity when one eye has been rendered blind but the other is normal. Then the total visual power is also normal, but the special advantages that belong to *binocular vision*, namely the binocular field of vision, and binocular perception of depth, are lost. The monocular field of vision is smaller than the binocular because it does not extend so far toward the side of the blind eye (see Fig. 271). But this will be found to be troublesome in only a few callings, and especially so since the patient by turning the head to the blind side and by abduction of the seeing eye soon learns how to get rid of the limitation in the visual field produced by the nose. Of more importance is the loss of binocular vision. A slight diminution in the visual acuity of one eye does not interfere with binocular vision, but a great one takes it away altogether. Where we are to draw the line here is a matter that varies greatly in individual cases, being dependent not only on the degree of visual acuity, but also on many other factors, such as the visual field and refraction of the worse eye, the relations of the eye muscles, etc. A frequent case is that in which a traumatic cataract has been produced as the result of an injury and has later been successfully operated upon. The visual acuity of this eye may then be rendered normal once more by a glass correcting the aphakia. Nevertheless, the eye is not adapted for binocular vision: without the correcting glass it sees too little, and, if the other eye has normal sight, the correcting glass is not borne. In such a case, therefore, binocular vision is abolished, in spite of there being a normal absolute visual power in both eyes. [This is by no means always the case. Useful binocular vision may be present even when the two eyes differ very greatly in visual acuity and in the distinctness of their retinal images. Moreover, the glass that corrects the imperfect eye may be well borne even though very different in strength from the glass worn by the other eye (§794).—D.]

Abolition of binocular vision may also occur when the injury in one eye is but slight, and yet latent disturbances of equilibrium of the eye muscles were previously present. These latent disturbances now become manifest, i.e., are transformed into a squint, and by this binocular vision is abolished (see §§ 671,686).

If we have determined that as the result of the injury binocular vision has become impossible we must further try to find out to what extent the injured man is prevented by this from doing his work. Binocular vision enables us to estimate dimensions of depth directly and with precision (stereoscopic vision). Such vision can be dispensed with in many callings, and that, too, not only in coarse sorts of work, such as labor in the fields, but also in fine work, like writing; even the watchmaker who does the most minute work with a magnifying glass does not require binocular vision. But many other kinds of work do require a precise and certain appreciation of

distances, and even such an ordinary thing as splitting wood would be extremely dangerous to the fingers of the workman if he did not have a correct judgment of dimensions of depth.

[The necessity for binocular vision in such cases has been apparently exaggerated. In chopping, hammering, wheeling a barrow along a narrow path, etc., the workman is concerned not so much with depth and distance as with direction, and this can be determined as well with one eye as with two (Fergus).—D.] By practice, to be sure, one can learn to estimate distances correctly with one eye alone, and, in fact, there are persons, who, because of unilateral blindness or because of squint, have had nothing but monocular vision since youth, and who yet are as serviceable workmen as those who have normal sight. They suffer an impairment in their working capacity only in this regard, that being one-eyed they do not secure work as readily, many employers, in fact, rejecting one-eyed persons as a matter of principle.

[In other words, they have suffered little loss in *working capacity*, but a considerable reduction in *earning capacity*—a distinction important to make. To put it mathematically (Holt), a man's earning power $E = F \times T \times C$, where F is the functional capacity of his various organs expressed as a percentage of the normal, This technical ability and training, and C his competing ability, i.e., his ability and opportunity to apply his functional capacity and technical skill to the earning of money. In the cases cited F is reduced but little, C a good deal—the men being serviceable, but not acceptable.—D.] But an older workman, if he loses an eye, frequently becomes really unserviceable for his former work because of this loss. Hence when one eye is lost and the second eye is normal, the working capacity will vary very greatly; more so than the compensation which is awarded in the individual cases and which ranges within comparatively narrow limits.

The *degree of impairment of earning capacity* is stated in percentages by the physician who is giving his opinion. In Germany in case of the entire loss of one eye the minimum estimate of this impairment for the ordinary workman is 25 per cent. At the same time it is not required that the other eye should have normal sight, but it must not be below one-half the normal. In Austria for the ordinary workman the loss of one eye is put as equivalent to an impairment of 25 per cent as a minimum, in case the other eye has normal sight, but even a small diminution in the visual acuity of this latter eye raises the percentage. For a skilled ("qualified") workman the impairment produced by loss of one eye is estimated in Germany and Austria at a higher figure, for instance at 33⅓ per cent. [A like rate obtains in France for complete loss of one eye with disfigurement. For unilateral blindness the reduction in earning capacity is held to range from 20 to 30 per cent, and for post-traumatic aphakia, after correction of the aphakic eye, from 20 to 28 per cent (Liégard and Prieur). Hertel regards the loss of earning capacity due to the loss of one eye as 25 per cent.—D.] The same rule is applied in those cases also in which even when the sight is normal there is paralysis with double vision, so that the paralyzed eye has not been rendered blind but has simply been prevented from doing its part in binocular vision. Since many workmen whose binocular vision has been impaired regain by practice after some time their former ability to work, it is proper in such cases to allow at the beginning a fairly high accident indemnity—indemnity for the period during which he is forming his new habits—which after one or two years is reduced. If both eyes have been enfeebled the amount of impairment must be estimated from the visual power of the better eye, [or rather of both eyes together], the nature of the work being also taken into consideration.

III. Ectasiæ of the Sclera

294. Partial Ectasia.—Partial ectasia of the sclera is represented by a circumscribed protrusion taking the form of a dark prominence or swelling. The sclera at this spot is thinned, so as to be readily dimpled with the point

of a sound; in consequence of the thinning, the chorioidal pigment appears through it, and imparts to the ectasia a dark, slate-gray, or bluish-black color. By means of focal illumination, light can often be made to pass through the sclera at the ectatic spot, and the coating of pigment on its inner surface can be seen through it. According to the situation of the ectasia, various forms of it are distinguished—namely:

1. *Anterior Ectasiæ* (anterior scleral staphylomata).—These occupy the portion of the sclera adjoining the cornea (Figs. 138 and 139). They appear in the beginning under the form of small, dark spots, which afterward become larger and bulge out. When several lie close together they become confluent, forming a large swelling which surrounds the cornea like an arch or ring. This swelling at various points is constricted in a radial direction by the stronger, less distended fibers of the sclera, so that in a small way it resembles the large intestine with its sacculations. The limbus, under the form of a somewhat depressed gray line, marks the boundary between the ectasia and the cornea. When the latter is also opaque and ectatic, the sharp line of demarcation between scleral and corneal ectasis is often lost, and both ectasiæ unite to form a single protuberance occupying the anterior segment of the eyeball. It often happens that an anterior scleral staphyloma exists, or, at all events, has its chief development, on one side only. Then the base of the cornea at this side is pushed forward, so that the entire cornea gets to lie obliquely. If, for instance, the scleral staphyloma occurs on the inner side, the cornea looks outward instead of straight forward (Fig. 139, *h*).

2. *Equatorial Ectasiæ* (equatorial staphylomata).—These are dark prominences in the region of the equator of the eyeball. They can be seen only when the eyeball is turned strongly toward the side opposite the staphyloma. They occur at either one or more spots upon the equator, but never surround the entire eyeball like a ring, as is frequently the case with anterior scleral staphylomata.

3. *Posterior Ectasiæ.*—These occupy the posterior segment of the eyeball, and cannot, therefore, be seen in the living eye. In respect to origin and significance, they are essentially distinct from anterior and equatorial staphylomata of the sclera. There are two kinds of posterior scleral ectasiæ:
α. The *staphyloma posticum Scarpæ.* This consists in a thinning and protrusion of the sclera at the posterior pole of the eye to the outer side of the optic-nerve entrance. If the ectasia takes on greater dimensions, the optic nerve is also involved in it (Fig. 380). This form of ectasia, as Arlt was the first to discover, is the most frequent cause of short-sightedness, because, owing to the recession of the sclera, the eyeball undergoes an elongation of its sagittal axis (axial myopia). The diagnosis of a posterior staphyloma can be made in the living eye only by demonstrating the existence of a high degree of myopia and of the changes in the fundus which accompany the

latter (§ 413). *β. Posterior scleral protrusion of Ammon.* This does not, like posterior staphyloma, lie just at the posterior pole, but below it. Contrary to the case of the other ectasiæ of the sclera, it is not acquired but congenital, being formed in consequence of an incomplete closure of the fetal ophthalmic cleft. It is found in conjunction with the formation of a cleft (coloboma) in the chorioid and frequently, also, with coloboma of the iris (see §§ 404 and 435).

Acquired ectasiæ of the sclera are designated under the name of staphylomata of the sclera, as has been done in the preceding lines, but the expression staphyloma is not applied to the congenital scleral protrusion of Ammon.

295. Morbid Anatomy.—The *anatomical structure* of scleral staphyloma is essentially different from that of staphyloma of the cornea. While the latter consists of cicatricial tissue which replaces the cornea that has been destroyed, a scleral staphy-

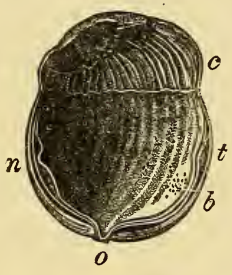


Fig. 138.

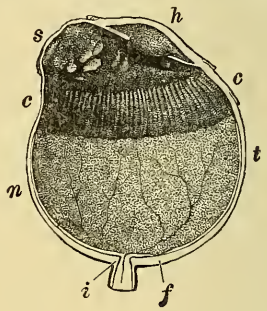


Fig. 139.

Fig. 138.—Staphyloma Ciliare. (After Pagenstecher.) The eye is bisected horizontally. Surrounding the cornea there is an ectasia, *c*, of the sclera, which attains its greatest breadth at the temporal side, *t*; and on the nasal side, *n*, is narrower and less prominent, for which reason the cornea appears displaced toward the nasal side. The inner surface of the ectasia is coated with the elongated ciliary processes; the iris is invisible because it is pressed against the posterior surface of the cornea, which hence looks pigmented in black. The retina and chorioid have been to some extent separated from their bed by the dissection; in the retina groups of punctate hæmorrhages, *b*, are observable. The head of the optic nerve, *O*, shows a deep excavation due to pressure.

Fig. 139.—Staphyloma Intercalare. The eyeball is horizontally bisected and is depicted of somewhat more than the natural size. The ectasia, *s*, of the sclera is interposed on the nasal side, *n*, between the ciliary body, *c*, and the cornea, *h*, so that the latter is displaced toward the temporal side. The inner surface of the ectasia is covered with pigment, representing the remains of the root of the iris which has become adherent to the thinned sclera; this pigment in consequence of being spread over so large a surface, shows numerous gaps. Toward the outer side the ectasia constantly diminishes in breadth, so that, at the spot where the temporal wall of the eyeball, *t*, has been cut through, nothing but a very narrow interspace is observable between the ciliary body and the iris, a condition due to that agglutination of the root of the iris to the sclera which characterizes an increase of tension. In the bisected optic nerve, *i*, no excavation is present; and, in the retina can be seen the fovea centralis, *f*, and the expansion of the retinal vessels.

loma is formed of the sclera itself, which has not disappeared at the site of the ectasis, but is simply thinned, so that often it is no thicker than a sheet of paper. In posterior staphyloma the thinning is uniform; in anterior and equatorial staphylomata we often find that the thinning is not uniform, and commences suddenly, owing to the abrupt disappearance of the inner layers of the sclera at the margin of the ectasis. The sclera then in the spot where it bulges looks as if it had been gnawed into from the inner side, and thus deprived of its innermost layers (Fig. 124, *a*). Probably this is owing to the fact that the innermost layers of fibers of the sclera, in consequence of the great stretching to which they are exposed, first rupture at some spot and then gradually separate from each other (Czermak and Birnbacher). The uvea is always solidly adherent to the inner surface of the ectasia, and is here so atrophic that scarcely anything is left of it but its pigment layer, which forms the dark coating of this inner surface.

Dissection of ectatic eyeballs shows that an anterior scleral staphyloma may be of two kinds—*ciliary* or *intercalary staphyloma*. The former (Fig. 138) belongs to that part of the sclera the inner surface of which is coated by the ciliary body; the latter (Figs. 139, 140), on the other hand, develops in that narrow portion of the sclera which is situated in front of the ciliary body, between it and the margin of the cornea; for the anterior border of the ciliary body, and hence, too, the root of the iris, as it springs from the ciliary body, do not correspond precisely to the sclero-corneal junction, but lie somewhat behind it (Figs. 142 and 147)—that is, the most anterior portion of the sclera, which lies in front of the root of the iris, belongs to the anterior chamber. But although it is just in this portion that an intercalary staphyloma develops, the iris does not lie behind the latter but in front of it, just as in the case of a ciliary staphyloma. This comes to pass in the following way: The formation of the ectasia is preceded by increase of tension, which causes the most peripheral portion of the iris to be pressed forward and to become united with the sclera (see § 450 and Figs. 215 and 216). Hence that part of the iris lying free in the anterior chamber is given off from the sclera at a

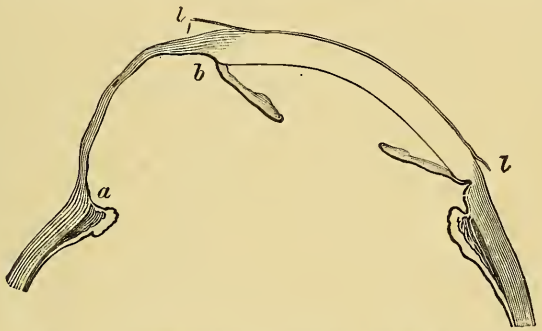


Fig. 140.—Intercalary Staphyloma. Magnified 4 × 1.

The figure represents a vertical section through the anterior half of the ectatic eyeball, which presents a great resemblance to the eye shown in Fig. 139, except that the most marked ectasia in the present case is situated above the cornea. The limits of the cornea are marked by the limbus *l* and *l*$_1$. At *l* may be seen how the root of the iris is applied to the sclera, and the beginning of a process of thinning in the sclera can be made out, while on the other side of the eye there is a fully developed intercalary staphyloma, which extends from *a* to *b*, and which in the living eye formed a dark translucent prominence. In the region of the staphyloma the sclera is reduced to half its normal thickness, and its inner surface is covered with a thin pigment coating representing the remains of the iris. The iris is adherent to the sclera from the ciliary body, *a*, to the anterior border, *b*, of the ectasia. The ciliary processes, owing to atrophy, are flatter than normal.

point farther forward than usual. Looked at with the naked eye, it seems as if the insertion of the iris had been pushed forward, up to the sclero-corneal junction or beyond it. Now, an intercalary staphyloma develops precisely in that region of the sclera which is united with the periphery of the iris—i.e., at *l* in Fig. 140, where the beginning of such an ectasia may be made out from the fact that just in front of the point where the iris is given off the innermost lamellæ of the sclera have separated and the iris has been pushed into the gap. Hence, too, later on and even when it has grown to be so very large, the ectasia always lies between the real origin of the iris at the anterior border of the ciliary body (Fig. 140, *a*) and its apparent origin at the spot where the portion of the iris that is yet free commences (Fig. 140, *b*). The inner surface of an intercalary staphyloma is coated with a layer of pigment, which is nothing but the completely atrophic root of the iris that has become united to the sclera.

In an eyeball which has not been dissected, the distinction between a ciliary and an intercalary staphyloma is more difficult to effect than in an anatomical specimen, but may still be made from the following diagnostic points: In intercalary staphyloma the anterior ciliary vessels are seen emerging from the sclera at the posterior border of

the ectasia, in ciliary staphyloma at its anterior border; furthermore, a thin ciliary staphyloma usually transmits light, and so admits of our recognizing the elongated ciliary processes as black striæ on its inner surface (*c*, Fig. 138).

296. Total Ectasia.—This consists in a uniform dilatation of the entire sclera, so that the eyeball is enlarged in toto. The sclera is everywhere thinned and the chorioidal pigment shows through it, so that it has a bluish-white appearance. Total ectasia can develop only in youth when the sclera is still everywhere yielding; the sclera of adults is so rigid that it can protrude only at certain weaker spots, and hence it admits of only partial ectasiæ. Total ectasia occurs most frequently at the same time with staphyloma of the cornea or with anterior scleral staphyloma. By the combination of these two kinds of ectasia a very extraordinary enlargement of the eyeball sometimes develops. Much more rarely a second, pure form of scleral ectasia is observed, in which the eye shows simply a uniform enlargement in all its dimensions—an enlargement in which the cornea also participates (megalocornea). This condition is characterized as hydrophthalmus or buphthalmus (βοῦς, ox, on account of the resemblance to the large eyes of oxen). Hydrophthalmus is either congenital or is acquired in early childhood, and is probably analogous to the glaucoma of adults, under which disease, therefore, hydrophthalmus will be treated of in detail (see § 447).

297. Etiology.—Every ectasia of the sclera is the result of a disproportion between the intra-ocular pressure and the resistance of the sclera. Either the tension of the eye is pathologically heightened or the tenacity of the sclera is diminished. The former is much the more frequent cause of scleral ectasiæ (if the posterior ectasiæ are excepted). Scleral ectasiæ develop slowly, and the disproportion between the tension of the eye and the resistance of the sclera must persist for a pretty long time before it can make the sclera become ectatic.

(*a*) The result of *elevation of the intra-ocular tension* is that every square millimetre of the interior surface of the sclera has to bear the same increase of pressure. If the sclera possessed the same constitution throughout, it would, in case it yielded to the pressure at all, expand in a perfectly uniform fashion. But some portions of the sclera are constructed less solidly than others, and these give way first to the increased pressure. These less tenacious spots are those in which the sclera has nerves or vessels passing through it into the interior of the eye, and in which, therefore, it is perforated and thinned. Chief among these places is the lamina cribrosa, and next those portions of the sclera where the venæ vorticosæ and the anterior ciliary vessels perforate it. At the site of the lamina cribrosa the sclera is reduced to a thin membrane, which is riddled with holes like a sieve, and which, under increased pressure, bulges out backward. This bulging, however, is not counted among the staphylomata of the sclera, but is designated as an excavation of the optic nerve, because the head of the optic nerve

recedes simultaneously with the lamina cribrosa (§ 439). Equatorial staphylomata develop at these spots where the venæ vorticosæ perforate the sclera, and anterior scleral staphylomata at the spots where the anterior ciliary vessels are transmitted. The other, more resistant sections of the sclera remain unchanged, even under increased intra-ocular pressure; it is only in children, in whom the whole sclera is distensible, that total ectasia develops.

The most frequent causes of the elevation of intra-ocular pressure are glaucoma, seclusio pupillæ, and ectatic cicatrices of the cornea. In glaucoma, in which the venæ vorticosæ are the main seat of congestion and inflammation, equatorial staphylomata generally develop; seclusio pupillæ and staphylomata of the cornea, on the contrary, in which the inflammation expends itself upon the most anterior sections of the eyeball, mostly induce anterior ectasiæ of the sclera.

(*b*) The result of *diminished resistance of the sclera* may be that the latter is unable any longer to withstand even the normal intra-ocular pressure. Diminished resistance develops in consequence of inflammations of the sclera, and hence occurs in the deep form of scleritis, which leads to anterior scleral ectasiæ (page 336); it also occurs when tumors (malignant new growths, gummy or tuberculous nodules) develop in or beneath the sclera. Injuries of the sclera also diminish its tenacity, and hence the cicatrices after penetrating wounds (and especially after ruptures) of the sclera very frequently become ectatic. Scleral ectasiæ arising in this way lead subsequently to elevation of the intra-ocular pressure, which then, however, must be regarded not as the cause, but as the result of ectasis, even though it does contribute to make the latter larger still. Here, then, the same process that occurs in ectasiæ of the cornea (page 327) is repeated.

Posterior scleral ectasiæ are likewise referred to a diminution in the resistance of the sclera. With regard to the development of staphyloma posticum, the cause of it is assumed to be a congenital weakness of the sclera in its posterior portion. With respect to Ammon's scleral protrusion, the idea is held that the fetal ophthalmic cleft is filled up with a sort of intermediary tissue which does not possess the firm texture of the normal sclera, and hence gives way before the ocular pressure.

298. Consequence of Scleral Ectasiæ.—In anterior and equatorial staphylomata of the sclera the sight is at length completely destroyed through rise of tension. If the ectasia does not come to a stop, the enlargement of the eyeball keeps growing greater and greater. The eyeball projects far beyond the palpebral fissure, can be covered but incompletely by the lids, and is extremely disfiguring. Conjunctival catarrh, lachrymation, and blepharospasm develop as a result of the mechanical irritation, and not infrequently the lower lid is pushed so far out by the enlarged eyeball as to be everted (ectropion). Finally, some slight injury suffices to cause the rupture of the staphyloma at a particularly thinned-out spot. The greater part

of the liquefied vitreous is evacuated, and in consequence a violent hæmorrhage may take place, and the eye may undergo destruction with the symptoms of panophthalmitis.

Anterior and equatorial ectases usually entail further changes in the interior of the eye. In consequence of the enlargement of the ring formed by the ciliary body, the iris becomes stretched and atrophic, and may even in places be separated from its insertion (spontaneous iridodialysis). The same is true of the zonule of Zinn, which, through atrophy, gets to be so deficient that the lens becomes tremulous or even undergoes luxation. The ciliary body, chorioid, retina, and optic nerve become atrophic; the latter generally presents a deep excavation due to the increase of tension (Fig. 139,*O*).

Staphyloma posticum, if it enlarges, causes a considerable increase in the short-sightedness, without, however, inducing elevation of tension and the other deleterious consequences of anterior and equatorial staphylomata. The scleral protrusion of Ammon remains stationary and entails no injurious consequences.

299 Treatment.—It is only anterior and equatorial, not posterior, ectasiæ of the sclera that are amenable to treatment. In the former, which, in the great majority of cases, have developed in consequence of an increase of tension, the main indication is iridectomy, provided that it is still technically practicable. Inasmuch as this operation diminishes the intra-ocular pressure, it puts a stop to the further enlargement of the scleral ectasiæ (and in especially favorable cases even causes diminution in the size of an ectasia already existing), and likewise preserves the sight, so far as it still exists, from total destruction. If, as indeed is generally the case, iridectomy is on technical grounds no longer practicable, there is nothing else left to do but enucleation, in case the eye distresses the patient by its size, its painfulness or the disfigurement it causes.

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300 Pigmentation of Sclera.—[Extensive pigmentation of the sclera may occur as a rare anomaly (Smyth). For the blue (thin) sclera see page 333.—D.]

Ulcers and Tumors of the Sclera.—The sclera is not very apt to become inflamed, and still less are the products of its inflammation apt to undergo purulent disintegration; thus, for example, ulceration of scleritic nodules is never observed. Ulcers which originate in the adjacent part of the cornea are always arrested as soon as they reach the sclera; nor are ulcers of the conjunctiva any more likely to extend to the sclera beneath them. Hence ulcers in the sclera are among the greatest of rarities. They originate from injuries with coincident infection and also from the disintegration of new growths (syphilitic, tuberculous, and leprous nodules, malignant neoplasms).

New growths, too, occurring primarily in the sclera are very rare; although tumors originating in other parts of the eye do pass over to the sclera. Fibromata, sarcomata, and osteomata are the primary tumors that have been observed in the sclera. [Perrod has reported a case of scleral cyst.]

CHAPTER IV

ANATOMY AND PHYSIOLOGY OF THE UVEA, EMBRYOLOGY OF THE EYE

I. Anatomy

301. If we carefully remove the sclera and cornea from an eyeball, we have presented to us the iris, ciliary body, and chorioid in connection. Together these form the middle tunic of the eye, which takes the shape of a sphere, colored dark brown by the pigment which it contains. In front this has a large aperture, the pupil; behind, it has a small one, the opening designed for the transmission of the optic nerve. On account of the similarity of the dark sphere, hanging upon the optic nerve as upon a stalk, to a grape (*uva*), the middle tunic of the eye has received the name of *uvea*, and also of uveal tract.

(*a*) *Iris*

302. The iris[1] is a disk-shaped membrane, perforated in the center by the pupil.[2] By its peripheral or ciliary border it springs from the anterior surface of the ciliary body. From this point it stretches over the lens, its central or pupillary border lying upon the anterior capsule, and gliding upon it with the movements of the pupil (Fig. 142). By lying in this way upon the lens, the iris obtains a firm support. Hence, when the lens is absent or has lost contact with the iris, the latter is seen to tremble or vibrate with movements of the eyeball (tremulousness of the iris, *iridodonesis*[3]). Since the umbo of the lens lies farther forward than the spot where the iris originates in the ciliary body, the iris forms a shallow cone, whose apex, directed forward, is cut off short by the pupil. The shallower the anterior chamber becomes through advancement of the lens, the greater is the altitude of this cone; if, on the other hand, the lens is absent, the iris extends in a plane.

In looking at the iris with the naked eye, or, still better, with the magnifying glass, we recognize in it delicate markings, which are formed by elevations and depressions of its anterior surface (relief of the iris, Fig. 141). Sharp and clear in the normal eye, these markings are blurred or absolutely indistinguishable in an inflamed or atrophic iris, so that they constitute an important sign in iridic affections. The markings are chiefly formed by radially directed, projecting ridges, which are nothing but the blood-vessels lying in the stroma of the iris, and running from the ciliary to the pupillary

[1] Iris on account of its rainbow shape, not on account of its color.

[2] Pupilla properly means girl; perhaps so called because in the pupil one sees a diminutive image of himself reflected from the cornea. So, also, in old German works the pupil is named "Kindlein" (= little child). In Greek, too, the pupil is called κόρη, girl, from which the expressions corectopia, corelysis, etc., are derived.

[3] From *iris* and δονέομαι, I vibrate.

margin. Near the latter they interlace with a ring of circular ridges—the lesser circle (circulus minor) of the iris (*k*, Fig. 141). This latter divides the iris into two zones: that lying to the periphery of the circulus minor is the ciliary zone (*C*); that lying to the central side of it is the much narrower pupillary zone (*P*), which is often distinguished from the ciliary zone by a different coloration. Along the circulus minor may be noticed pit-like depressions (crypts, *c*) in the surface of the iris. Similar but much smaller openings in the anterior surface are also present at the periphery of the iris, close to its root; but these are not perceived in the living eye, partly because they are too small, partly because they are concealed by the margin of the sclera, which projects in front of them. It is only in blue eyes, especially in children, that this peripheral perforated zone becomes apparent as a dark, almost black, circle (*p*) close to the root of the iris. The pupillary margin of the iris is seen to be lined by a narrow black fringe (*r*), which stands out with especial prominence in eyes affected with cataract; for it contrasts much more forcibly with the white background of the clouded lens than with the black of the pupil of a normal eye.

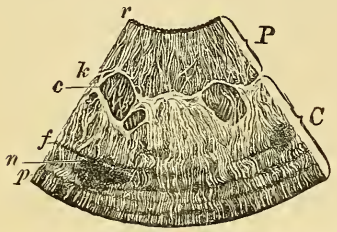


FIG. 141.—ANTERIOR SURFACE OF THE IRIS. Magnified 6 × 1.

P, pupillary zone; *C*, ciliary zone; *r*, fringe of retinal pigment; *k*, lesser circle; *c*, crypt; *f*, contraction groove; *n*, nævus; *p*, peripheral dark zone.

The examination of the iris in the living eye shows us, besides the details of relief mentioned above, a number of concentric curved lines near the ciliary margin of the iris (*f*, Fig. 141). They are particularly well seen in a dark iris with a contracted pupil, when owing to their light color they show off well upon the brown background. These are the *contraction furrows* of the iris; so called because, as the iris becomes narrower during the dilatation of the pupil, its anterior surface is disposed in folds, and depressions between the folds (*f*, *f*, Fig. 142) form the furrows in question, at the bottom of which the stroma of the iris generally contains less pigment. When the pupil contracts, these folds are smoothed down, and the furrows open out and are then easier to be seen.

EXPLANATION OF FIG. 142.—MERIDIONAL SECTION THROUGH THE ANTERIOR PORTION OF THE EYE. Magnified 16 × 1.

The boundary between cornea, *C*, and sclera, *S*, is marked at its posterior surface by the cross section of Schlemm's canal, *s*. Anteriorly it is covered by the limbus conjunctivæ, *L*; farther back the cross section of an anterior ciliary vein, *ci*, is seen in the sclera. The iris is attached by the ligamentum pectinatum, *l*, to the inner posterior wall of Schlemm's canal. Since the pupil in this eye was contracted, the iris is long and thin, and its pupillary border is drawn out into a thin edge (cf. Fig. 61). On the anterior surface of the iris may be recognized the orifices of the crypts both in the circulus minor (*cr*) and in the periphery (*c*), also the contraction furrows, *f*, *f*. The posterior surface of the iris is covered with a sheet of retinal pigment which turns forward sharply like a spur at the pupillary margin, *p*. At one spot the posterior layer, *h*, of the pigment has separated so that the anterior layer, *v*, can be seen isolated. Close to the pupillary margin, the cross section, *sp*, of the sphincter pupillæ is visible. From the posterior wall of Schlemm's canal rises the ciliary muscle, consisting of longitudinal fibers, *M*, and circular fibers, *Mu*; the transition from one portion to another is effected by the radial fibers, *r*. At the anterior margin of the circular portion is seen the cross section of the circulus arteriosus iridis major (*a*). Upon the ciliary muscle are situated the ciliary processes, *P*, which are covered by the two layers of the pars ciliaris retinæ—namely, by the pigmented cellular layer, *pe*, which is the continuation of the pigment epithelium, *Pe*, and by the non-pigmented layer, *pc*, the continuation of the retina proper, *R*. The flat part of the ciliary body, the orbiculus ciliaris, *O*, extends to the ora serrata, *o*, where the chorioid, *Ch*, and the retina, *R*, begin. Upon the orbiculus lie the fibers of the zonule of Zinn, *z*, which farther forward pass into the free portion, z_1, of the zonula and there inclose the cavity of the canal of Petit, *i*. The lens, *L*, shows at its equator besides the attachments of the zonular fibers, the cross section, *k*, of the ring of nuclei.

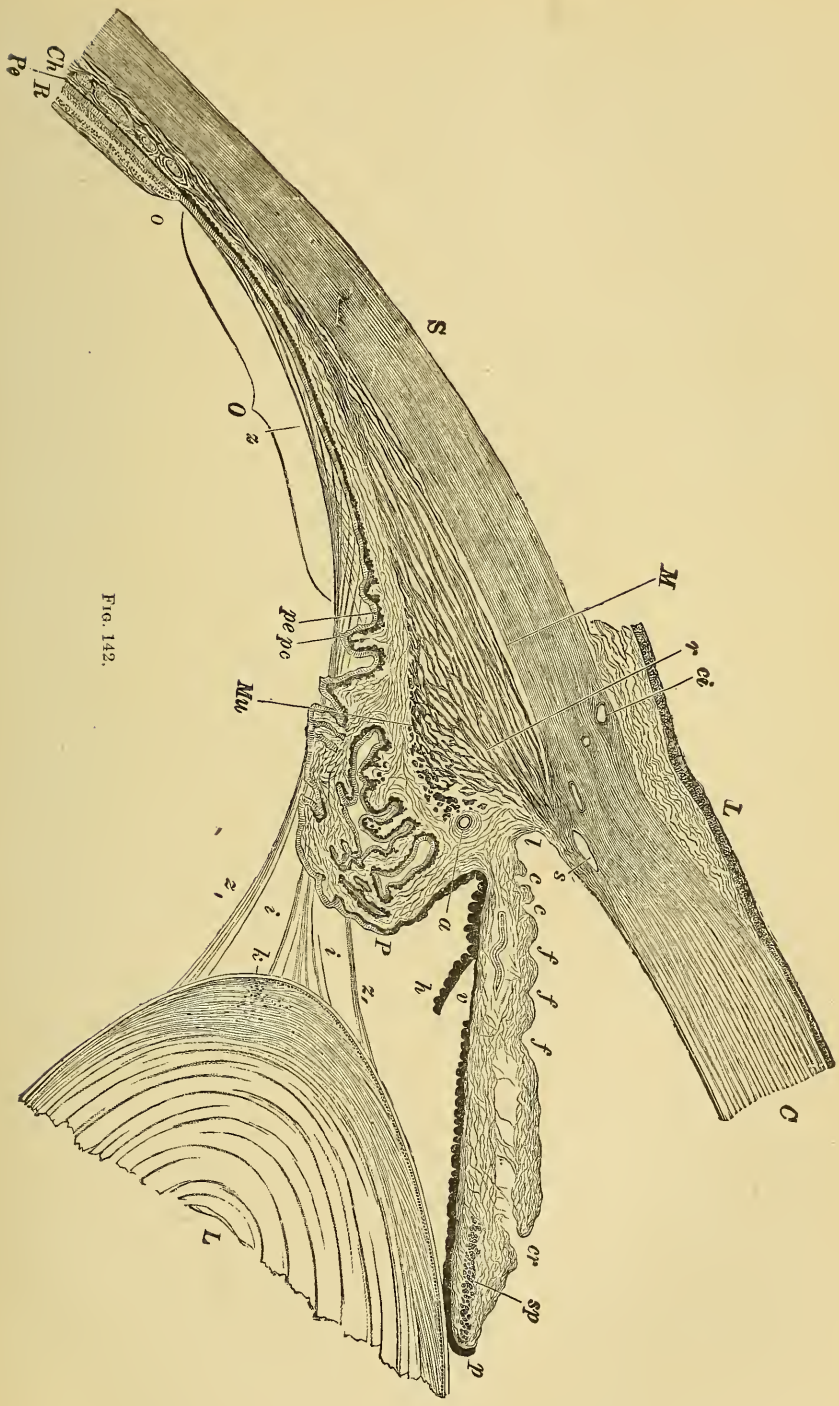


Fig. 142.

With the varying dilatation and contraction of the pupil we also notice a change in the rim of pigment upon the pupillary margin: the more contracted the pupil is, the broader this becomes; on the other hand, when the pupil is greatly dilated, it disappears entirely. (Compare Fig. 142 with Fig. 61.)

When the pupil is very much contracted, we not infrequently observe even in normal eyes a faint tremulousness of the iris (iridodonesis), which otherwise occurs only in dislocation of the lens. This is due to the fact that with a contracted pupil the posterior chamber is deeper and at the same time the greatly dilated iris is considerably thinned—circumstances both of which favor wavering of the iris.

303. Microscopical Anatomy.—The iris *stroma* consists essentially of numerous vessels running in a radial direction from the ciliary to the pupillary margin. The vessels are inclosed in a thick adventitia, and are surrounded by a loose meshwork of branched and pigmented cells, which fill up the interspaces between them (Fig. 164). The vessels, together with the cellular meshwork, form the stroma of the iris, which consequently is a very loose, spongy sort of tissue. Close to the pupillary margin of the iris the muscle which closes the pupil—the *sphincter iridis*—is found imbedded in the stroma (Fig. 142, *sp*). This is a flat band of smooth muscular fibers, 1 mm. broad, lying close to the posterior surface of the iris.

On the *anterior surface* of the iris there is a specially dense layer of cells (anterior limiting layer, Fig. 164, *v*). Next to this is a layer of endothelium, which is a continuation of the endothelium of Descemet's membrane, and covers the entire anterior surface of the iris as far as the pupillary margin. It is deficient only at those spots which correspond to the crypts, including both those at the pupillary (Fig. 142, *cr*) and those at the ciliary margin (*c*, *c*). These crypts, therefore, form apertures which lead into the interior of the tissue of the iris and place its tissue spaces in free communication with the cavity of the anterior chamber. This arrangement favors the rapid change in volume of the iris in the alternating movements of the pupil, since it enables fluid to pass quickly from the tissue of the iris into the anterior chamber and vice versa.

The *posterior surface* of the stroma of the iris is covered by the posterior limiting membrane and the retinal pigment layer. The former (also called Bruch's membrane, Fig. 143, *m*, and Fig. 147, *h*) consists of fibers, which extend in a radial direction from the ciliary to the pupillary margin, and form the *dilatator pupillæ*. These are muscle fibers of a peculiar character, which, just like the fibers of the sphincter, are derived from the anterior row of cells of the retinal pigment layer (ectodermal muscle cells, Vialleton, Grynfeltt). To the posterior limiting membrane succeeds the retinal pigment layer, which coats the posterior surface of the iris. It extends to the pupillary margin, round which it turns so as to appear a little on the anterior surface of the iris (Fig. 142, at *p*), and so forms that black rim which we perceive along the pupillary margin, when we look at the eye from in front. The pigment layer consists of two strata of epithelial cells (*v* and *h*, Fig. 142,

and *v* and *h*, Fig. 143), which merge into each other at the pupillary margin. The two together, as embryology teaches us, represent the continuation of the retina to its termination at the pupillary margin (Fig. 163). This layer of the iris is therefore designated as the retinal layer (pars retinalis iridis sive pars iridica retinæ), in contradistinction to the anterior layers, which, as they belong to the uvea, are comprised under the name of pars uvealis iridis (Schwalbe).

The *retinal pigment layer* is composed of two strata of cells, the recognition of which, however, is rendered very difficult by their profuse pigmentation. It is only in the embryo (occasionally also in the newborn infant) and in the albinotic eye that the two strata can be clearly distinguished from each other without some special preparation. In other cases we must first decolorize the pigment cells artificially by some bleaching process (Fig. 143). We then can distinguish the two strata from each other and then also establish the fact that they are the continuation of the two layers of the secondary ocular vesicle upon the posterior surface of the iris. The anterior stratum of pigment arises from the pigment epithelium of the retina. From the anterior por-

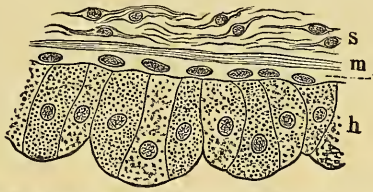


Fig. 143.—Posterior Layers of the Iris. Meridional Section through the Iris of an Adult, Decolorized by Fick's Method. Magnified 300 × 1.

To the posterior layers of the stroma of the iris, *s*, with its branched cells, succeeds the dilatator or the posterior limiting membrane, *m*, whose posterior surface is lined by the protoplasm and the oblong nuclei of the anterior stratum *v*, of the retinal pigment layer. In this stratum the boundaries of the individual cells cannot be recognized. The posterior stratum, *h*, of the retinal pigment layer, consists of elevated cylindrical cells with round nuclei. The cells are of different heights, and are so grouped that their posterior surface in section forms a series of rounded eminences such as also are to be found on a smaller scale in the iris shown in Fig. 142. These eminences represent the cross section of the dark brown, annular projections, running concentrically with the pupil, which the iris presents on its posterior surface when examined with the naked eye. The posterior border of this layer of cells is characterized by its peculiarly sharp contour, which does not, however, in this spot assume the appearance of a true limiting membrane. The cells of the posterior layer still contain a few pigment granules which have escaped decolorization.

tion of the protoplasm of these cells develop the fibers of the dilatator (Fig. 143, *m*), while the nuclei remain in the posterior, pigmentiferous portion of the cells (*v*). The posterior stratum (*h*) is the continuation of the retina proper (Fig. 163). In pathological cases a separation not infrequently takes place between the two strata, because they are not attached with the same degree of firmness to the iris. While the anterior stratum is very intimately adherent to the posterior surface of the iris, the posterior stratum readily becomes separated from it (in Fig. 142 the separation has resulted accidentally from the dissection). When, for example, adhesions of the posterior surface of the iris to the capsule of the lens (posterior synechiæ) are torn away the posterior stratum is left as a black coating upon the anterior capsule, while the anterior stratum remains upon the iris. So, too, by penciling the iris we can easily remove the posterior stratum, leaving the anterior stratum behind upon the posterior surface of the iris. Then, when we make a microscopical examination of the iris that we have penciled, we find the anterior stratum intimately connected with the posterior limiting membrane.

304. The *color* of the iris, which is either light (blue or gray) or dark (brown), is caused by the iridic pigment. There are two kinds of pigment

in the iris: one lies in the branched cells of the stroma, and is hence called the stroma pigment; the other fills up the epithelial cells of the retinal pigment layer (retinal pigment). Upon the proportion between the amount of pigment deposited in these two the color of the iris depends. The retinal layer of the iris always abounds in pigment, while the amount of stroma pigment that the iris contains varies greatly. When the stroma contains little pigment, the retinal pigment shows through the thin iris, and appears blue. This is due to the same phenomenon that causes a dark background always to appear blue when looked at through a more or less opaque medium. Thus, for instance, through a delicate skin the veins look blue. If the stroma is deficient in pigment, but pretty thick and compact, the iris appears gray. And, finally, the greater the amount of brown stroma pigment that the iris contains, the more this pigment becomes visible and makes the iris appear brown, while the retinal pigment layer, which lies behind, is more and more concealed by the stroma pigment and withdrawn from view.

Not infrequently in an iris, that is but slightly pigmented as a whole, one or two isolated accumulations of pigment are found in the stroma. These then stand out as dark (rust-colored, brown, or black) spots in an otherwise gray or blue iris (nævi iridis, *n* in Fig. 141 and Fig. 61). The presence of a pretty large number of them gives the iris a mottled appearance. Exceptionally, cases occur in which the iris has no pigment either in its stroma or in its retinal layer. Such an iris is found in albinos; it is translucent, and, on account of its numerous vessels, has a delicate, grayish-red color.

As is universally known, the *color of the iris changes* in the first years of life. Most children are born with a deep-blue iris. The stroma contains but little pigment and is still very thin, so that the posterior pigment layer is seen through it, having a bluish look. With increasing age the stroma becomes thicker and thicker. If, while this is taking place, the pigmentation does not increase, the iris simply becomes of a light blue or gray; but if, simultaneously, there is an increase of the pigment of the stroma, the iris takes on a brown color. The transformation of a blue iris into a brown one is sometimes confined to a part of the membrane, so that a brown sector is seen in an otherwise light-colored iris. Moreover, the iris of one eye may be blue and that of the other brown. [This is called *heterochromia iridis.*] In such cases the eye with the lighter iris often becomes affected with cyclitis and cataract. [See § 490.]

The color of the iris is always proportioned to the pigmentation of the rest of the body. The dark races always have a dark iris.

(*b*) *Ciliary Body*

305. The ciliary[4] body is brought into view when the eyeball is bisected, and the vitreous, the lens, and the retina are removed, so that the uvea is everywhere exposed. The spot where the retina is torn away anteriorly is marked by a jagged line—the ora serrata (*o*, *o*, Fig. 144). Corre-

[4] From *cilia*, lashes, because of the fine, radiating folds. The ciliary body is also called *cyclon* (hence cyclitis) from κύκλος, a circle.

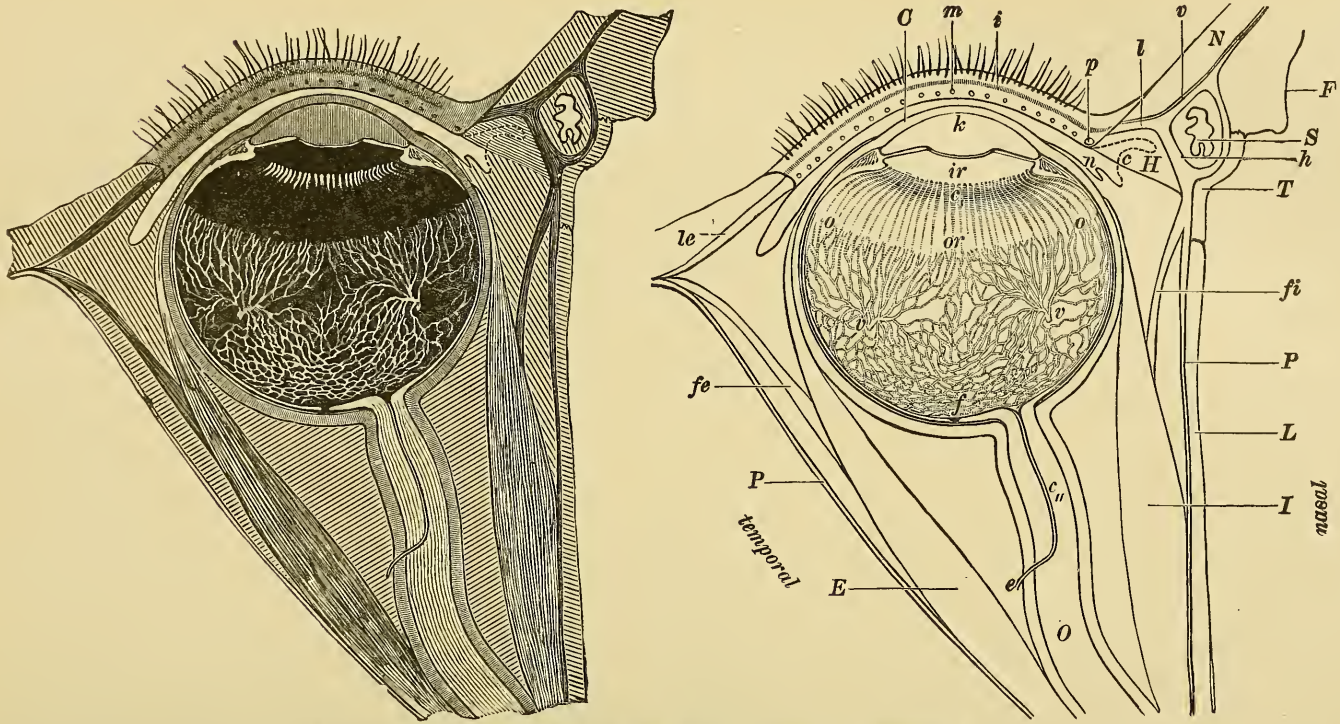


FIG. 144.—HORIZONTAL SECTION OF THE ORBIT. (SCHEMATIC.) Magnified 2 × 1.

The nasal wall of the orbit is formed by the lamina papyracea (os planum) of the ethmoid, *L*, the lachrymal bone, *T*, and the frontal process, *F*, of the superior maxilla. The last two bones bound the fossa sacci lacrimalis, in which lies the lachrymal sac, *S*. The bony walls of the orbit are coated by a periosteum (periorbita), *P*, from which the palpebral ligaments take their origin. The internal palpebral ligament, *l*, divides into an anterior limb, *v*, and a posterior limb, *h*, which together inclose the lachrymal sac. From the posterior limb arise the fibers of Horner's muscle, *H*. *le*, external palpebral ligament; *fi* and *fe*, the slips of fascia [check ligaments], likewise passing from the periosteum to the internal rectus muscle, *I*, and the external rectus, *E*. The skin, *N*, of the dorsum of the nose passes into the lower lid, at whose free border are seen the cilia and the orifices of the Meibomian glands, *m;* between the two extends a gray line, *i*. At the inner extremity of the lid lies the inferior punctum lacrimale, *p*, and farther along in the conjunctival sac the caruncle, *c*, and the plica semilunaris, *n*. From the eyeball, the lower half of which is presented to view, the lens and along with it the vitreous have been taken out, and the pigment epithelium has been removed by penciling. The anterior chamber, *k*, the iris, *ir*, and the ciliary body, consisting of the corona ciliaris, *c*, and the orbiculus ciliaris, *or*, are visible. Back of the ora serrata *o*, is the chorioid with its veins which are aggregated into vortices, *v*. *f*, fovea centralis retinæ; c_{II}, central vessels of the optic nerve, *O*, entering it at *e*.

sponding to this there is a change in the coloration of the uvea, which behind this line is brown (chorioid), in front of it black (ciliary body). At the anterior margin of the black zone rise the ciliary processes, about seventy in number. These are conspicuous not only by their prominence, but also because of their lighter color, their apices being less strongly pigmented than are the depressions between them. The anterior zone of the ciliary body, bearing the ciliary processes, is called the folded part of the ciliary body—corona ciliaris (*c*, Fig. 144); back of this is the posterior part of the ciliary body—pars plana or orbiculus ciliaris (*or*)—which is smooth and of a uniform black color.

If we strip off the entire uvea from the cornea and sclera, we get a view of the outer side of the ciliary body. This is covered by a layer of gray tissue—the ciliary muscle.

Longitudinal sections (i. e., those carried in a meridional direction, Fig. 142) are the ones best adapted for accurate study of the ciliary body. In such sections the ciliary body appears triangular. Its shortest side looks forward, and at about its center gives origin to the iris. The two long sides of the triangle look inward and outward respectively. The inner side bears the ciliary processes (*P*, Fig. 142), while the outer side is formed by the ciliary muscle (*M*).

306. Ciliary Muscle.—If we examine the separate layers of the ciliary body, proceeding from without inward, we first come upon the ciliary muscle. This was discovered by Brücke, and was called by him the tensor chorioideæ. It consists of two portions, distinguished by the differing direction of their muscular fibers. (*a*) The external portion contains the longitudinal or meridional fibers—that is, those running from before backward (*M*, Fig. 142). As these are the ones first discovered by Brücke, they are also called Brücke's portion. The longitudinal fibers arise from the external fibrous tunic of the eye, at the boundary between the cornea and sclera (at *l*, Fig. 142), and run straight backward to a point at which they gradually are lost in the external layers of the chorioid (Fig. 142, *Ch*). (*b*) The second portion of the ciliary muscle lies to the inner side of the first, and contains those fibers which have a circular course, and which, hence, in sections made meridionally, are seen in cross section (*Mu*, Fig. 142). They are designated as Müller's portion, from their discoverer, Heinrich Müller.

The ciliary muscle is composed of smooth muscular fibers, which do not present a compact mass but are disposed in flat bundles, which are separated by connective tissue, and which interlace repeatedly so as to form a sort of plexus. For this reason there is no well-marked separation between the two portions of the ciliary muscle; on the contrary, the longitudinal fibers by a very gradual transformation become bent so as to take a circular direction. Those bundles which effect the transition from fibers of one direction to those of another have been denoted by the name of radial bundles (*r*, Figs. 142 and 147).

The proportion between longitudinal and circular fibers varies according to the refractive state of the eye. In hypermetropic eyes the circular fibers are strongly developed, while in myopic eyes they are present in much smaller numbers (see § 771, and Figs. 383 and 384).

[The circular fibers effect the changes in the shape of the lens, upon which accommodation depends (see § 758). According to Thomson, they also act as a pump, withdrawing fluid from the aqueous chamber and discharging it into the spaces of the ligamentum pectinatum and Schlemm's canal. On this hypothesis their absence would lead to insufficient outflow and to increase of intra-ocular pressure (see §§ 452, 453).—D.]

307. Ciliary Processes.—The ciliary processes (*P*, Fig. 142) are placed upon the ciliary muscle. They consist of a connective-tissue stroma, which, along with branched pigment cells, contains an extraordinary number of blood-vessels, so that the ciliary processes must be regarded as the most vascular portion of the entire eyeball. The inner surface of the ciliary body is covered by three layers of tissue. The first of these is a homogeneous membrane, the vitreous lamina of the ciliary body (Fig. 145, *l*). Succeeding this is a layer of pigmented cells (*P*, Figs. 145 and 146); and, lastly, a single

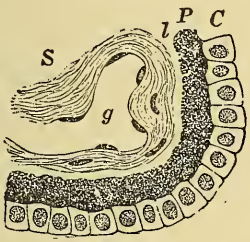


Fig. 145.

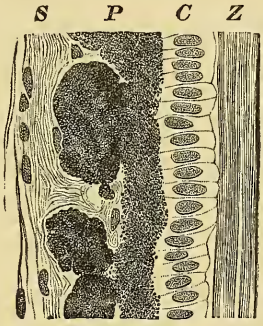


Fig. 146.

Fig. 145.—Portion of a Meridional Section through a Ciliary Process, somewhat Behind its Apex. Magnified 240 × 1. The stroma of the ciliary process, *S*, consists of delicate connective tissue in which lie the broad and very thin-walled blood-vessels, *g*. Succeeding these are the vitreous lamina, *l*, and next the two layers of the pars ciliaris retinæ. One is the pigment layer, *P*, representing the continuation of the pigment epithelium; its cells, on account of their deep pigmentation, disclose neither their nucleus nor their contour. The second layer, *C*, which is unpigmented, consists of a single stratum of cubical cells, representing the continuation of the retina proper.

Fig. 146.—Superficial Layers of the Flat Portion (Orbiculus Ciliaris) of the Ciliary Body in Meridional Section. Magnified 270 × 1. Taken from the same section as Fig. 145. The cells of the pigmented layer, *P*, of the ciliary body dip into the stroma, *S*, of the latter in the form of processes which are club-shaped or gland-shaped (but contain no gland cavity). The light area surrounding them represents the vitreous lamina, which in this case is but indistinctly visible. The cells of the superficial non-pigmented layer *C* are here longer and cylindrical. On their free surface lie the fibers, *Z*, of the zonule of Zinn.

stratum of non-pigmented, cylindrical cells (*C*) forming the most superficial layer—i. e., the one that adjoins the vitreous. The last two layers are the continuation of the secondary ocular vesicle, namely of the pigment epithelium and of the retina in the narrower sense of the word, which is here reduced to a single stratum of unpigmented cells. The two layers together are called the *pars ciliaris retinæ*. They pass over upon the posterior surface of the iris, where they are converted into the two strata of the retinal pigment layer of the iris (pars iridica retinæ, *v* and *h*, Fig. 143).

308. Ligamentum Pectinatum.—The place where the *iris and the ciliary body are attached to the sclera* deserves particular attention. We can readily

convince ourselves that the iris does not arise from the sclero-corneal junction, but farther back, so that the most anterior portion of the sclera is still in the confines of the anterior chamber. The connection between the sclera and the root of the iris is made by means of loose tissue which arises at the margin of the cornea, and from this point extends backward to the root of the iris (Fig. 147). This tissue, which is called the *ligamentum pectinatum*, fills up the angle between the iris and the corneo-sclera, so that this angle is rounded off in a sinus—the sinus of the anterior chamber. Histologically, the tissue of the ligamentum pectinatum is composed of superimposed, laminated lamellæ, which start from the margin of Descemet's membrane and run backward to a spot at which the most posterior of them serve as a point of attachment for a portion of the longitudinal fibers of the ciliary muscle. These lamellæ consist of trabeculæ inclosing rounded alveoli (Fig. 148) so as to form, when superimposed, a spongy tissue (Fig. 147). Directly to the outer side of them, just at the boundary between the cornea and sclera, is observed an open space, representing Schlemm's canal (sinus venosus scleræ), whose inner (posterior) wall is thus formed by the ligamentum pectinatum.

The region of the *angle* (or *sinus*) *of the anterior chamber* demands particular consideration, both because of its complicated anatomical relations and also because of its importance with regard to the metabolic processes and the diseases of the eye. This region was studied in the eyes of animals before it was in human eyes, and hence names were selected at that time which are still in vogue, although they are not appropriate for the human eye. Thus Hueck introduced the name ligamentum pectinatum, because he found in the eyes of the ungulata that, upon stripping the iris from the sclera, the tissue that unites these parts projects in a series of ridges resembling the teeth of a comb. The triangular space between the sclera and the root of the iris which is filled by the ligamentum pectinatum is also called Fontana's space, because Fontana was the first to describe the rather large cavities which are found in many animals between the lamellæ of the ligamentum pectinatum.

The *ligamentum pectinatum* is covered by the endothelial layer which passes over it from the posterior surface of Descemet's membrane to the anterior surface of the iris. Through the gaps in the lamellæ of the ligamentum pectinatum the endothelium passes from the surface of the ligament into the deeper parts of it, and supplies all the lamellæ and trabeculæ of this spongy tissue with an endothelial lining (Fig. 148).

The ligamentum pectinatum has two divisions that we can distinguish. The anterior is the one which runs from the edge of Descemet's membrane to the scleral spur (scleral portion or trabeculum sclero-corneale). The posterior division consists of those lamellæ which pass over into the delicate tissue of the sinus of the chamber and the root of the iris (uveal portion or trabeculum sclero-ciliare).

By stripping off the uvea, together with the ligamentum pectinatum, from the corneo-sclera, an opening is made into *Schlemm's canal*, the inner wall of which is formed by the ligamentum pectinatum. It is then visible as an open groove running along the boundary between the cornea and sclera—scleral groove.

The anterior surface of the ciliary body belongs in part to the region of the anterior chamber, and in this portion of its extent is covered partly by the most posterior lamellæ of the ligamentum pectinatum, partly by the delicate tissue of the root of the iris (Fig. 147). Hence inflammatory products, and especially pus, may pass from the ciliary body directly into the anterior chamber, traversing the tissue of the ligamentum pec-

tinatum as they do so. New growths also sometimes take this path, starting from the ciliary body and growing forward into the anterior chamber in the region of its sinus (Fig. 192).

309. Aqueous Chamber.—The iris and ciliary body take part in the formation of the two chambers of the eye. The *anterior chamber* is bounded

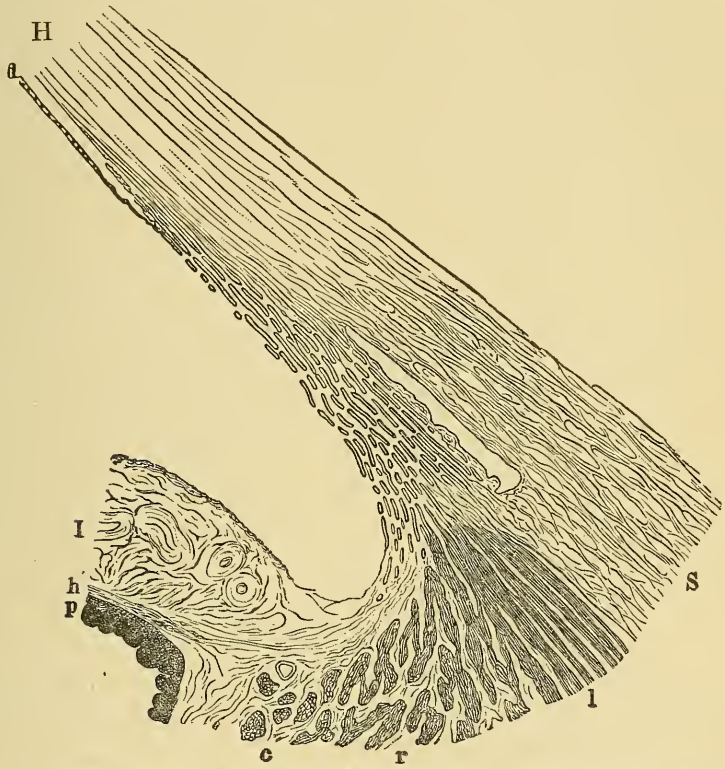


Fig. 147.—Sinus of the Anterior Chamber. Magnified 83 × 1.

H, the most posterior layers of the cornea; *S*, those of the sclera. The boundary between the two is marked by the elongated lumen of Schlemm's canal. On the posterior surface of the cornea can be seen Descemet's membrane, *d*, with its endothelium. It ends in a thinned-out edge and just before it comes to a stop the lamellæ of the cornea which lie directly in front of it can be seen to become fibrillated and to merge into the anterior lamellæ of the ligamentum pectinatum. The ligamentum pectinatum forms the posterior wall of Schlemm's canal and apparently consists solely of short fragments of fibers. For the lamellæ of the ligament which have been cut transversely by the section appear under the form of fibers which must show an interruption at all spots where the section happens to enter gaps between the lamellæ (Fig. 148). At the posterior end of Schlemm's canal is seen a bundle of circularly directed scleral fibers (represented of lighter tint in the drawing), which are divided transversely by the section, and jut out obliquely toward the sinus of the chamber (scleral spur). To the anterior surface of this spur are attached the anterior lamellæ of the ligamentum pectinatum, while its posterior surface serves as a point of attachment for the longitudinal bundles, *l*, of the ciliary muscle. The posterior lamellæ of the ligamentum pectinatum merge directly, without any interposition of scleral tissue, either into the longitudinal or into the radiating fibers (*r*) of the ciliary muscle; the most posterior (innermost) lamellæ of the ligamentum pectinatum curve backwards and are lost in the delicate tissue of the root of the iris. On the posterior surface of the iris, *I*, is seen the posterior limiting membrane, *h*, whose fibers can be followed into the root of the iris. Behind the limiting membrane lies the retinal pigment layer, *p*. *c*, circular fibers of the ciliary muscle; in front of them is the cross section of the circulus arteriosus iridis major, which here is very small.

in front by the cornea, behind by the iris and in the region of the pupil by the anterior capsule of the lens, and at its margin by the tissue of the ligamentum pectinatum, beneath which lie Schlemm's canal and the anterior

border of the ciliary body. Even under normal conditions the depth of the anterior chamber is variable. It is greatest in the eyes of the young, and diminishes with advancing age. Myopic eyes have a deep anterior chamber, hyperopic eyes a shallow one. Even in the same eye the depth of the anterior chamber varies, as it becomes shallower during the accommodative act from the protrusion of the anterior surface of the lens. The *posterior chamber* is produced by the fact that the iris is not in contact with the capsule of the lens by its whole posterior surface, but only by its pupillary margin. Thus an open space is left between the iris and the lens, which increases in depth from the pupillary to the ciliary margin of the iris, and hence in cross section has a triangular shape. This space, the posterior chamber of the eye, is bounded

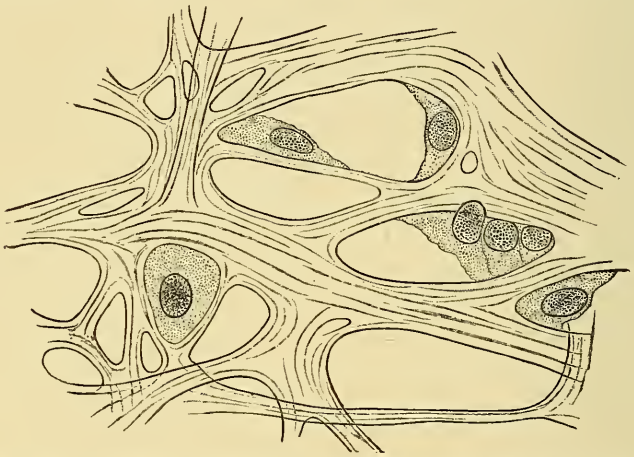


Fig. 148.—Ligamentum Pectinatum (Surface View). Magnified 700 × 1.

Trabeculæ, which show a delicately fibrillar structure, inclose alveoli, the larger of which are elliptical, and directed so that their long axis lies parallel to the margin of the cornea. Upon the walls of these alveoli lie cells (endothelial cells) provided with nucleus and large protoplasmic cell body; small alveoli are sometimes entirely filled by such cells.

in front by the iris and to the outer side by the ciliary body, while its inner and posterior wall is formed by the lens (*L*, Fig. 142) and the zonule of Zinn (z_1, Fig. 142), the latter bridging over the interspace between the lens and the ciliary body. The two chambers communicate only by means of the pupil.

It was a good while before people got a correct idea of the anatomical relations existing in the region of the anterior and posterior chamber, and even at the present time we very frequently find drawings which represent these relations incorrectly. The existence of the *posterior chamber* was for a long time contested, it being supposed that the iris came into contact with the lens by its whole posterior surface. If this were the case, the anterior chamber would present quite a different shape, since it would have to be much deeper at its periphery than it is. This state of things is actually observed in those pathological cases in which the iris is adherent throughout by means of an exudate to the capsule of the lens. The iris is then found to be retracted at its periphery much more than usual (see Fig. 173).

(c) *Chorioid*

310. The chorioid[5] is that part of the uvea which lines the posterior section of the eye from the ora serrata to the aperture for the optic nerve. If we observe it in situ, after opening the eyeball and removing the vitreous together with the retina, its inner surface appears smooth and uniformly brown. Then, if we try to strip it off from the sclera, we notice that at several spots it is attached more firmly than at others. The most intimate connection is at the margin of the aperture for the optic nerve; in addition, loose attachments exist in the places where vessels and nerves enter the chorioid from the sclera, and especially in the region of the posterior pole (region of the posterior ciliary arteries) and of the equator (venæ vorticosæ). When, after tearing away these connections, we have separated the chorioid from the sclera, we get a view of the outer surface of the chorioid, which has a shaggy appearance on account of the shreds of membrane adhering to it.

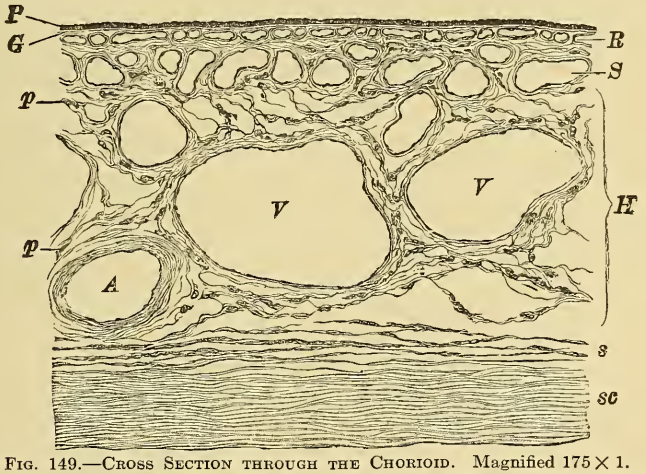


Fig. 149.—Cross Section through the Chorioid. Magnified 175 × 1.

The chorioid consists of the suprachorioid, *s*, the layer of large vessels, *H*, the layer of medium-sized vessels, *S*, the chorio-capillaris, *R*, and the lamina vitrea, *G*. In the layer of large vessels are recognizable arteries, *A*, veins, *V*, and pigment cells, *p*. The inner surface of the chorioid is covered by the pigment epithelium, *P*, its outer surface by the sclera, *sc*.

311. Microscopical Anatomy.—The chorioid consists of five layers which succeed one another in the following order, proceeding from without inward:

1. The *suprachorioid* (*s*, Fig. 149) consists of numerous minute non-vascular but richly pigmented lamellæ lying between the chorioid proper and the sclera (*sc*). Upon stripping these latter apart these lamellæ are torn in two, and are left hanging partly upon the inner surface of the sclera, partly upon the outer surface of the chorioid, which thus acquires the rough, shaggy aspect above mentioned.

[5] From χοριοειδής—i. e., like the χόριον(= Lat., *corium*). This latter word signifies "skin," and not merely the epidermis, but also the envelope (chorion) of the embryo in utero; and, in fact, it is the latter that the chorioid resembles, from its abundant supply of vessels. This word is also erroneously written choroid or choroïd.

2. The *layer of large vessels* (Haller) (*H*, Figs. 149 and 150). These are chiefly veins, which are placed very close to each other and anastomose repeatedly. The intervals between the vessels (intervascular spaces) are richly supplied with pigment cells (*p*), and are hence of a brown color. This layer, accordingly, gives the same appearance upon a surface view as if we were looking at a plexus of bright lines (the vessels) upon a dark ground (Fig. 144). This is a picture which we often have the opportunity of seeing with the ophthalmoscope in the living eye (tessellated fundus, see Fig. 26).

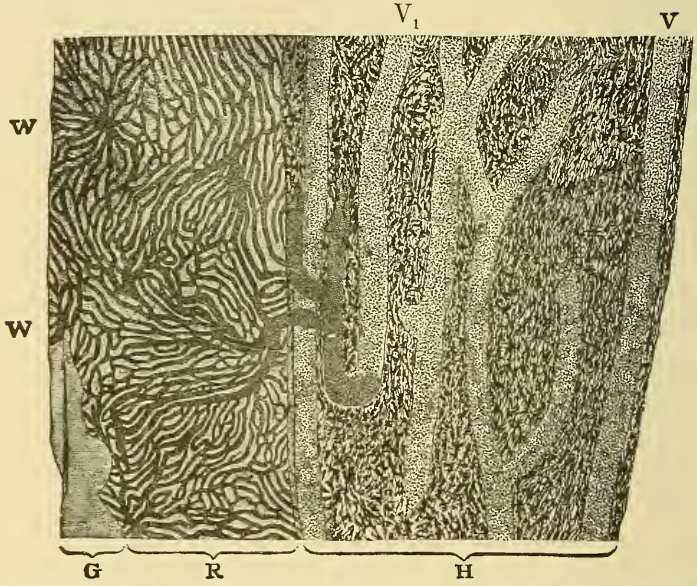


FIG. 150.—DISSECTION OF THE CHORIOID MADE PARALLEL WITH THE SURFACE. Magnified 27 × 1. The suprachorioid has everywhere been stripped off and the pigment epithelium has been removed by penciling. Furthermore in the area marked *R*, the layer of large vessels was removed so that here the chorio-capillaris lies exposed. At *H* where the layer of large vessels is still present, the large veins, *V V*1, which run approximately parallel to one another, appear like light-colored passageways, because the intervascular spaces are colored dark-brown by the presence of numerous stellate pigment cells. Some veins appear to have a blind ending, because at the point where they dip down into the opaque tissue they get out of view. The vein, *V*1, bends on itself, enters the layer of medium-sized vessels, and here receives branches from the chorio-capillaris. This bent portion of the vein and also the capillaries are filled full of red blood corpuscles, while the large veins contain but few corpuscles. As a result of this natural injection the chorio-capillaris stands out with special distinctness. The distension with blood, however, is not everywhere alike, and for this reason the capillaries in the upper part of the drawing appear thinner and separated by wider intervals than in the lower part. So, too, the blind ending of some capillaries in the drawing is only apparent, being due to the fact that in this spot short segments of the capillaries are bloodless and hence invisible. The veins on their entrance into the chorio-capillaris at once divide into a number of capillaries and thus small vortices are produced. These are seen in the continuation of the vein *V*1 and also at *W W*; at the latter spot their continuation into a small vein cannot be seen because the latter was torn off when Haller's layer was removed. At G when Haller's layer was removed the chorio-capillaris also went with it, so that here the lamina vitrea alone is present; in making the preparation the latter was somewhat folded.

3. The *layer of medium-sized vessels* (Sattler) (Fig. 149, *S*), which is very thin and but slightly pigmented.

4. The *layer of capillaries* (*chorio-capillaris*, or membrana Ruyschii—although it was not discovered by Ruysch—*R*, Figs. 149 and 150). This consists almost exclusively of capillaries which have a very wide bore, and at the same time are so closely packed together that the interspaces between

the capillaries are often narrower than the capillaries themselves. This layer contains no pigment.

5. The *lamina vitrea* (or *lamina basalis G*, Figs. 149 and 150), a homogeneous membrane coating the inner surface of the chorioid.

The chorioid is continuous with the flat part of the ciliary body (orbiculus ciliaris, pars plana), which possesses essentially the same structure as the chorioid, and is distinguished from it only by a somewhat different arrangement of the blood-vessels, and also by the absence of the chorio-capillaris, which ends at the ora serrata. The difference in color between the brown chorioid and the black orbiculus (Fig. 144), so striking to the naked eye, is not referable to a difference in the pigmentation of these parts of the uvea, but to a difference in the pigment epithelium which covers them and which belongs to the retina.

312. We may briefly summarize the structure of the chorioid as follows: The chorioid consists mainly of vessels which are arranged according to their caliber in three superimposed layers, in such a way that the largest vessels lie farthest to the outside, the smallest vessels farthest to the inside. The purpose of this arrangement is easily comprehended, since the

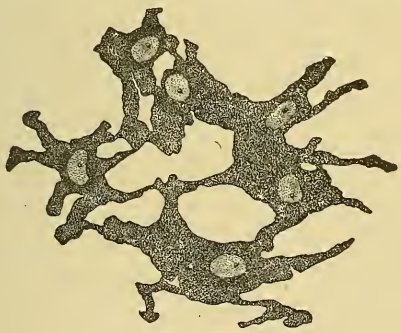


Fig. 151.

Fig. 152.

Fig. 151.—Chromatophores of the Chorioid. Magnified 400 × 1. They are branched, anastomosing, connective-tissue cells, with numerous pigment granules and an unpigmented nucleus.

Fig. 152.—Pigmented Epithelial Cells of the Retina. Magnified 500 × 1. They are hexagonal epithelial cells, with pigment granules and an unpigmented nucleus.

chorioid is in great part designed for the nourishment of the retina which lies to the inner side of it. Hence the minutest vessels, the capillaries, from which the nutrient plasma of the blood exudes, must lie upon the inner surface of the chorioid. The vascular part of the chorioid is covered on either side by a non-vascular layer—i. e., on the outside by the suprachorioid, on the inside by the lamina vitrea. All the layers of the chorioid, with the exception of the two innermost ones—the capillary layer and the lamina vitrea—contain pigment enclosed in branched pigment cells (chromatophores, Fig. 151). To its abundant supply of pigment the chorioid owes its dark-brown color. The inner surface of the chorioid is covered by the pigment epithelium (*P*, Fig. 149) which lies upon the lamina vitrea. This, too, was formerly reckoned in with the chorioid, because it remains attached to it after the retina has been stripped off; embryological researches, however,

have shown that it really belongs to the retina. It consists of regular hexagonal cells, each of which has an unpigmented nucleus, while the protoplasm contains an abundance of pigment granules (Fig. 152). From this the entire layer acquires a dark-brown color.

All the pigment that is contained in such abundance in the interior of the eye belongs to two categories: 1. In the tissue of the uvea itself there are everywhere found branched cells (according to Münch, muscle cells) which contain pigment granules (Fig. 151). These are the chromatophores (pigment cells of the stroma), and the pigment contained in them is called *stroma pigment*, or, because it lies in the uvea itself, uveal pigment. 2. The inner surface of the uvea is everywhere coated with a layer of pigmented cells belonging to the retina and having the character of epithelial cells (*pigment epithelium*, Fig. 152). This pigment, which hence lies not in the uvea but to the inner side of it, is called the retinal pigment.

These two kinds of pigment are further distinguished by their minute structure. The pigment of the chromatophores consists of small amorphous masses; but the pigment granules in the cells of the pigment epithelium are short, rod-shaped structures, which should probably be regarded as small crystals, such as occur, very distinctly marked, in some of the lower vertebrates.

The pigmentiferous cells, including both of the chromatophores and the cells of the pigment epithelium, are similar in all eyes but the amount of pigment which they contain varies greatly. To this fact is due the inequality in the pigmentation of eyes; if the cells contain no pigment whatever the eye is albinotic.

313. The uvea in all of its parts is very rich in *nerves*. The ciliary nerves get to the uvea by piercing the sclera near its posterior pole. They form in the chorioid, and particularly in the ciliary muscle, a dense plexus, in which numerous ganglion cells are intercalated. This iris is also very rich in nerves, but contains no ganglion cells. The iris and the ciliary body contain, in addition to the motor nerves designed for the ciliary muscle and the muscular apparatus of the iris, a very great number of sensory nerve fibers which arise from the trigeminus; hence, inflammation of these parts is frequently attended with great pain. The chorioid, on the contrary, seems to possess no sensory nerves, since inflammation of this membrane runs its course without producing any sensations of pain.

(*d*) *Blood-vessels of the Uvea*

314. Three systems of blood-vessels exist in the eye—that of the conjunctiva, that of the retina, and that of the uvea (ciliary system of vessels). The *arteries* of the ciliary system are: 1. The posterior ciliary arteries. These arise from the ophthalmic artery, and enter the interior of the eye through the sclera in the region of the posterior pole. The majority of them pass at once into the chorioid (short posterior ciliary arteries, *c*, *c*, Fig. 153 and *Ab*, Fig. 154). Two of them, however (the long posterior ciliary arteries, *d*, Fig. 153, *Al*, Fig. 154), run, one on the outer side, the other on the inner side, between chorioid and sclera as far forward as the ciliary muscle. Here each divides into two branches, which run in a direction concentric with the margin of the cornea, and unite with the branches of the

artery of the opposite side to form an arterial circle, the circulus arteriosus iridis major (Fig. 153, *h*, Fig. 154, *Cima*, and Fig. 142, *a*). This gives off the arteries for the iris, which extend radially from its ciliary to its pupillary mar-

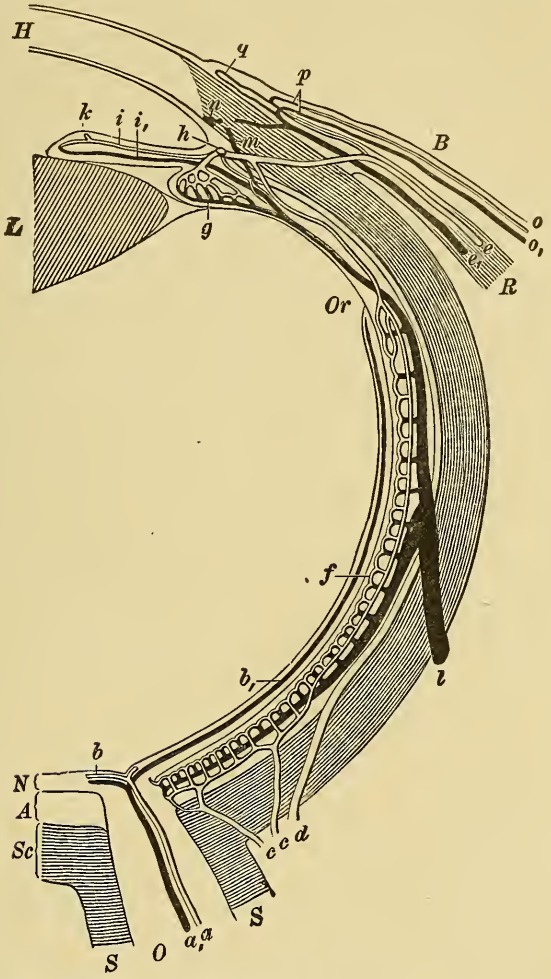


FIG. 153.—BLOOD-VESSELS OF THE EYE (SCHEMATIC). (After Leber.)

The *retinal system of vessels* is derived from the central artery, a, and the central vein, a_1, of the optic nerve, which give off the retinal arteries, b, and the retinal veins, b_1. These end at the ora serrata, *Or*.

The *system of ciliary vessels* is fed by the short posterior ciliary arteries, c, c, the long posterior ciliary arteries, d, and the anterior ciliary arteries, e. From these arise the vascular network of the chorioidal capillaries, f, and of the ciliary body, g, and the circulus arteriosus iridis major, h. From this last spring the arteries of the iris, i, which at the lesser [inner] circumference of the latter form the circulus arteriosus iridis minor, k. The veins of the iris, i_1, of the ciliary body, and of the chorioid are collected into the vasa vorticosa, l; some veins (m), however, that come from the ciliary muscle leave the eye as anterior ciliary veins, e_1. With the latter Schlemm's canal, n, forms anastomoses.

The *system of conjunctival vessels* consists of the posterior conjunctival vessels, o and o_1. These communicate with those branches of the anterior ciliary vessels which run to meet them; that is, with the anterior conjunctival vessels, p, and form with these the marginal loops of the cornea, q. *O*, optic nerve; *S*, its sheath; *Sc*, sclera; *A*, chorioid; *N*, retina; *L*, lens; *H*, cornea; *R*, internal rectus; *B*, conjunctiva.

gin (Fig. 153, *i*). Shortly before they reach the latter they form by anastomoses a second, smaller vascular circle, the circulus arteriosus iridis minor or the small circle of the iris (Fig. 153, *k*, Fig. 154, *Cimi*). 2. The anterior ciliary arteries come from in front, arising from the arteries of the four recti muscles (Fig. 153, *e*, Fig. 154, *Aa*). They perforate the sclera near the margin of the cornea and help to form the circulus arteriosus iridis major. The

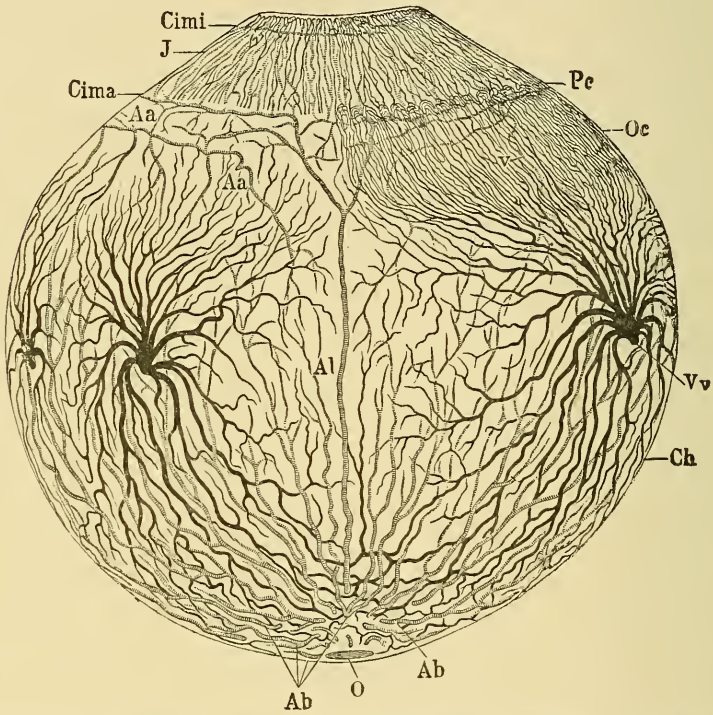


FIG. 154.—BLOOD-VESSELS OF THE UVEA (CILIARY SYSTEM). (SCHEMATIC.) (After Leber.)

In the region surrounding the optic nerve, *o*, the short posterior ciliary arteries, *Ab*, enter the chorioid, *Ch*, and end in the anterior segment of the latter. In the horizontal meridian of the eye the long posterior ciliary artery, *Al*, runs to the orbiculus ciliaris, *Oc*, and here divides dichotomously to form the circulus arteriosus iridis major, *Cima*. Branches of the anterior ciliary arteries, *Aa*, also take part in forming the circulus major, while other branches run backwards into the chorioid to meet the branches of the short posterior ciliary arteries. The circulus arteriosus iridis major gives off the radiating arteries of the iris, *J*, and these near the pupillary border of the iris form the circulus arteriosus iridis minor, *Cimi*. On the right side of the drawing the ciliary muscle is supposed to be detached, so that the abundant vascular network of the ciliary processes, *Pc*, comes into view. From the latter, numerous veins, *v*, run within the orbiculus ciliaris, at first parallel with each other, afterward converging, back to the venæ vorticosæ, *Vv*, to which also pass the veins from the posterior segment of the chorioid.

short posterior ciliary arteries are therefore designed mainly for the chorioid, the long posterior ciliary arteries and the anterior ciliary arteries for the ciliary body and the iris. Some recurrent branches of the latter, however, also supply a portion of the anterior segment of the chorioid.

315. The arrangement of the *veins* is essentially different from that of the arteries. In the chorioid the capillary network of the chorio-capillaris (Fig. 153, *f*) is fed by the arteries directly. The blood from this flows off

through a very great number of veins that keep uniting to form larger trunks. A number of these trunks simultaneously converge to a common center, where, consequently, a sort of whorl or vortex (Fig. 154, *Vv*) is produced by veins coming together from all sides. These vortices, the number of which amounts to four at least, usually more, lie somewhat behind the equator of the eye; from them are given off the venæ vorticosæ, which, perforating the sclera in a very oblique direction, carry off the blood to the outside (Fig. 153, *l*).

In the ciliary processes the arteries break up into a great number of twigs, which pass over into thin-walled veins (*g*, Fig. 153). These constitute the greater part of the ciliary processes, which, accordingly, consist mainly of vessels. The larger veins (Fig. 154, *Vv*) which are formed by the union of these vessels, and also most of the veins of the ciliary muscle, pass backward to the venæ vorticosæ. The veins that run back from the iris (i_1, Fig. 153) likewise pass to the venæ vorticosæ. Hence almost all the venous blood of the uvea empties into the latter. A portion of the veins coming from the ciliary muscle (*m*, Fig. 153), however, take another course, as they pass out directly through the sclera and thus come into view beneath the conjunctiva, near the margin of the cornea (anterior ciliary veins, Fig. 153,e_1). In their course these correspond to the anterior ciliary arteries, but have a much smaller area of ramification; it is they that principally constitute the violet vessels which we see running backward beneath the conjunctiva (see Fig. 68) in ciliary injection or in stasis within the eyeball (glaucoma). The anterior ciliary veins anastomose with the conjunctival veins and also with Schlemm's canal. The latter is a venous ring (sinus) running along the sclera-corneal junction (Fig. 147: Fig. 153,*n*).

The blood-vessels of the eye belong for the most part to the region of the uvea. It is this fact which determines the part played by the latter; for, while the firm corneo-sclera serves for the protection of the eye exteriorly and the retina for the perception of light, to the uvea is allotted the task of providing for the nourishment of the eyeball. Such is the abundance of blood-vessels which it contains that it really consists mainly of them; and by this fact its great tendency to become inflamed is accounted for.

The separate branches of the ciliary system of vessels anastomose repeatedly with each other—a circumstance which favors the compensation of circulatory disturbances. Thus, for instance, in glaucoma, in which the outflow of venous blood through the venæ vorticosæ is impeded, we see the anterior ciliary veins taking their place and carrying off larger quantities of blood than usual.

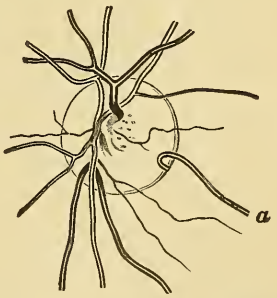


Fig. 155.—Cilio-retinal Artery.

From the outer and lower margin of the papilla rises a cilio-retinal artery (*a*), making a hook-like bend. In this case it is larger than usual because it is destined to replace the main infero-external branch (inferior temporal branch) of the central artery, which branch is wanting.

The ciliary vessels likewise supply the sclera with blood, giving off a few minute twigs to it as they pass through it. The number of blood-vessels in the sclera, how-

ever, is very small. Nevertheless, in the immediate neighborhood of the entrance of the optic nerve, from two to four branches of the short posterior ciliary arteries enter the sclera and form in it, by anastomoses, an arterial ring, *Zinn's scleral circle of vessels* (Fig. 24), surrounding the foramen for the optic nerve. This is of importance for the nutrition of the optic nerve, because numerous little branches go from it to the optic nerve and its sheaths, and anastomose with the branches of the central artery of the nerve. It is here, then, that the only connection between the ciliary and the retinal system of vessels exists.

It not infrequently happens that individual branches arising from the scleral circle of Zinn, instead of remaining in the optic nerve, make a bend and leave the nerve. They then enter the retina and run in it toward the macula lutea. These vessels, which are called *cilio-retinal*, ordinarily supply with blood a small district of the retina lying between the papilla and macula (Fig. 155).

By *optico-ciliary veins* we mean veins that branch off from the central vein of the optic nerve or from one of its ramifications, and pass over the head of the optic nerve into the chorioid (Elschnig).

II. The Pupil

316. The *iris* forms a diaphragm which, as in the case of many optical instruments, is interposed between the refracting portions of the eye. It has a double task to perform: it prevents an excessive amount of light from entering the eye and so dazzling it and injuring the retina, and it cuts off the marginal rays. These are the rays that pass through the periphery of the cornea and of the lens, and which, being less regularly refracted, would, unless arrested, impair the sharpness of the retinal image. In order to be perfectly impermeable to light, the iris has a pigment layer on its posterior surface. The iris has the advantage over the artificial diaphragms of optical instruments in that its size changes spontaneously to suit the circumstances of the case. For this purpose there exist contracting fibers (sphincter pupillæ) and dilating fibers (dilatator pupillæ).

The *contraction* of the pupil is governed by the oculo-motor nerve, which supplies the sphincter pupillæ (and also the ciliary muscle) through the ciliary ganglion and the ciliary nerves. By stimulation of the oculo-motor nerve, contraction of the pupil is produced; by its section or paralysis, dilatation of the pupil.

Dilatation of the pupil is dependent upon the sympathetic, which derives the fibers destined for the pupil from the cilio-spinal center of the cervical spinal cord. Irritation of this center or of the cervical sympathetic produces dilatation, and paralysis of it contraction of the pupil.

Lastly, the vessels of the iris are of moment in determining the width of the pupil, since by their distention the iris is widened and the pupil consequently contracted, and vice versa.

The width of the pupil may be affected by purely *mechanical* factors. This is the case, for instance, with the contraction of the pupil that regularly occurs when the aqueous escapes. This contraction is of practical importance in the performance of many operations. When, for instance, we perform discission of cataract through the

cornea, we take care not to let the aqueous escape, since the consequent contraction of the pupil would expose the iris to greater pressure from the swelling lens. That this contraction owes its origin to purely mechanical causes, is deduced from the fact that it occurs even in the eye of a dead man when the aqueous is let out.

Dilatation of the pupil manifests itself by a sense of dazzling. Sometimes patients also allege that objects appear smaller (micropsia). This phenomenon, however, does not depend on the dilatation of the pupil, but on the paralysis of accommodation, which is generally present at the same time, and is therefore also observed when the latter alone is present. (For its explanation, see Paralysis of Accommodation, § 797.)

In *contraction of the pupil*, sometimes—i.e., if spasm of accommodation is at the same time present—objects appear larger (macropsia). Moreover, obscuration of vision is often complained of, because less light enters the eye through the contracted pupil. In very marked miosis, whether occurring after the employment of miotics or appearing spontaneously (e.g., as the result of tabes), the pupil is frequently found to be irregular and slightly angular, although no synechiæ are present.

317. Reflex Reaction. Light Reaction.—The *reaction* of the pupil takes place involuntarily and unconsciously. It is either reflex, in which case the stimulus is transmitted from centripetal nerve channels to the nerves of the iris, or it is associated, in which case the pupillary fibers of the oculo-motor nerve are set into action simultaneously with other fibers of the same nerve.

The *reflex action* of the pupil is set in action—

(1) By *light*. This produces *contraction* of the pupil, while conversely, as the illumination diminishes, the pupil dilates. The reflex arc in this case passes through the optic nerve to the anterior corpus quadrigeminum, thence to the nucleus of the oculo-motor nerve, and then along this nerve to the eye. The reaction for light always affects both eyes—i. e., if the light falls into one eye alone, the pupil of the other eye also always contracts (consensual reaction). The reaction takes place in both eyes in exactly the same way—that is, appears at the same time and reaches the same pitch. The reaction of the pupil to light is exceedingly sensitive, and is employed with great advantage to determine objectively whether an eye has any sensation of light or not (particularly in children, malingerers, etc.). [For the method of determining the light reaction, see page 81.]

If in a darkened room we place a source of light to one side of the eye and make the person upon whom we are experimenting look straight ahead into the darkness, his pupil is dilated. If then we direct the person to concentrate his attention on the light without looking at it, that is while maintaining a straightforward gaze, the pupil contracts (*cortical reflex* of Haab).

The *reaction of the pupil to light* is a very valuable sign of the presence of perception of light: in the first place, because it is exceedingly sensitive; and, secondly, because it demonstrates the existence of perception of light independently of the statements of the patient. Its usefulness is still further enhanced by its disclosing in the pupils of both eyes (through the consensual reaction) the perception of light by one eye. How is this *consensual reaction* effected? From the retina of each eye the pupillary fibers, like the fibers subserving vision, pass through the optic nerve into the chiasm. There, like the visual fibers, they undergo semidecussation. Hence, from the retina of the

right eye (*R*, Fig. 156), fibers pass through the chiasm partly into the right, partly into the left, optic tract (Fig. 156, *T* and T_1). From these the stimulus is transmitted directly

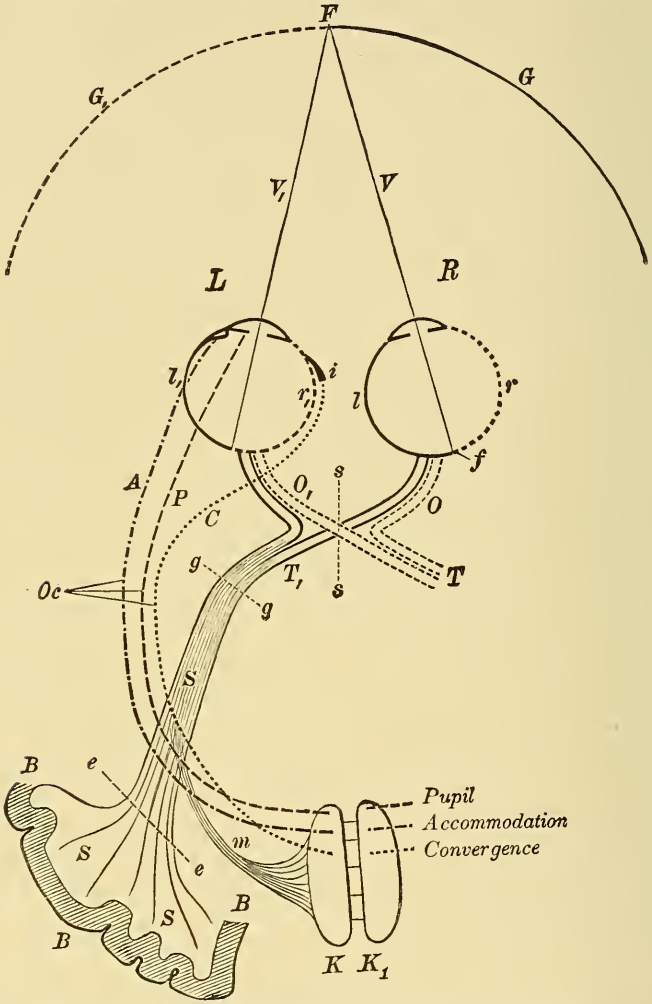


Fig. 156.—Schematic Representation of the Optic Pathway.

The field of vision common to the two eyes is composed of a right half, *G*, and a left half, G_1. The former corresponds to the left halves, *l* and l_1, of the two retinæ, the latter to the right halves, *r* and r_1. The dividing line between the two halves of the retina is formed by the vertical meridian. This passes through the fovea centralis, *f*, in which the visual line (*V*) drawn from the point of fixation, *F*, impinges upon the retina. The optic-nerve fibers arising from the right halves, *r* and r_1, of the two retinæ (indicated by the dotted line) all pass into the right optic tract, *T*, while the fibers belonging to the left halves, *l* and l_1, of the two retinæ pass into tract T_1. The fibers of each optic tract for the most part pass to the cortex of the occipital lobe, *B*, forming Gratiolet's optic radiation, *S;* the smaller portion of them, *m*, goes to the oculo-motor nucleus, *K*. This consists of a series of partial nuclei, the most anterior of which sends fibers, *P*, to the pupil (sphincter iridis); the next one sends fibers, *A*, to the muscle of accommodation; and the third sends fibers, *C*, to the converging muscle (internal rectus, *i*). All three bundles of fibers run to the eye in the trunk of the oculo-motor nerve, *Oc*. Division of the optic tract at *g g* or at *e e* produces right hemiopia; and in the former case there would be no reaction to light on illuminating the left half of either retina. Division of the chiasm at *s s* produces temporal hemiopia. Division of the fibers, *m*, abolishes the reaction of the pupil to light, but leaves the sight and also the associated contraction of the pupil in accommodation and convergence unaffected.

to both right and left oculo-motor nuclei (K and K_1); then each nucleus sets up a contraction of the pupil on its own side. The consensual reaction, therefore, is really as direct as is the pupillary reaction of the illuminated eye itself.

The result of the consensual reaction is that under normal conditions both pupils must be of equal diameter, even if only one of the eyes is exposed to the impact of light, or if the sensitiveness of the two eyes to light is different. Inequality of the pupil (*anisocoria*) is always pathological. [Yet according to a number of observers anisocoria may occur in those who are perfectly healthy or who at most suffer from a functional neurosis. In such cases it may be transient. In this non-pathological anisocoria, which is probably quite rare, the pupils are round, and the pupillary reactions are perfectly normal—a feature which distinguishes it from most cases of pathological anisocoria (Uhthoff).—D.] For the reasons given anisocoria can never take its origin from the centripetal fibers (optic-nerve fibers), but is always caused by a disturbance in the centrifugal channels (oculo-motor nerve and its center or its connections with the sympathetic) [or by purely mechanical causes, such as adhesions or rupture of the iris—D.].

(As a matter of fact when only one eye is illuminated, the pupil of this eye is somewhat narrower than that of the eye which is not illuminated, perhaps because in addition to the reflex contraction on the illuminated side there is also a direct contraction due to the illumination of the iris tissue itself. In lower animals the pupil even in an excised iris contracts distinctly to light, and the same thing is true, though to a very slight extent, of the human iris also. However, the difference between the two pupils when unequally illuminated is so slight in man that it can be demonstrated only by precise methods of measurement, and in practice we may hold fast to the statement above made that any inequality of the pupils is to be regarded as pathological.)

Contraction of the pupil to light is greatest when the light falls on the fovea centralis; stimulation of peripheral portions of the retina by light is followed by but slight contraction of the pupil. The width of the pupil is not precisely proportional to the quantity of light that enters the eye, but is the same with light of the most varying degrees of intensity, so long as the intensity of the light remains uniform. When this intensity changes the pupil alters its diameter accordingly, but if the light is maintained at this new intensity the pupil gradually returns to its former diameter when the retina has become completely adapted to the new intensity.

Even in cases in which the sight is very much reduced because a portion of the fibers conveying visual impressions have been destroyed by inflammation or compression of the optic nerve, the mean diameter of the pupil is often normal. We conclude from this that the fibers for the pupil are more resistant than the fibers conveying visual impressions (Schirmer). [See also page 386.]

318. Relation of Reaction to Light Perception.—In testing the perception of light by the reaction of the pupils, we must not lose sight of the fact that there are cases in which, although the perception of light is present, the reaction is absent; and, conversely, cases in which, with good reaction, there is yet no perception of light.

(*a*) The cases in which the *pupil does not react to light, although the perception of light is present*, are frequent. They may have the following causes: (1) Changes in the iris itself, such as inflammatory swelling, adhesion, increase of tension, laceration in the sphincter, atrophy of the tissue, etc. (2) Disturbances in the motor portion of the reflex path, i.e., in the oculo-motor nerve. Under this head belong paralysis of the oculo-motor terminals in the iris itself produced by mydriatics, and paralysis of the trunk of the oculo-motor nerve or of its nucleus of origin. In all the cases above

mentioned the synergic reaction with convergence and accommodation is impaired or quite abolished along with the light reaction (*absolute* [or *total*] *iridoplegia*). (3) Disturbance in that portion of the reflex path which lies between its centripetal limb (optic nerve) and its centrifugal limb (oculo-motor nerve) (Fig. 156, *m*). We then find that the reaction for accommodation and convergence is retained, while the reaction for light has disappeared (*Argyll-Robertson pupil* or *reflex iridoplegia*). This is very frequent in tabes and progressive paresis and is so rare in other nervous diseases that it furnishes an important diagnostic evidence that one of these two diseases is present or impending. In these cases it is often associated with a marked contraction of the pupil (so-called spinal miosis), but is also found at times with a normally wide pupil. In case the diagnosis is doubtful the state of the pupil is often of importance—absolute iridoplegia with dilated pupil being rather an evidence of cerebral lues, and reflex iridoplegia with contracted pupil an evidence of tabes or general paresis.

(*b*) It also happens that the *reaction of the pupil to light is present, without there being any perception of light.* This occurs when the lesion is situated high up in the optic pathway. The fibers of the optic nerve ascend to the cerebral hemispheres and terminate in the cortex of the occipital lobe (*B*, Fig. 156). But, some time before this takes place, those fibers (*m*) of the reflex arc which pass to the center for the pupillary movements branch off from the optic tract. If, then, the optic pathway is interrupted above the place where they are given off (e.g., at *e e*), stimulation of the optic nerve fibers no longer reaches the cerebral cortex and hence excites no perception, and yet the pupillary reflex is still regularly produced. The same thing would happen if the cerebral cortex itself were through some lesion incapacitated for performing its functions. In these cases, however, the lesion of the optic pathway would have to be bilateral, as otherwise hemiopia and not blindness would be present (see § 545). For this reason it is clear why such cases, in which blindness exists, even though the examination of the eye gives negative results and the reaction of the pupil to light is preserved, are very rare (occurring, for example, in uræmic amaurosis, see § 522), so that under these circumstances our first thought would be of simulation or of hysteria, and we would examine for these conditions first. [Cases, however, do occur, although very rarely indeed, in which the reaction of the pupil to light is retained although the eye is completely blind, and this may occur even when the blindness is evidently due to disease of the optic nerve itself (Wilbrand and Saenger). This can be explained only on the hypothesis above stated (page 385), that the fibers for the conduction of the light reflex are more resistant than those conveying visual impressions. I have seen one such case. So, too, as Leber points out, in certain forms of complete blindness due to retinal degeneration a very slow reaction to light occurs, the pupil dilating very gradually in the dark and contracting again very gradually on return to light. Such a reaction, again, is no proof of the presence of light perception.—D.]

319. Sensory Reaction.—(2) Toward *sensory stimuli*, no matter what part of the body they affect, the pupil reacts by *dilating*. Hence, in deep sleep, and also in profound narcosis, in which sensory stimuli no longer produce reflexes, the pupil is very much contracted, dilating, however, the moment that waking from the sleep or from narcosis occurs. Strong psychic stimuli—e. g., fright—in like fashion produce dilatation of the pupil.

When sensory stimuli act intensely upon the eye itself the pupil contracts instead of dilating. The contraction here is due to the hyperæmia of the iris produced by the stimulus (see page 396). [This is probably the explanation of the miosis that sometimes results from the application of dionin.—D.]

320. Associated Reaction.—The associated reaction of the pupil always consists of a *contraction*. It occurs in conjunction with *convergence*, i. e., in synergy with the contraction of the internal recti. Since under physiological conditions every act of *accommodation* is accompanied by a corresponding convergence, and the contraction of the pupil keeps pace with it, too, we have here as a regular thing a uniform consentaneous action of the sphincter pupillæ, the ciliary muscle, and the internal rectus. These muscles are all supplied by the oculo-motor nerve, so that their associated action depends upon a simultaneous excitation of the portion of this nerve that supplies them. [For the method of determining the associated reaction, see page 81.]

If a man is told to close the eye tight, but at the same time by holding the lid apart prevents it from closing, we see the pupil contract. This phenomenon comes out most plainly with pupils which otherwise are rigid—*lid reaction* of the pupil (Von Graefe).

321. Since the pupil reacts to stimuli of so many kinds and varying so greatly in degree, it is in a state of constant motion. But in every case the pupils of the two eyes are equally large. Inequality of the pupils is always pathological. [See, however, page 385.] The mean width of the pupil differs with the individual and also alters with the age. Very greatly contracted in new-born infants, the pupil soon becomes more dilated, and then becomes smaller again in manhood, and still more in old age. In old people, also, the reaction of the pupil becomes sluggish, in consequence of the unyielding character of the tissue of the iris, and especially of the sphincter (rigidity of the sphincter). [For the method of determining the width of the pupil, see page 80.]

322. Reaction of the Pupil and Ciliary Body to Poisons.—There are poisons which act to stimulate the terminals of the autonomous nervous system in general, i. e., in the eye stimulate the terminals of the oculomotor nerve in the iris and ciliary body, and there are other poisons which act in an antagonistic way upon the same nerves, i. e., paralyze them. The poisons of the muscarine group (including besides muscarine, eserine and pilocarpine) stimulate, and atropine and its congeners paralyze the interior muscles of the eye. The first named, therefore, cause contraction of the pupil or miosis[6] and are hence called miotics, the second cause dilatation of the pupil or mydriasis and are called mydriatics. In the iris, however, there is also a dilatator which is supplied by the sympathetic and by whose contraction mydriasis is likewise produced. Hence to the mydriatics in the wider sense of the term belong also those poisons which stimulate the terminals of the sympathetic nerve, namely, cocaine and adrenaline. The physiological action of these poisons upon the eye will be considered here; their therapeutic application will be found on pages 60 et seq.

[6] From μείωσις, contraction; hence miosis, and not myosis, as it is generally written (Hirschberg). The derivation of μυδρίασις is uncertain. This word was already used by the ancients to signify dilatation of the pupil, and also the blindness that is so frequently associated with it.

323. (1) *Atropine* paralyzes the nerve terminals in the sphincter and the ciliary muscle, and hence is followed by dilatation [and immobility] of the pupil and also by an inability to accommodate. The dilatation of the pupil is a very considerable one. If, in the case of a dilatation of the pupil caused by oculo-motor paralysis, atropine is instilled, the pupil becomes still more dilated, because the atropine abolishes the tone of the muscular fibers of the sphincter. The effect of the atropine makes its appearance in from ten to fifteen minutes after the instillation, and soon reaches its maximum. Commencing with the third day it begins to decrease again, but does not disappear completely until after the lapse of a week [or longer]. Quite as long lasts the disturbance of sight which is caused by the dilatation of the pupil, but mainly by the paralysis of the accommodation, and hence makes itself apparent most of all in near vision (causing inability to read).

[In infants atropine, even in poisonous doses, often exerts very little effect on the pupil. In others the width of the atropinized pupil averages 7.5 mm., but varies from 6 to 9 mm., the least dilatation being found in those over 40 years of age.—D.]

Atropine is such an efficient mydriatic that the millionth part of a gramme suffices to dilate the pupil. If we instil atropine into another person's eye, and in so doing wet our finger, and then through carelessness touch our own eye with it, this is enough to produce dilatation of our own pupil.

[The instillation of atropine and still more its application in substance (see page 60) sometimes cause symptoms of *poisoning*.] These consist in a troublesome feeling of dryness in the throat, nausea, reddening of the face, and an acceleration of the pulse; also excitement and tremor, or, in case of severe poisoning, loss of consciousness. In marked poisoning the pupil of the other eye that has not been treated with atropine is dilated, too. General poisoning may also, of course, take place through the internal use of atropine or remedies containing it (belladonna). Patients who take such preparations internally—these being commonly patients with lung disease, who have got them in order to relieve cough or night-sweats—often complain that they are dazzled and do not see well for work at near points. We then find moderate dilatation of the pupil and reduction of the accommodation.

Some persons show an *intolerance of atropine*. This makes itself apparent in various ways: (*a*) By the appearance of symptoms of poisoning, like dryness of the throat and nausea, even with small doses. This occurs particularly after long-continued use of atropine. (*b*) By exciting a catarrh (atropine catarrh) which is generally marked by the formation of numerous follicles. For this also a prolonged use of atropine is usually requisite. (*c*) In some persons a single drop of atropine produces great redness and swelling of the lids, like an attack of erysipelas.

324. Other mydriatics are hyoscyamine and [the isomeric] scopolamine, homatropine, euphthalmine, and eumydrine.

Scopolamine is more active than atropine, a $\frac{1}{2}$- or $\frac{1}{4}$-per-cent solution of the former producing as much effect as a 1-per-cent solution of the latter. It is also more poisonous. [In $\frac{1}{5}$-per-cent solution it produces satisfactory relaxation of the accommodation for refraction work, its effect on the pupil and vision then lasting four days (Marlow).—D.]

[*Homatropine* in 1-per-cent solution causes a dilatation of the pupil lasting several hours and a more or less incomplete paralysis of the ciliary muscle. As employed in

refraction work, i.e., in 2-per-cent solution repeatedly instilled, it produces, according to my observations, an effect on the pupil and ciliary muscle equal to that of atropine, but lasting about one-third as long. The dilatation of the pupil usually just equals that produced by atropine in the same subject. The pupil is 5 to 9 (usually 7 to 8) mm. wide, and is generally widest in persons of 15 to 25 and narrowest in those over 45. The mydriasis begins in 10 to 15 minutes, and reaches its maximum in 30 to 50 minutes. The light reaction disappears usually in 25 to 30 minutes, sometimes not till much later, and may not disappear at all. The mydriasis generally lasts at least 48 and often 72 hours. Indeed, accurate measurements of the accommodation show that the latter may be affected even four or five days after the instillation. On a pupil thus dilated pilocarpine has little if any effect, and although eserine can contract the pupil and produce spasm of accommodation, this effect soon passes away and the cycloplegia and mydriasis return. The effect of homatropine on the pupil bears no constant relation to its effect on the ciliary muscle. A good deal of accommodation may be left when the pupil is wide and the light reaction abolished; and the accommodation may be almost gone when the pupil is quite small.—D.]

[*Euphthalmine* in 2-per-cent solution produces a comparatively transient dilatation of the pupil, which can be abrogated by 1-per-cent pilocarpine. A 5-per-cent solution produces a much more lasting effect, which pilocarpine may not suffice to counteract.—D.]

325. (2) *Eserine* (also called *physostigmine*) has an action exactly the opposite of that of atropine, since it places the sphincter iridis and the ciliary muscle in a state of tonic contraction. Consequently, miosis develops so that the pupil is about the size of a pin's head, with adjustment of the eye for the near point, as if marked myopia were present. Eserine diminishes the intra-ocular pressure somewhat. [See page 19.] *Pilocarpine* acts in a similar way but less energetically.

The action of miotics is of shorter duration than that of the mydriatics, and is also less powerful. Hence a pupil contracted by eserine or pilocarpine can be dilated by atropine, but a pupil dilated by atropine cannot be contracted by a miotic.

Eserine frequently excites severe pain in the eye and head, painful contractions in the lids, and even nausea and vomiting. These are not symptoms of poisoning, but are the results of the great narrowing of the pupil and contraction of the ciliary muscle, by which the ciliary nerves are pulled upon. This bad by-effect, therefore, usually is absent when, for any cause, the eserine fails to produce any marked contraction of the pupil.

A solution of eserine after being a few days in the light turns red (for which reason it ought to be prescribed in dark or paraffined bottles), without, however, suffering any impairment of its activity.

326. (3) *Cocaine* dilates the pupil—not, however, as in the case of mydriatics proper, by paralyzing the sphincter pupillæ, but by contracting the dilatator. The dilatation of the pupil is therefore only a moderate one [but considerable if a strong solution or repeated instillations are used], and the reaction of the pupil to light persists; moreover, mydriatics and miotics still produce an effect. If cocaine is instilled into an eye the pupil of which has been dilated by atropine, the dilatation increases somewhat; hence the mydriasis produced by the simultaneous action of atropine

and cocaine is the most considerable that can possibly be attained. The accommodation is not paralyzed by cocaine, but only somewhat weakened.

Besides action upon the iris, cocaine produces also the following effects: The conjunctiva becomes very pale, and at the same time the patient has a feeling of cold [and dryness] in the eye. The palpebral fissure is more widely open and the act of winking is less frequent. Sometimes the eye is protruded somewhat forward and the intra-ocular tension slightly diminished (see page 19). The practically important phenomenon, however, is the anæsthesia shown by the superficial tissues of the eye (cornea and conjunctiva).

The effects of cocaine are best explained upon the assumption that it acts as a stimulant to the fibers of the sympathetic. The contraction of the vessels thus produced causes the pallor of the conjunctiva. The sympathetic also innervates the musculus tarsalis superior and inferior (see § 581) and the dilatator pupillæ, and the contraction of these muscles causes the dilatation of the palpebral fissure and of the pupil. The anæsthesia of the surface of the eyeball has nothing to do with the sympathetic, but depends on the paralysis of the sensory nerves.

For the other alkaloids belonging to the cocaine group see page 63.

327. (4) *Adrenaline.* The extract of the suprarenal capsules stimulates the muscle fibers supplied by the sympathetic. Instilled into the eye it causes [great and immediate] contraction of the blood-vessels, while the dilatation of the pupil due to contraction of the dilatator pupillæ occurs in special cases only.

III. Development of the Eye

328. Primitive Ocular Vesicle. Lens.—The eye develops from a pouch which forms on each side of the first cerebral vesicle. The pouch, which is called the primitive ocular vesicle (Fig. 157, *a*), remains in connection with the cerebral vesicle by means of a pedicle, which, at first broad, afterward more narrow, becomes subsequently the optic nerve (*b*). Its surface is covered by the ectoderm (*E E*). Upon this ectoderm, at a point corresponding to the apex of the ocular vesicle, there soon forms a thickening. This is the first rudiment of the lens, and the way in which it is formed is that the ectoderm here grows thicker, becomes folded upon itself, and forms an everted pouch directed toward the ocular vesicle (*L*, Fig. 158). This pouch deepens, and finally becomes shut in in front so as to form a closed sac, the *lens vesicle* (*L*, Fig. 159). The lens is accordingly an epithelial structure, being a derivative of the external germinal layer, and in the beginning consists of a hollow vesicle, which afterward becomes filled up by the growth of its cells and is converted into a solid sphere.

329. Secondary Ocular Vesicle. Retina.—In proportion as the ectoderm at the site of the primitive lens pushes against the ocular vesicle, the surface of the latter becomes more and more indented. Thus a flask-shaped structure with double walls is formed out of what was once a round sac (Fig. 159). This is called the *secondary ocular vesicle*, which is hence the

primitive ocular vesicle that has been invaginated and thus, so to speak, reduplicated. From the ocular vesicle is subsequently formed the retina, which thus must be looked upon as an isolated portion of the brain itself. The exterior and interior layers of the secondary ocular vesicle become differentiated early. In Fig. 161, the interior layer, *r*, is seen to be already considerably thicker than the exterior, *p*, although the latter also consists of several rows of cells. The exterior layer later becomes composed of a single row of cells, takes up pigment (Fig. 162), and ultimately becomes the pigment epithelium, which therefore is rightly counted in with the retina. The interior layer (*r*) soon gets to surpass the exterior one considerably in thickness, especially at the posterior portion of the eye, where its cells acquire a radial arrangement and develop into the retina proper. The anterior margin of the ocular vesicle, where the two layers become continuous, corresponds to the margin of the pupil in the fully developed eye (Fig. 163).

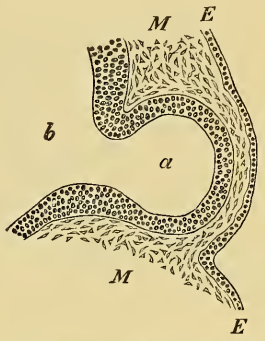


FIG. 157.—SECTION THROUGH THE EMBRYONIC EYE AT A PERIOD OF DEVELOPMENT CORRESPONDING TO THAT OF A HUMAN EMBRYO OF TWENTY-ONE DAYS. Magnified 100 × 1.

The primitive ocular vesicle, *a*, is a protrusion from the first cerebral vesicle, *b*, from which it is separated by a shallow constriction. It is surrounded by the cells of the mesoderm, *M*, over which passes the ectoderm, *E*, consisting for the most part of a single layer of cells.

330. Fetal Cleft. Vitreous.—At the time when the indenting of the ocular vesicle by the lens takes place, the latter completely fills the cavity of the vesicle, no vitreous cavity as yet existing. The formation of the latter cavity is effected by the pushing in of vessels between the ocular cup and the lens and by the development of the tissue of the vitreous. The vessels are derived from the mesoderm, which surrounds the ocular vesicle (Fig. 157, *M*), and which makes its way into the interior of the latter through an opening—the *fetal ocular cleft*—in its lower side. Even as early as the time when the ocular vesicle is undergoing invagination so as to form a flask, we notice that at one spot in its lower side the wall of the flask is altogether deficient (Figs. 158 and 161). Here, then, a fissure-like defect exists in the wall of the flask, a defect which is continued backward upon the pedicle of the ocular vesicle (the optic nerve) in the form of a furrow (Fig. 160). Through this fissure vessels together with some mesodermal tissue gradually grow from the outside into the interior of the eye, pushing a way in between the retina and the lens.

The development of the tissue of the vitreous takes place from the cells of the inner layer of the ocular vesicle, i. e., from the part that afterward becomes the retina, and mainly from its most anterior, or ciliary, portion. In proportion as the vitreous develops, the lens pushes away from the bottom of the ocular cup and against its anterior wall.

The cleft in the optic pedicle, which represents the continuation of the ocular cleft backwards (Fig. 160), also becomes united later on by the growing together of its walls, and thus shuts off the vessels which lie at its bottom. These are converted into the central vessels of the optic nerve, since the fibers of the optic nerve develop along the course of the optic pedicle.

331. Anterior Chamber.—In the early stages of development not only is there no vitreous, but the anterior chamber is also wanting. The lens which has budded out from the ectodermal lining lies in direct contact with

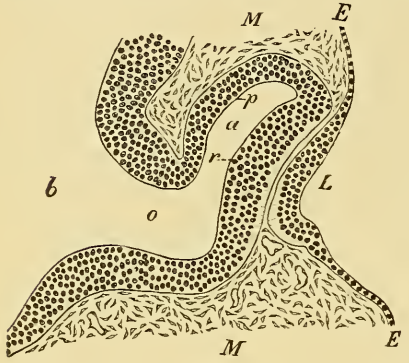


Fig. 158.

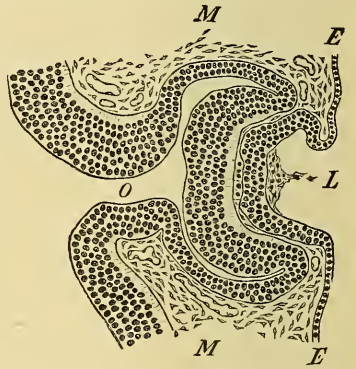


Fig. 159.

Fig. 158.—Section through the Embryonic Eye at a Period of Development Corresponding to that of a Human Embryo of Twenty-two to Twenty-three Days. Magnified 100 × 1. The vertically made section passes through the fetal ocular fissure. The ectoderm, *E*, dips in somewhat at the site of the rudimentary lens, *L*, and besides is thickened, being made up here of several layers of cells. Corresponding to this in-dipping, the ocular vesicle, *a*, presents an indentation in its cavity, and is consequently converted into the ocular cup, the inner wall, *r*, of which subsequently becomes the retina, the outer wall, *p*, becoming the pigment epithelium. The interior of the ocular cup communicates with the first cerebral vesicle, *b*, through the rudimentary optic nerve, *o*. The projecting portion of the wall of the ocular cup is wanting below, because the fetal ocular cleft is situated here. *M*, mesoderm, in which, near the lower edge of the rudimentary lens, can be seen the cross section of a capillary vessel.

Fig. 159.—Section through the Embryonic Eye at a Period of Development Corresponding to that of a Human Embryo of Twenty-four to Twenty-five Days. Magnified 100 × 1. The section here depicted does not pass through the fetal ocular cleft, so that the secondary ocular vesicle appears as a complete cup; the more so since the portion of ectoderm, *E*, representing the rudimentary lens, *L*, has become invaginated further than in Fig. 158. In the bottom of the depression occupied by the lens there lies some cell detritus, and between the lens and the inner wall of the ocular cup are seen a few cells derived from the mesoderm, *M*. In one or two spots in the mesoderm are visible the cross sections of capillaries. *O*, rudimentary optic nerve.

the latter. Then the mesoderm grows in from all sides at the anterior margin of the ocular cup between ectoderm and lens. In this mass of mesodermal tissue there afterwards develops a slit, which is the anterior chamber. The portion of the mesoderm lying in front of the slit forms the cornea, its posterior portion forms the iris and pupillary membrane.

332. Embryonic Vessels.—The arrangement of vessels in the embryo is essentially different from that in the adult eye. Most of the vessels of the embryonic eye are given off from the central artery of the optic nerve. This continues its course, as the arteria centralis corporis vitrei or arteria hyaloidea (Fig. 162), through the vitreous to the posterior pole of the lens, lying in the central canal of the vitreous (canalis hyaloideus seu Cloqueti). Furthermore, as it enters the eye, the central artery of the optic nerve gives off

lateral branches which form an arterial network in the peripheral portions of the vitreous (vasa hyaloidea propria—not yet present in the eye that is represented in Fig. 162), and likewise extend forward to the margin of the lens. The main trunk of the arteria centralis corporis vitrei, upon arriving at the posterior pole of the lens, divides into branches, ramifying over the posterior surface of the lens and running forward to the margin of the latter,

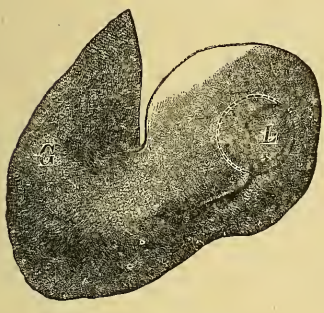


Fig. 160.

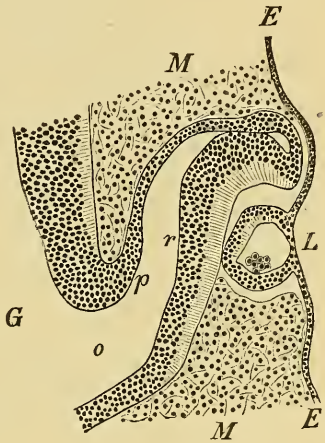


Fig. 161.

Fig. 160.—Secondary Ocular Vesicle with Ocular Cleft Seen from in Front and a little Below. The drawing is taken from a model which Prof. Hochstetter has made from his dissections by the plate-method. The model represents the rudimentary brain with its processes, but omitting the ectoderm and mesoderm. The eye rises by a thick hollow pedicle from the lateral wall of the first cerebral vesicle, *G*. At its distal end it presents an indentation, *L*, which represents the invagination of the lens vesicle into the ocular vesicle. The limits of the lens vesicle are indicated by a dotted curved line. From the lower margin of the indentation runs the fetal ocular cleft. This, at first very narrow, afterward widens somewhat, and extends down upon the pedicle of the optic vesicle.

Fig. 161.—Secondary Ocular Vesicle with Ocular Cleft. Left Eye of a Human Fetus Twenty-seven Days Old, Seen in Vertical Section. Magnified 88 × 1. Out of the sections combined to form Fig. 160, the one selected for representation is that passing precisely through the ocular cleft; hence the lower wall of the ocular fissure is wanting here, just as it is in Fig. 158. The walls of the first cerebral vesicle, *G*, approach each other and form the pedicle of the ocular vesicle, *o*, (subsequently the optic nerve), and further along form the reduplicated wall of the secondary ocular vesicle itself. The external lamina, *p*, of this reduplicated wall, which later is transformed into the pigment epithelium, composed of a single layer of cells, is at this time still destitute of pigment and composed of several layers of cells. At the anterior border of the ocular vesicle it is reflected to form the thick inner lamina, *r*. This latter, from which the retina proper is developed, is already beginning to show a radial arrangement of nuclei. The anterior border of the ocular vesicle is covered by the ectoderm, *E*, *E*, upon which is the rudimentary lens, *L*, which has already become closed so as to form a vesicle, but is not yet completely detached. Above, the mesoderm, *M*, fills the space between the cerebral vesicle, the ocular vesicle, and ectoderm; but below, the mesoderm, wherever the ocular cleft extends, penetrates into the interior of the ocular cup till it reaches the lens vesicle.

where the anterior extremities of the vasa hyaloidea propria unite with them and form a specially dense network of vessels surrounding the border of the lens. In front of the equator of the lens branches run to this vascular network, which come round the anterior border of the ocular cup from that portion of the mesoderm which afterward forms the iris. They assist in covering the anterior capsule of the lens also with a vascular network. Among the vessels derived from the iris are found veins as well as arteries, and these veins provide for the escape of all the blood, since all the other vessels going to the lens are arteries. The lens in the fetal eye is accordingly surrounded

by a vascular membrane, the tunica vasculosa lentis, which in the region occupied by the pupil bears the name of pupillary membrane (membrana pupillaris, *P*, Fig. 163), while its remaining portion is known as the membrana capsularis (*C*, Fig. 163). The tunica vasculosa lentis disappears in

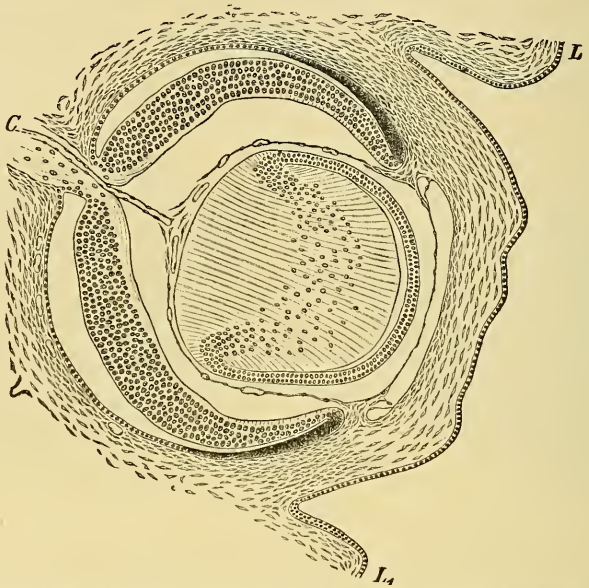


FIG. 162.—SECTION THROUGH AN EYE AT A PERIOD OF DEVELOPMENT CORRESPONDING TO THAT OF A HUMAN EMBRYO IN THE MIDDLE OF THE THIRD MONTH. Magnified 73 × 1.

The envelope of the ocular vesicle is formed of mesoderm, and in its anterior segment consists of the cornea, which contains an abundance of nuclei throughout and is separated by a particularly marked accumulation of nuclei from the posterior segment. In this posterior segment no delimitation between sclera and uvea has as yet taken place. The uvea will develop from the inner layers which are distinguished by containing more nuclei—a characteristic which is continued over into the hindmost, or uveal, layers of the cornea. At a point corresponding to the anterior margin of the ocular vesicle the mesoderm projects into the interior of the eye, and from the free border of the ring-shaped process thus formed (constituting what is later the iris) rise two delicate vascular membranes which surround the lens, constituting a sort of vascular lenticular capsule. Into the hinder of these two membranes the hyaloid artery enters at a point corresponding to the posterior pole of the lens. This artery rises from the central artery, *C*, of the optic nerve. Of the two laminæ composing the secondary ocular vesicle, the external, or pigment epithelium, has been reduced in its posterior part to a single layer of cells, while in its anterior portion there are still several cell layers present which have already taken up pigment. The inner lamina, or retina, consists of numerous layers of cells, the nuclei of which show a partially radial arrangement. In the immediate vicinity of the optic-nerve entrance can be seen the way in which one lamina is reflected into the other. The anterior point of reflection corresponds to what is later the pupillary margin of the iris. The lens is of an almost spherical shape; its antero-posterior diameter, in fact, is actually somewhat larger than its equatorial. Upon the anterior surface of the lens lies the epithelium, which still consists of several layers of cells; and no distinct lens capsule has yet been differentiated. In the region of what is later the equator of the lens the epithelial cells are growing out into lens fibers, which are still nucleated throughout, and take a sagittal direction. The posterior surface of the lens is destitute of epithelium, and is covered by an extremely delicate capsule. The vitreous cavity is very small. *L*, L_1, the eyelids growing out.

the last two months before birth, although scattered remnants of the pupillary membrane are quite frequently found still present in newborn infants.

The vessels of the retina develop by growing out from vessels that branch off from the central artery at the optic-nerve entrance and push on into the fiber-layer of the retina, while the vessels of the vitreous, previously present, undergo obliteration.

333. Cornea, Sclera, Iris.—The mesoderm that envelops the ocular vesicle forms through its outer layers the cornea and sclera, and through its inner layers the uvea. The most anterior portions of the latter—i. e., the ciliary body and iris—arise from that layer of the mesoderm which juts out like a spur into the interior of the eye and helps to form the vascular capsule of the lens (Fig. 162). This spur-like process keeps pushing on into the interior of the eye, carrying with it on its posterior surface the anterior wall of the ocular vesicle (Fig. 163). That portion of the mesoderm which directly follows the free, projecting part constituting the rudimentary iris, becomes thickened to form the ciliary body. Over the ciliary body the outer lamina of the ocular vesicle alone is pigmented, and the inner is not, just as is the case with the two layers of cells of the pars ciliaris retinæ in the adult eye (*P* and *C* in Figs. 145 and 146). Farther forward, over an area corresponding to the posterior surface of the mesodermal rudimentary iris, both laminæ of the ocular vesicle are pigmented, and the two unite after undergoing reflection at the border of the pupil. Conjointly they form the retinal pigment layer of the iris (see page 367).

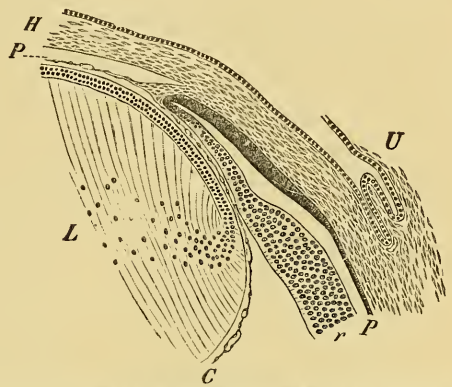


FIG. 163.—ANTERIOR SEGMENT OF THE EYE AT A PERIOD OF DEVELOPMENT CORRESPONDING TO THAT OF A HUMAN EMBRYO AT THE END OF THE THIRD MONTH. Magnified 80 × 1.

The epithelium of the cornea, *H*, is continued over upon the conjunctiva as far as the retrotarsal fold, *U*. Behind the cornea is seen the front wall of the ocular vesicle. The two laminæ forming this wall are reflected so as to unite with each other at a point corresponding to the margin of the pupil. The external lamina, *P*, is pigmented throughout, the internal lamina being pigmented only in its anterior portion, where it later is converted into the posterior stratum of the retinal pigment layer of the iris. Farther back, where the inner lamina is destitute of pigment, it is afterward converted into the inner layer of cells of the pars ciliaris retinæ, which lines the ciliary body. Still farther back a sudden swelling out of the inner lamina denotes the beginning of the retina proper, *r*, at a spot corresponding to what is afterward the ora serrata. The two laminæ of the ocular vesicle, so far as they constitute the coating of the ciliary body, lie closely applied to the mesodermal envelope. Farther, forward, at a point corresponding to the rudimentary iris they separate from the cornea, from which they receive a covering of mesodermal tissue, afterward converted into the stroma of the iris. From the free border of this tissue are given off two membranes—the membrana pupillaris, *P*, which passes to the opposite pupillary border, and the membrana capsularis, *C*, which runs backward, between ciliary body and lens, to the posterior surface of the latter. In the lens, *L*, the circle of nuclei is carried farther forward than in Fig. 162, and the shape of the lens in cross section has already become more elliptical.

334. Lids, Lachrymal Passages.—The lids originate as folds, which keep growing out above and below the eye from the skin surrounding it, until their edges come into contact. They then become united, but only by their epithelial lining; and shortly before birth this union is dissolved. The lachrymal gland originates from a bud-like intrusion of the epithelium of the conjunctiva into the orbital tissue. The lachrymal canal starts from a channel which exists even at an early period between the superior maxillary and the external nasal processes.

CHAPTER V

DISEASES OF THE IRIS AND CILIARY BODY

I. Inflammation

335. The iris and the ciliary body form a continuous whole, inasmuch as the iris springs from the ciliary body; both, moreover, are supplied by the same blood-vessels. It is hence quite easy to understand that both organs are very frequently diseased at the same time. Unmixed inflammation of the iris (iritis) or of the ciliary body (cyclitis) is rare; in most cases we have to do with a combination of the two (irido-cyclitis). For practical reasons, however, it is advisable to describe the symptoms of iritis and of cyclitis separately, and then show what sort of clinical picture is produced by their combination.

336. Symptoms of Iritis.—The symptoms of iritis are partly referable to the hyperæmia of the iris, partly to the formation of exudation.

Hyperæmia of the iris manifests itself chiefly by the discoloration which causes a blue or gray iris to appear greenish—a change which is particularly striking when comparison is made with the iris of the other eye, in case this is healthy. In dark eyes the discoloration is less pronounced. Sometimes, with the aid of a magnifying glass, we can clearly distinguish the separate dilated blood-vessels under the form of red striæ or maculæ. The other changes found concern the pupil, which is contracted, and does not react as well as usual. The contraction is a necessary result of the dilatation of the iris due to the increased fullness of the vessels; besides, there is a spasm of the sphincter produced by the irritation. For these reasons the reaction of the iris to light is diminished, and atropine also acts less promptly and less thoroughly than usual. The hyperæmia of the iris is accompanied by [conjunctival and] ciliary injection, photophobia, and increased secretion of tears.

The symptoms of congestion just described may exist by themselves without symptoms of exudation, in which case we do not speak of iritis, but merely of hyperæmia iridis. This is observed as a result of the same causes as iritis itself, in case the irritation is not great enough to provoke actual inflammation. Pure hyperæmia of the iris is most frequently seen in corneal affections, and particularly in case of small ulcers or foreign bodies in the cornea. Hyperæmia of the iris, provided it is not the precursor of an iritis, disappears without leaving any lasting traces of its presence.

Inexperienced physicians often fail to recognize slight cases of iritis, considering it as catarrh on account of the injection of the eyeball that coexists with it. The treatment then employed, such as the application of the silver-nitrate solution or the instilla-

tion of irritating collyria, commonly aggravates the iritis. We can avoid this mistake if in every case we pay careful attention to any discoloration that there may be in the iris (particularly by comparing it with the iris of the other eye), and also to the dimensions of the pupil, which in an eye with iritis is contracted. Moreover, a slight turbidity of the aqueous can be recognized, even in the early stages, from the fact that the pupil has not the same pure black look as in the other eye. On the other hand, the mistake is often made of considering an inflammatory glaucoma as an iritis on account of the ciliary injection and the discoloration of the iris—a mistake which is the more disastrous because in glaucoma great harm is done by the instillation of atropine. Here, besides the testing of the tension, our chief means of guarding against mistakes is the examination of the pupil, which in iritis is always more contracted, in glaucoma always more dilated, than usual.

[In the earliest stages of iritis when there are simply hyperæmia and cramp of the iris, the diagnosis is sometimes difficult. Homatropine should then be used. (If there is a suspicion of glaucoma, the homatropine may be replaced by euphthalmine or even cocaine. In that case if increase of tension occurs with the dilatation, this can easily be controlled by eserine, and if, on the other hand, the euphthalmine or cocaine fails to act, they can at once be replaced by homatropine.) If one instillation of a 2-per-cent solution of homatropine combined with 4-per-cent cocaine fails to dilate the pupil in twenty minutes, repeated instillations should be made until the pupil does dilate, or it is evident that it will not do so. In the latter case atropine should be instilled at once. If under either homatropine or atropine the pupil dilates sluggishly, imperfectly or unevenly and if, furthermore, when the dilatation is finally effected the pain and photophobia are relieved, the diagnosis of iritis is confirmed. Adrenaline helps, too, by dispelling a superficial injection and so bringing to view a deep injection which the drug does not much reduce.—D.]

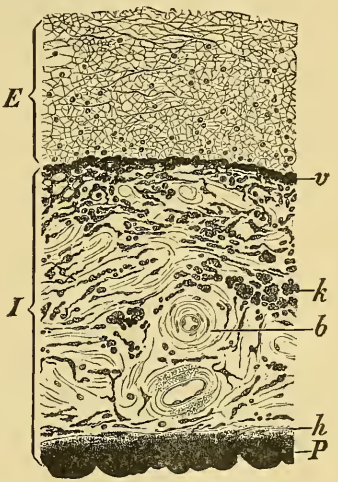


Fig. 164.—Iritis. Magnified 116 × 1.

Upon the surface of the iris is a layer of exudate, *E*, consisting mainly of a fine network of coagulated fibrin, in which lie scattered pus corpuscles. The iris, *I*, is bounded in front by the anterior limiting layer, *v*, behind by the posterior limiting layer, *h*, and the retinal pigment layer, *P*. In the tissue of the iris are recognized the cross sections of blood-vessels, *b*, with very developed adventitia, and a great quantity of pigmented cells (the specimen is taken from a dark-brown iris). Most of these (chromatophores) still retain their elongated and branching shape, but others, at *k*, are transformed into shapeless aggiomerations of pigment, as is usually the case in inflammation of the iris. In the anterior layers of the iris many small cells (emigrated leucocytes)—likewise an evidence of inflammation—lie between the pigment cells.

337. *Exudation* takes place partly into the tissue of the iris itself, partly into the surrounding cavities, the anterior and posterior chambers, and is accordingly characterized by varying symptoms:

(1) *Exudation into the tissue of the iris* makes the latter, since it is filled with an abundance of round cells, appear swollen and thicker than usual. The *discoloration* is still more pronounced than in simple hyperæmia, and the clear-cut markings upon the anterior surface of the iris are obscured. It is easy to understand that the rigid and swollen iris should react but insufficiently to light; the pupil is greatly contracted.

338. (2) *Exudation into the anterior chamber* manifests itself first by *turbidity of the aqueous*, in which numerous exudation cells are suspended. The turbidity is best recognized upon the dark background of the pupil, which in this case looks gray instead of being a pure black. Gradually the formed constituents floating in the aqueous sink to the bottom of the chamber, where they produce a *hypopyon* (Fig. 98, A and B). When there is very great hyperæmia, rupture of the blood-vessels in the iris may take place with an extravasation of blood, which also sinks to the bottom of the anterior chamber (hyphæma).

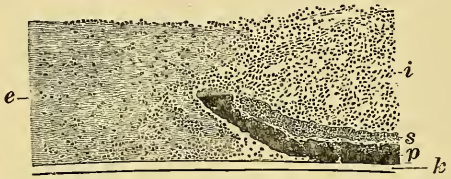


FIG. 165.—RECENT IRITIS WITH PUPILLARY MEMBRANE. Magnified 55 × 1.

The iris, *i*, is greatly thickened by swelling and infiltrated by numerous round cells. Attached to the pupillary margin is an exudate, *e*, which fills the whole pupil, and which, on the one hand, pushes its way somewhat beneath the pigment layer, *p*, and, on the other hand, extends up on the anterior surface of the iris. That it is a quite recent exudate is evident from its thickness and from its being composed of a network of fibrin, inclosing scattered pus corpuscles. The number of the latter increases toward the surface of the iris. *k*, capsule of the lens.

FIG. 166.—OCCLUSIO PUPILLÆ TWO MONTHS AFTER A PERFORATING INJURY. Magnified 55 × 1.

The exudate, *e*, has become converted into connective tissue, and has, in consequence, shriveled down to a thin pellicle, which, becoming constantly more and more attenuated, can be traced over the entire surface of the iris. The iris itself has become thinner from atrophy, and where it ends at the pupillary margin is tapered off owing to its being pulled upon by the pupillary membrane. The pigment layer, *p*, is the part most drawn out into the pupil, so that it projects a good bit beyond the sphincter, *s*, and the stroma of the iris, *i*. Hence the margin of the pupil in the living eye, when looked at from in front, seems as though encompassed by a broad brown rim, which appears to attach the edge of the pupil to the capsule of the lens.

FIG. 167.—OCCLUSIO PUPILLÆ THREE MONTHS AFTER A PERFORATING INJURY. Magnified 55 × 1.

The exudate, *e*, is converted into a thin membrane of connective tissue, which, at the pupillary border of the iris, extends mainly beneath the latter, and can be traced as a delicate pellicle for a long distance between the pigment layer, *p*, and the lens capsule, *k*. The tension set up by the pupillary membrane upon the border of the atrophic iris, *i*, has drawn the latter down over the pigment layer, so that the sphincter iridis, *s*, which is solidly united with the pigment layer, has its anterior border turned back in a hook-shaped bend. In the living eye the margin of the pupil appeared encompassed by a gray rim which merged gradually into the less gray, because more transparent, pupillary membrane.

Besides the exudates suspended in the aqueous, a layer of exudate is also found covering the walls of the anterior chamber. Owing to the exudate deposited on its posterior surface (Fig. 170, *f*) the cornea appears faintly and uniformly clouded. Moreover, the layer of exudate deposited upon the iris (Fig. 164) contributes essentially to the hazy appearance of the iris markings. The layer of exudate extends from the iris upon the anterior capsule of the lens (Fig. 165, *e*), and covers the latter throughout the area of the pupil, which consequently appears gray.

If this exudate becomes organized, a membrane is produced which closes the pupil and is connected with the pupillary margin of the iris (Figs. 166 and 167, *e*). This is called a pupillary membrane, and the condition thus brought about is called *occlusio pupillæ* (Fig. 172). It is evident that this condition must result in a very considerable impairment of vision.

A peculiar appearance is furnished by the presence of a pretty large *fibrinous* exudate in the anterior chamber, such as is at times observed in every sort of acute iritis. If, for example, much fibrin is deposited in the aqueous, it may become coagulated into a uniformly gray, translucent mass (hence the name gelatinous exudate and also lenticular exudate, because with its rounded edges it sometimes looks like a half-transparent lens that has been dislocated into the anterior chamber). The exudate speedily shrinks, since the fibrin in contracting expels the liquid from its meshes (for which reason we also speak of it as a spongy exudate).[1] After a few days the exudate has either completely disappeared or it has been reduced to a thin pellicle lying in the pupil and often still connected with the pupillary margin by one or two slender filaments.

339. (3) A special form of exudate that is found particularly in the chronic cases, are the *precipitates* (deposits) upon the posterior surface of the cornea. These are small dots, no bigger than a pin's head, of a light

Fig. 168.

Fig. 169.

Fig. 168.—Precipitates. Besides minute deposits, there are found also large ones, which are light gray and lardaceous looking.

Fig. 169.—Precipitates. These are small and disposed in the form of a triangle.

gray or brownish color, which lie upon the posterior corneal surface (*p*, Fig. 172). They were formerly thought to be located in the cornea itself; but if by puncturing the cornea the aqueous is allowed to escape, some of the deposit may be seen to disappear too, being swept away with the aqueous—a proof that they simply lie upon the posterior surface of the cornea. If the deposits are large, but few of them are generally present, and they are then commonly scattered irregularly over the cornea (Fig. 168). The smaller the deposits, the more numerous they generally are. They then occupy the lower half of the cornea, in which they cover a surface having the shape of a triangle. The base of the triangle corresponds to the lower corneal margin, and its apex is directed upward toward the center of the cornea. It is frequently observed that the deposits diminish in size from the base toward the apex of the triangle (Fig. 169).

The peculiar arrangement of the deposits is readily explained by their mode of origin. The deposits are conglomerations of cells, agglutinated into masses by means of fibrin (Fig. 170). At first they are suspended in the aqueous, and by the movements of the eye are thrown by virtue of centrifugal force against the posterior surface of the

[1] [Iritis associated with this sort of exudate is often called *spongy iritis*.—D.]

cornea and adhere to it. In so doing they arrange themselves according to weight, the largest being lowest down. The triangular shape of the arrangement is a result of the movements of the eyeball, by which the deposits are cast upon the cornea. We have only to think of what happens when we throw sand through a wire screen or shake grain in a sieve. The little fragments of stone or of grain always form a pointed figure with the apex, which contains the finest particles, running upward, while successively coarser particles follow in order below. The same is the case with the deposits. By their peculiar arrangement the deposits are generally easily distinguishable from macular opacities in the cornea itself (in keratitis punctata, see pages 287 and 290). Other distinguishing marks are the clearer outline and frequently the brownish color of the deposits, which, moreover, do not lie at different depths like maculæ in the cornea itself, but lie all in the same plane—that is, on the posterior surface of the cornea.

The deposits are easily overlooked, because they are often so extremely *minute*. We should, therefore, in every case in which there is a suspicion of the existence of an affection of the uveal tract, look for them with a strong magnifying glass. In slight cases of chronic cyclitis (see page 407) a couple of such very minute precipitates are often the only objective sign of the disease, and if we overlook them we are often unable to account for the patient's subjective troubles, for the sensitiveness of the eye to light,

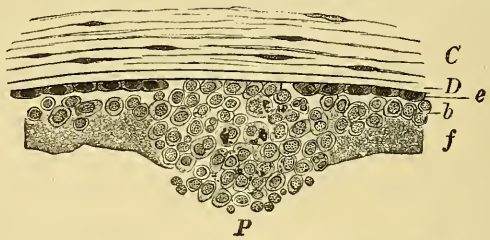


Fig. 170.—Deposit Upon the Posterior Surface of the Cornea. Magnified 140 × 1.

The posterior surface of the cornea, *C*, is covered by Descemet's membrane, *D*, and the endothelium, *e*. The latter, which as a whole is of normal character, is wanting at the spot where the deposit, *P*, is situated. This deposit forms an accumulation of cells with interspersed pigment granules which are partly free and partly inclosed in the round cells. In the place where no deposits are situated the posterior surface of the cornea is covered by a layer of exudation consisting of two strata, an anterior one, *b*, composed of round cells, and a posterior one, *f*, formed of coagulated fibrin.

for the readiness with which it tires, or for the occasional slight pain or moderate reddening of the eye. Faulty diagnoses, too, are easily made. As an example for this I may adduce the fact that repeatedly patients have been sent to me in order that I might do an iridectomy on them for chronic glaucoma. It was usually a case of persons who were not yet of the advanced age of ordinary glaucoma patients and who from time to time got attacks of increase of tension associated with the appearance of colored rings about a light, pain, dilatation of the pupil, etc. They had, accordingly, the symptoms that belong to the prodromal stage of glaucoma (§ 442). But the discovery of a few minute deposits proved to me that the case was not one of genuine primary glaucoma, but of an increase of tension due to a chronic cyclitis (secondary glaucoma). There was, moreover, another symptom that perforce led one on the right track, and that was the fact that the anterior chamber was deeper than normal, while in glaucoma it is shallower. In such cases the diagnosis is important because in chronic cyclitis the increase in tension can often be relieved even without an iridectomy.

In contradistinction to the scarcely visible deposits, spoken of above, there are also found especially *large* gray lardaceous-looking deposits which as the disease progresses, change their shape, enlarge, and coalesce with neighboring precipitates to form biscuit- or trefoil-shaped masses of exudation (Fig. 168). Large deposits of this sort occur pre-eminently in the tuberculous or scrofulous inflammations of the uvea.

On careful inspection with the loupe we sometimes find deposits in the pupillary area and particularly *on the iris*. It is true, they are hard to see, in this situation, especially on a light-colored iris with which they do not contrast well. The deposits probably get on the iris at night, when the patient lies for a long time quietly on his back, so that the agglomerations of exudate floating in the aqueous are not thrown against the cornea by the movements of the eye, but sink gradually down upon the iris.

Most deposits get *darker*, as they get older. For, the cells of the exudate break down and become absorbed, so that the pigment granules, which lie between them and which are left behind, come more and more clearly into view. Ultimately there remain dark brown or black dots, which may readily be confounded with small black foreign bodies, if no heed is paid to their deep situation. In rare cases the deposits are dark brown, even when recent, if at the outset they have carried much pigment with them from the uvea.

I have quite frequently found minute deposits in eyes in which a soft cataract had been operated upon by discission, and was consequently swelling up and undergoing absorption at the time. These so-called *lens deposits* are, however, essentially different from the genuine deposits; they are not agglomerations of exudation, but small rounded lens fragments, detached from the swelling lens and thrown against the posterior surface of the cornea, to which they then adhere. They are therefore in no sense to be regarded as a sign of inflammation; and this view of their nature is confirmed by the favorable course that such cases pursue.

340. (4) Exudation poured out into the *posterior chamber* is not accessible to direct observation, but manifests itself only by the adhesions which it causes between the iris and the capsule of the lens (*posterior synechiæ*). These adhesions develop principally at the spot where the iris and the capsule of the lens are in contact—i. e., at the pupillary margin. They form at the time when the iritis is at its height, and when, therefore, the pupil is greatly contracted. When, after the iritis has run its course, the pupil tends to resume its usual mean width, this is only possible over the area in which the pupillary margin has remained unattached. The portions that are adherent to the capsule of the lens cannot retract, but remain as tags of greater or less size, projecting in toward the center of the pupil. The pupil thus acquires an irregular shape which is still more obvious if atropine is instilled; for, as the iris then retracts strongly at its unattached portions, the synechiæ stand out in the clearest possible way (*a* and *b*, Fig. 171). Atropine is hence a very valuable agent for the diagnosis of posterior synechiæ.

In the formation of posterior synechiæ, it is not the stroma of the iris, but the layer of the retinal pigment [Figs. 165, 166 and 167, *p*] covering its posterior surface, that becomes adherent to the capsule of the lens When the iris starts to retract, the pigment layer is held back at the points of adhesion, and is thus exposed to view more extensively than usual. Hence in dilatation of the pupil, especially by atropine, the tags jutting out into the pupil look brown (Fig. 166). From the traction exerted by the iris, rupture of the synechiæ may result. This sometimes is effected spontaneously by the traction which is constantly being made upon the

adhesions during the ceaseless movements of the iris; for the most part, however, rupture is produced artificially by the instillation of atropine. In that case we see, at the spot where the synechia has been set free, a brown spot remaining upon the anterior capsule of the lens. This is the pigment layer whose pathological adhesion to the capsule of the lens is firmer than its physiological connection with the tissue of the iris. If several synechiæ have been ruptured, we find remains of them in the shape of a corresponding number of brown dots arranged in a circle upon the anterior capsule of the lens (Fig. 171, between and on either side of *a* and *b*). This circle is narrower than the mean diameter of the pupil, because the synechiæ were formed at the time when the pupil was contracted by iritis. The dots of pigment never disappear, and hence during the whole life give evidence of an iritis that has once existed.

It happens sometimes that posterior synechiæ develop in the presence of a dilated, instead of contracted, pupil; as, for example, when they form in an eye under the influence of atropine. In this case the margin of the pupil becomes adherent to the capsule of the lens at some peripherally located spot, so that afterward, when the pupil assumes its mean width, the synechia is marked, not by a projecting process, but by a re-entrant angle.

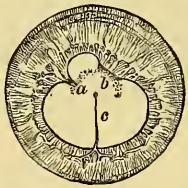


FIG. 171.—POSTERIOR SYNECHIÆ AND REMAINS OF FETAL PUPILLARY MEMBRANE. Magnified 2 × 1.

The pupil has been dilated by the instillation of atropine. The dilatation, however, is unequal, because the upper part of the pupillary margin is fixed by means of adhesions to the anterior capsule of the lens. At *a* there is a slender synechia which is drawn out into a fine black point; at *b*, on the other hand, is found a broad and but slightly elongated adhesion, such as frequently occurs, especially in syphilitic iritis. Between the synechiæ, and by the side of them, the capsule of the lens is covered with minute black dots arranged in a semicircle. They correspond to the situation of the pupillary margin when the pupil was contracted, and have been left by the rupture of the adhesions. From the lower part of the iris a filament, *c*, runs straight upward. This arises from the trabeculæ of the circulus iridis minor, and passes in the region of the pupil to the anterior capsule of the lens, where it is attached to a small, round, capsular opacity. This filament is not a posterior synechia, but a remnant of the fetal pupillary membrane. It does not prevent the iris from retracting properly under atropine, but is simply stretched and drawn out thin itself.

341. If the adhesion of the iris to the capsule of the lens is not confined to single points, but comprises the whole extent of the pupillary margin, we speak of an *annular posterior synechia*. No projecting tags are then apparent, because the iris is no longer able to retract at any spot, the pupil remaining unchanged all the time, even after the instillation of atropine. There is generally a brown rim (pigment) or a gray rim (exudate) investing the pupillary margin (Figs. 166 and 167). An annular synechia is seldom formed all at once; it is for the most part the result of a number of recurring attacks of iritis, which little by little produce a more and more extensive adhesion of the iris to the lens. The direct consequence of an annular synechia is the shutting off of the anterior from the posterior chamber—shutting off of the pupil (*seclusio pupillæ*).

The two sequelæ of iritis, shutting off of the pupil (seclusio pupillæ) and shutting up of the pupil (occlusio pupillæ), very often occur together owing to the fact that the exudate which attaches the pupillary margin to the lens may also extend over the entire pupil (Fig. 172). But they may also occur separately and then have very different consequences. Occlusion of the pupil occurring by itself produces very great diminution of sight, without, however, entailing any dangers for the future. Seclusion of the pupil in itself does not affect the sight, if the pupil is free from membrane, but subsequently induces changes (increase of tension) which cause blinding of the eye.

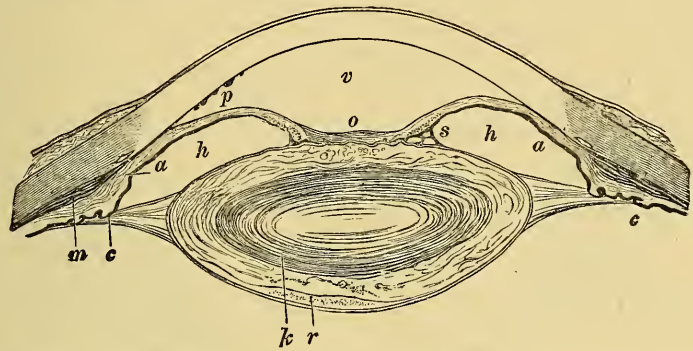


FIG. 172.—SECLUSION AND OCCLUSION OF THE PUPIL. Magnified 5 × 1.

The iris is adherent by its entire pupillary margin to the lens, but elsewhere is pushed forward. The posterior chamber, *h*, is thus made deeper, the anterior chamber, *v*, shallower, especially at the periphery where the root of the iris, *a*, is pressed against the cornea by the increase of tension. In consequence of the traction made upon the iris, its retinal pigment is beginning to separate (at *s*) and to be left upon the capsule of the lens. The pupil is closed by an exudate membrane, *o*, by the shrinking of which the anterior capsule is thrown into folds. In the lower part of the anterior chamber there is matter, *p*, precipitated upon the posterior surface of the cornea. In consequence of the increase in tension, both the ciliary processes, *c*, and the ciliary muscle, *m*, are atrophic and flattened. The cortex of the lens has undergone cataractous disintegration, and at *r* is separated from the capsule by liquor Morgagni; the nucleus, *k*, of the lens is unaltered.

For the diagnosis of *seclusio pupillæ*, the employment of atropine is often indispensable. It frequently happens that the pupillary margin is thought to be adherent to the capsule all round, when, nevertheless, upon the employment of atropine it retracts at one small spot. At this unattached portion, which is most frequently situated above, there is thus formed a curved or horseshoe-shaped indentation of the pupillary margin. Similarly we may infer the existence of a small opening if, after the case has been under observation for a long time, no protrusion of the iris takes place, since in true seclusio pupillæ such protrusion never fails to appear. Of course, we must be able to exclude the existence of a total posterior synechia, in which case evidently protrusion of the iris could not take place.

Seclusio pupillæ appears to occur frequently without there being any simultaneous formation of membrane in the pupil (occlusio pupillæ). This, however, is only apparent, as a rule. Upon careful examination we usually notice that the gray fringe of exudate which runs along the adherent pupillary margin projects far into the pupil, becoming gradually thinner as it does so, so that perhaps only the center of the pupil appears to be quite free. Nay more, if, after performing an iridectomy, we compare the pupil with the pure black coloboma, we can almost always convince ourselves that really no part of the pupil is quite free from a membrane.

Much more frequently than seclusion without occlusion, the converse—i.e., occlusion without seclusion—occurs. That is, there is a membrane in the pupil, and sometimes quite a thick one, which is not connected with the pupillary margin all round, but only at separate spots.

The presence of a well-marked reaction of the pupil to light is not altogether conclusive evidence against the existence of seclusio pupillæ. If the tissue of the iris is not yet atrophic, and a fair perception of light at the same time exists, the anterior layers of the iris, when the illumination is varied, move quite perceptibly over the fixed posterior pigment layer.

342. Symptoms of Cyclitis.—Exudation from the ciliary body, apart from the infiltration of the tissue itself, takes place into the anterior chamber, the posterior chamber, and the vitreous:

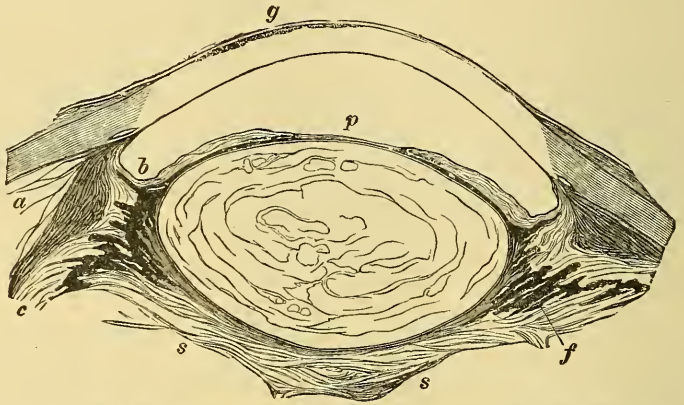


Fig. 173.—Total Posterior Synechia. Vertical Section through the Eye. Magnified 5 × 1.

The iris is adherent by its posterior surface to the capsule of the lens and also to the anterior surface of the ciliary body. The posterior chamber consequently is obliterated and the anterior chamber deepened at its periphery, *b*; at this spot the iris is strongly retracted and at the same time is here the most thinned through atrophy. The exudate connecting the iris with the lens also stretches as a thin membrane, *p*, across the pupil. The hull of exudate, *s*, springing from the ciliary body, envelops the posterior surface of the lens and by its shrinking draws the ciliary processes toward the center. As a result of this, a separation of the ciliary body, *c*, from its bed has already taken place below, and in the intermediate space are seen the disjoined lamellæ of the suprachorioid membrane, *a*. The pigment epithelium, *f*, of the ciliary processes has undergone proliferation. At the lower part of the cornea there is a zonular opacity, *g*. The lens is swollen and is opaque throughout; there is no hard, undisintegrated nucleus (i. e., it is a soft cataract).

1. The exudate produced by the ciliary body may get into the *anterior chamber* directly, in case there is implication of the most anterior portion of the ciliary body—i. e., that covered by the ligamentum pectinatum and lying at the confines of the anterior chamber. Thus a hypopyon may be poured out by the ciliary body, and especially there may be produced the precipitates, which are so frequent in chronic cyclitis. Particularly characteristic of cyclitis are the gray or grayish-white exudates that develop in many cases and which appear to grow out in the form of spongy masses from the sinus of the chamber, and that not merely below, but also from other directions.

2. In consonance with the anatomical situation of the ciliary body the great mass of the exudate is deposited *in the posterior chamber* (Fig. 177)

and into the anterior portion of the vitreous. This exudation, if excessive, leads to the adhesion of the whole posterior surface of the iris to the anterior capsule of the lens (*total posterior synechia*, Fig. 173). This adhesion of the surface of the iris is distinguished from the annular synechia, in which only the pupillary border of the iris is attached to the capsule, chiefly from the altered form of the anterior chamber. The exudate, as it shrinks, draws the iris everywhere up to the anterior surface of the lens, so that the posterior chamber is completely obliterated. The anterior chamber is hence proportionally deeper, above all at the periphery, where the iris is displaced farthest backward [*b*, Fig. 173].

3. Exudation *into the vitreous* appears under the form of opacities of the vitreous, which can be made out with the ophthalmoscope if the condition of the refracting media permits. Moreover, they manifest themselves by a corresponding diminution of the vision. In severe cases, an exudation of large size is present in the anterior division of the vitreous (*s*, Fig. 173), and under favorable circumstances can be seen with lateral illumination as a gray mass behind the lens. The sight is then almost completely abolished, and atrophy of the whole eye is subsequently produced by the shrinking of the mass of exudation.

The *tension* of the eye, which in iritis is usually unchanged, often shows an alteration in cyclitis. It is not infrequently found to be elevated in the beginning of the cyclitis; indeed, such a great elevation of tension may be developed that blindness is produced by it. In the later stages of cyclitis, on the contrary, diminution of the intra-ocular pressure is more frequent, and is the result of the shrinking of the exudate while in process of organization.

Since the aqueous in irido-cyclitis is more albuminous, it filters with more difficulty through the ligamentum pectinatum, especially if the gaps in the latter are partially blocked by coagula or leucocytes. In this probably consists the cause of the frequently occurring increase of tension. In this case, in consequence of the retention of the aqueous, the anterior chamber is deeper than normal, while in true glaucoma it is shallower. The increase of tension in irido-cyclitis is rarely so lasting that the eye would be blinded by it without an operation; usually the increase of tension is but transient, although it may occur repeatedly in the course of the disease.

343. Subjective Symptoms.—Both iritis and cyclitis are associated with *symptoms of inflammatory irritation*, consisting of ciliary injection, photophobia, lachrymation, and pain. The pain is not only situated in the eye itself, but radiates to the parts in the vicinity, especially the supra-orbital region. The intensity of the irritative symptoms is regulated by the greater or less acuteness with which the case runs its course. Chronic cases occur in which inflammatory symptoms are wanting altogether, so that the eye is at no time reddened or painful; on the other hand, there are cases of irido-cyclitis in which the pain reaches an absolutely intolerable

pitch and is associated with vomiting and a febrile movement. Sometimes the pain sets in with special violence at night (particularly in syphilitic iritis and irido-cyclitis).

Vision is always diminished through turbidity of the aqueous or through exudation in the region of the pupil or in the cavity of the vitreous.

In recent acute iritis and irido-cyclitis examination of the sight discloses sometimes the existence of a moderate degree of *myopia*, which did not exist previous to the inflammation, and which, moreover, gradually disappears again after the latter has run its course. [This has been attributed to spasm of the accommodation due to irritation of the ciliary muscle and to increased refractivity of the aqueous due to exudation. Neither explanation is satisfactory. In testing the vision in iritis it is important to remember that iritis of this sort may be present and to see if the poor vision found cannot be improved by a concave glass of moderate strength; otherwise, we may think that the sight is poorer than it really is.—D.]

In severe cases of irido-cyclitis (especially of sympathetic origin) *blanching of the cilia* on the lids of the affected eye is sometimes observed.

344. Differential Diagnosis Between Iritis and Cyclitis.—We speak of *iritis* if the symptoms enumerated above are present, and there is no direct proof of the participation of the ciliary body in the inflammation. That the ciliary body is pathologically altered in most cases of apparently simple iritis is put beyond a doubt by anatomical investigations. But, since we cannot see the ciliary body directly, slight changes in it escape diagnosis. We hence make the diagnosis of *irido-cyclitis* only in those cases in which in addition to the symptoms of iritis positive evidences of involvement of the ciliary body are also present. This is the case—

1. When the inflammatory symptoms reach a considerable pitch, and especially if œdema of the upper lid is associated with them—a thing which does not occur in simple iritis.

2. When the eyeball in the ciliary region is painful to the touch.

3. When from the presence of a pupillary membrane, of many posterior synechiæ, or of an annular or a total adhesion of the iris to the lens capsule, we can infer that the exudation is especially great.

4. When the disturbance of vision is more considerable than one would expect from the opacities within the confines of the anterior chamber. We are then justified in inferring the existence of opacities in the vitreous causing diminution of vision.

5. If the tension is altered—either elevated or lowered.

The participation of the ciliary body in the inflammation of the iris makes the disease a far more serious one and renders the prognosis worse. Not only is the inflammation more violent, but it induces changes which are much more difficult to remedy. The exudates, such as posterior synechiæ and pupillary membranes, produced by iritis in the confines of the anterior chamber, may be attacked successfully by operative procedures. But the exudates left by cyclitis in the vitreous, so far as they do not become

absorbed spontaneously, are in no way susceptible of removal. Severe cyclitis leads to destruction of the eye (atrophy of the eyeball)—a thing that never occurs with iritis alone.

Simple cyclitis[2] without iritis occurs only in the chronic form. The inflammatory symptoms are slight or absent, the iris is of normal appearance, and the pupil is generally somewhat dilated. The chief symptoms are the presence of deposits upon the cornea and opacities in the vitreous.

345. Course and Termination of Iritis and Cyclitis.—With regard to the course, a distinction is made between acute and chronic cases. The former are associated with marked inflammatory symptoms, but run a quicker course. But even in the acute cases—if we except the very slightest ones—it takes four weeks or more before the inflammation entirely subsides. The first signs of a change for the better in the inflammation are the decrease of the injection and of the pain, and especially the prompt action of atropine, while at the acme of the inflammation the pupil is so spasmodically contracted that atropine has little or no effect.

Chronic cases run their course with few or absolutely no symptoms of inflammation. The patients, in the latter case, become aware of the existence of their trouble only at a comparatively late period, and then only from the increasing disturbance of vision. Chronic iritis (irido-cyclitis and irido-chorioiditis) is not infrequently protracted over a course of years.

Inflammations of the iris and of the ciliary body often show a great tendency to *relapse*. Formerly the chief cause for the recurrence of the inflammation was supposed to lie in the posterior synechiæ left by the first attack of iritis. It was believed that in the constant movement of the pupil traction was continually made upon the iris at the places of adhesion, and thus a new source of inflammatory irritation was provided. But it has been demonstrated that relapses are to be apprehended only in certain cases of posterior synechiæ. If, for instance, anybody has had an ulcer of the cornea, and, as a result of it, an iritis from which there are some synechiæ remaining, he need never fear having a relapse of his iritis. But another man who has got synechiæ as the result of an iritis with a constitutional basis (e. g., a syphilitic or rheumatic iritis) can very readily have recurrences. We hence conclude that what produces the recurrences is not the synechiæ, but the continuance of the same constitutional cause that was accountable for the first attack of iritis; and, as a matter of fact, we sometimes see a syphilitic patient suffering from a recurrence of his iritis even when the first attack has got well without leaving any synechiæ; or we see a recurrence affecting, not the eye that was previously diseased but the other eye which has hitherto been sound. The recognition of the fact that one or two synechiæ are in themselves of no great significance has had an important practical result: the numerous operative methods designed for the division of synechiæ have now been entirely given up.

[2] The serous iritis of authors.

Recurrences of iritis are often less severe than the primary inflammation; but, as they are pretty frequently repeated, and as they leave a new exudate after them each time, they ultimately lead to serious changes, such as seclusio and occlusio pupillæ.

346. Favorable Outcome.—The outcome of inflammation may be a perfect cure in light cases. The synechiæ rupture, leaving behind tags of pigment attached to the anterior capsule, which are unproductive of injury to the eye. The hypopyon disappears by resorption. The deposits generally persist for a long time (for months), until they also are removed by resorption. In many cases they leave at the spot where they were situated a permanent opacity of the cornea in the shape of a gray speck, or the pigment that is contained in them remains permanently as a black dot. The slighter vitreous opacities may all disappear completely by resorption.

347. Sequelæ.—In most cases, however, permanent sequelæ remain after iritis and cyclitis. These are posterior synechiæ, pupillary membrane, exudates between the iris and lens and in the vitreous, changes in the cornea, and opacity of the lens.

348. (1) *Posterior Synechiæ.*—These are the most frequent of the sequelæ of iritis. If only a few are present, they cause no special injury to the eye, and also produce little or no impairment of sight. Very much worse is the annular posterior synechia or *seclusio pupillæ.* By this the communication between the anterior and the posterior chamber is obliterated. The aqueous secreted by the ciliary processes can no longer pass through the pupil into the anterior chamber; it hence collects in the posterior chamber, pressing the iris forward. A hump-shaped protrusion of the iris is thus produced, which finally reaches as far forward as the cornea, while the pupil is represented by a crater-shaped retraction of the pupillary margin which is attached to the lens capsule (Figs. 172 and 174). In consequence of being so greatly stretched, the iris becomes atrophic. To this condition elevation of the intra-ocular pressure (secondary glaucoma; see § 459) is added. The increased hardness of the eye can be established by palpation; the anterior ciliary veins are dilated; the cornea is dull and less sensitive to touch; the sight fails, with a decrease in the field of vision starting from the nasal side, until at length the perception of light is entirely abolished. Then the formation of scleral ectasiæ takes place in the blinded eye in the shape of anterior and equatorial staphylomata of the sclera. Seclusio pupillæ, accordingly, if it is not remedied in time, infallibly leads to blindness.

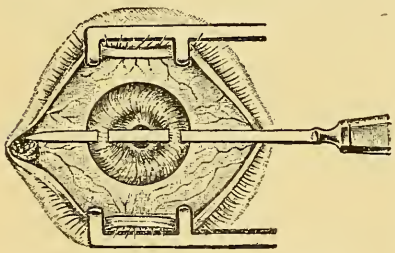


Fig. 174.—Protrusion of the Iris in Seclusio Pupillæ.

After Czermak-Elschnig.—The figure also shows the method of performing transfixion of the iris (see § 858).

The *protrusion of the iris* in seclusio pupillæ does not take place uniformly, but with the formation of projecting prominences separated by constrictions. The latter represent the more resistant, radial fibers which do not give way before the pressure of the aqueous until afterward. The condition in which the iris is found protruding in a greater part of its circumference, while one sector of it remains in its normal situation, is generally referable to the fact that at this spot an adhesion of the surface of the iris to the lens exists, preventing its protrusion (Figs. 179 and 180). It would, therefore, be a mistake to select just this spot for performing an iridectomy, although on other accounts it would seem to be very suitable for such a purpose, because of the greater depth of the chamber there. If the iris has been pushed forward as far as the cornea, it may become agglutinated to the latter in places, and in this way anterior synechiæ may be produced without there having ever been a perforation of the cornea (see page 317).

When in a case of seclusio pupillæ the iris is pushed forward as far as the cornea, there is not, for all that, a complete *abolition of the anterior chamber*, the latter being still present in the region of the pupil. It is otherwise in those cases in which the seclusio pupillæ has been produced not by irido-cyclitis, but by inclusion of the whole pupillary margin in the orifice of a corneal perforation due to ulcer or injury. In this case the liquid which accumulates in the posterior chamber pushes the iris in actually its whole extent against the cornea so that all traces of the anterior chamber vanish. Afterwards the iris becomes agglutinated to the cornea and ultimately becomes so atrophic as a result of the increase of tension that only its retinal pigment is left to cover the posterior surface of the cornea—a state of things which is particularly often met with in staphylomata of the cornea (see page 325 and Figs. 122–124). Finally complete abolition of the anterior chamber is produced in rare cases because the lens together with the iris is pressed against the cornea as a result of a tremendous increase of tension in the vitreous cavity (glaucoma malignum, intra-ocular tumors). In all the cases above cited of abolition of the anterior chamber the intra-ocular pressure is increased. In this fact lies a diagnostic sign distinguishing them from other cases of absence of the anterior chamber, in which the intra-ocular pressure is found to be diminished. To these latter cases belongs abolition of the anterior chamber due to leakage of the aqueous, either outside through a wound or fistula or into the suprachorioidal space in detachment of the chorioid (§ 427). Furthermore, the iris and lens may be pushed forward by a cyclitic membrane situated behind the latter, in which case, too, the eye is softer than normal.

349. (2) *Pupillary Membrane (Occlusio Pupillæ).*—This causes an interference with vision, the degree of which depends upon the thickness of the membrane.

350. (3) *Exudates behind the Iris.*—These lie either between the iris and the lens (forming a flat or, when of greatest extent, a *total posterior synechia*) or between the ciliary body and the lens and upon the posterior surface of the latter. In severe cases they form a coherent fibrous mass which completely envelops the lens, and which, on account of its solidity, is called the cyclitic hull (*s*, Fig. 173). This has a great tendency to shrink. In total posterior synechia there is evidently no hump-shaped protrusion of the iris—on the contrary, the anterior chamber in the beginning is usually deep at the periphery owing to the retraction of the iris (*b*, Fig. 173). Afterward, when atrophy of the eye sets in, the anterior chamber often becomes shallower again, because iris and lens together are pushed forward.

351. Either the repeated recurrences of an acute iritis or the sluggishly progressing chronic inflammations may lead ultimately to *atrophy of the iris*. This is characterized by a bleached-out, gray, or grayish-brown aspect of the iris (resembling gray felt or blotting paper); the delicate markings of the anterior surface have disappeared, and in their stead dilated vessels can often be recognized as reddish blotches upon the surface of the iris. The pupillary margin is thinned down, often looking as if it had been frayed out; the reaction of the iris is diminished or altogether lost. The great friability of the atrophic iris often renders the correct performance of iridectomy impossible.

Atrophy of the iris may set in: (1) As a result of long continued or frequently recurring inflammation. (2) In consequence of increase of tension. Here the main agent is the compression of the blood-vessels at the root of the iris, which is pushed away from the ciliary body and against the sclera (Figs. 215 and 216). The atrophy often develops quickly—in attacks of inflammatory glaucoma, sometimes within a few days. (3) In consequence of iridodialysis, as the result of which the vessels running from the circulus arteriosus iridis major to the iris are ruptured. (4) In consequence of

FIG. 175.—GAPS IN THE IRIS. Magnified 2 × 1.

Left eye of a girl who as the result of hereditary lues had become affected in the first year of her life with a bilateral irido-cyclitis with seclusio and occlusio pupillæ. Up to the age of six the girl was almost blind; then owing to the spontaneous formation of gaps in the iris of both eyes the sight improved to such an extent that she was able to go to school. At the age of twenty-two she suffered from a bilateral parenchymatous keratitis. In the membrane which closes the pupil are visible two thin spots appearing as black gaps. On the nasal side the stroma of the iris has disappeared to such an extent that there are left of it only a few gray ridges corresponding to the larger vessels, which run from the ciliary border to the region of the sphincter of the iris. Between these ridges the iris looks black either because the retinal pigment is exposed to view or else because it is absent altogether. In the places where the pigment is absent we can look right into the interior of the eye with the ophthalmoscope. The rest of the iris is to a moderate extent atrophic and its radiating fibers are tensely stretched.

traction. This occurs when the iris is attached both at its ciliary and at its pupillary border, and the distance between these two points gradually increases. This is most frequently observed when the iris is incarcerated in a scar of the cornea, and subsequently this scar or the entire eyeball becomes ectatic. So, too, stretching of the iris occurs when the pupillary margin of the iris becomes attached to the capsule of the lens in childhood as the result of iritis, and later on, with the growth of the eye the distance between the ciliary and pupillary margins increases (Fig. 175). (5) In consequence of a too thoroughgoing absorption; for example, when swelling fragments of the lens lie upon the iris and are gradually absorbed—a process, the result of which sometimes is that at the spot where the fragments are situated a portion of the iris tissue also disappears. (6) In advanced age.

The appearance of the atrophic iris likewise varies, as follows: (1) After inflammation and increase of tension the iris is like gray blotting paper, since owing to the shrinking it has become flat and hence has lost its delicate relief, and since furthermore the pigment of the stroma in great part has been destroyed. For this reason, too, a brown iris by atrophy becomes gray. (2) As a result of a circumscribed effacement of the stroma pigment, light colored or even white spots are formed in the iris (in glaucoma and after absorption of lens fragments). There are cases in which the iris has

numerous little white spots on its anterior surface (vitiligo iridis). Since the first case which Müller described in my clinic I have seen several others. In all these cases the patients had had variola, which without doubt must be regarded as connected with the spotty atrophy of the iris stroma. (3) Black spots form, when not only the stroma pigment but also the stroma itself has disappeared, so that the retinal pigment layer lies exposed. (4) If the latter also is destroyed, complete gaps are formed in the iris. We can then throw light through these with the ophthalmoscope, and the patient, too, can see through them, thus regaining vision when previously he was blind because of closure of the pupil proper (Fig. 175). If the atrophy affects the root of the iris, which is a specially thin part even when the iris is normal, a spontaneous iridodialysis is produced (§ 390). (5) Diminution in size of the iris always occurs in conjunction with atrophy of the latter, when the pupillary margin is not so attached to the capsule that such a diminution in size would be prevented. In glaucoma the iris may become so small as to disappear either in part or altogether, because it is concealed behind the limbus. (6) When the pupillary margin is free, the shrinking iris stroma usually draws the retinal pigment layer gradually over upon the anterior surface. The brown rim lining the pupillary margin hence becomes progressively broader (ectropion of the pigment layer, Fig. 216, *e*).

The dilated vessels that are frequently visible in the atrophic iris have quite often a course that does not in any way correspond with the regular radial arrangement of the normal vessels of the iris. As a matter of fact, vessels of this sort, as anatomical examination shows, do not lie in the iris itself, but in a thin exudation membrane deposited upon it. Often they extend over the pupillary border into the pupil, i.e., into the pupillary membrane.

352. (4) *Exudates in Vitreous.*—The exudates which lie behind the lens in the vitreous (*c*, Fig. 176) cause by their contraction a diminution of volume of the vitreous; the eyeball then becomes softer. The shrinking of the vitreous (*g*, Fig. 176) results in detachment of the retina (*r*) from the chorioid; in part also this detachment is produced by direct traction, since the hull of cyclitic membranes as it shrinks attaches itself to the inner surface of the retina and draws it out of its bed. In consequence of the detachment of the retina, complete blindness ensues. This state of things, consisting of diminished tension of the eyeball, with decrease in its size and with complete blindness, is known as *atrophy of the eyeball.* An atrophic eyeball presents the following picture: The whole eye is smaller and of slightly quadrangular shape. This is because the four recti muscles, stretching across the equator of the eyeball, press the sclera in somewhat at this spot, and hence produce flattening of the four sides. With the higher degrees of atrophy quite deep furrows are formed, so that the eyeball has the form of a bale of goods grooved by the cord with which it is tied. The cornea is smaller, often opaque and flattened; at other times, again, transparent, but abnormally protuberant or wrinkled. The atrophic iris is either pressed quite against the posterior surface of the cornea, or an anterior chamber still exists. In the latter case, we find the chamber bounded behind by a firm diaphragm in which the iris, which is imbedded in the hull of exudate, is often but indistinctly recognizable.

If the pupil is still distinguishable, a membrane and the opaque lens are found in it. The eye is softer, and is often sensitive to the touch. In the later stages markedly hard spots (ossified exudates) may sometimes be felt through the sclera.

Atrophy develops gradually during a course of months and years. The inflammation and the pain, which have been present for a long time, disappear when the atrophy is complete. But even then secondary attacks of pain occur, especially if the eye harbors a foreign body, or if ossification of the exudate takes place.

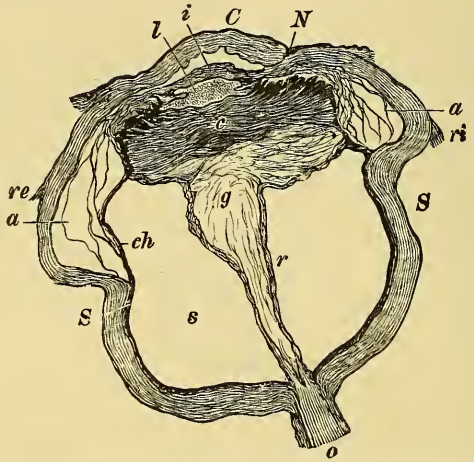


FIG. 176.—ATROPHY OF THE EYEBALL. (In part after Wedl-Bock.)

The eye is smaller and of irregular shape, chiefly from the grooving of the sclera, *S*, behind the points of attachment of the ocular muscles, the rectus internus, *ri*, and the rectus externus, *re*. The cornea, *C*, is diminished in size, flattened, and wrinkled, especially on its posterior surface. At its inner border it bears the depressed cicatrix, *N*, which was produced by the injury. The anterior chamber is shallow; the iris, *i*, is thickened and forms an unbroken surface, because the pupil is closed by exudate. Behind the iris lies the shrunken lens, *l*, and behind this is the great hull of cyclitic membrane, *c*, the shrinking of which is the cause of the atrophy of the eyeball. By reason of this shrinking, the ciliary processes, the pigment layer of which has markedly proliferated, are drawn in toward the center and, together with the adjacent chorioid, *ch*, are detached from the sclera; between the two structures are seen the disjoined lamellæ of the suprachorioid membrane, *a*. The retina, *r*, is detached and folded up into a funnel, which incloses the remains of the degenerated vitreous, *g*. The subretinal space, *s*, is filled with a fluid rich in albumin. The optic nerve, *o*, is thinner than normal and is atrophic.

353. (5) *Opacity of the Cornea.*—The cornea in marked irido-cyclitis is always somewhat dull; moreover, it may suffer from deeply situated infiltrates (keratitis pustuliformis profunda, see page 302), from the deposition on it of exudate (deposits, hypopyon), or from the contact of the iris, which when lasting a long time produces corneal opacity (page 301). In the stage of atrophy of the eyeball a zonular opacity frequently forms upon the cornea. Keratitis bullosa and vesiculosa also often make their appearance in eyes which are blinded by irido-cyclitis.

354. (6) *Opacity of the Lens.*—This develops in consequence of the disturbed nutrition of the lens. If merely a few synechiæ are present, it is rarely observed; on the other hand, it is observed with proportionately great regularity when seclusio pupillæ has existed for a long time, and par-

ticularly so in those severe cases in which the lens is completely swathed in cyclitic exudates. Such a cataract is denoted as cataracta complicata or cataracta accreta (= grown fast to—i. e., grown fast to the iris). In atrophic eyeballs the lens is always opaque, and generally shrunken as well.

355. Morbid Anatomy. (1) **Irido-cyclitis.**—Inflammation of the uvea is generally produced by ectogenous or endogenous infection (see pages 33 and 36).

(1) *Ectogenous Infection.*—Our best information with regard to the anatomical changes is derived from the cases of ectogenous *infection produced by a perforating injury*, because such cases most often come to enucleation and consequently to anatomical investigation. It is these cases, therefore, that we shall consider first. The clinical and anatomical picture of inflammation produced by the injury varies according as its starting point is in the anterior or posterior division of the eye, that is, the aqueous chamber or the cavity of the vitreous.

356. (*a*) *Changes in Anterior Chamber.*—When an infection has taken place in the *region of the aqueous chamber*, either from the introduction of the bacteria into the aqueous or into the tissue of the iris and ciliary body, the inflammation breaks out first in these two structures.

The iris is thickened, its blood-vessels distended. In its tissue lie uninuclear and multinuclear leucocytes in great abundance, and also a few red blood corpuscles; in particularly severe infection the tissue is permeated with extensive extravasations of blood and is often to a large extent necrotic. In the ciliary body it is mainly the lax tissue which adjoins the sinus of the anterior chamber that is infiltrated with leucocytes; and when the infiltration is great this tissue often can scarcely be differentiated from the hypopyon. From the inflamed tissue the exudate migrates into the aqueous chamber; the endothelial membrane, which lines the sinus of the chamber and the iris, being at the same time exfoliated. The exudate coagulates on the surface of the iris, forming a layer which consists of a delicate network of coagulated fibrin, which encloses leucocytes in varying amounts (Fig. 164). This coating of fibrin extends, on the one hand, within the pupillary area over upon the anterior capsule (Fig. 165), and, on the other hand, it often also covers the posterior surface of the cornea (Fig. 170). The aqueous chamber may even be entirely filled by such an exudate (Fig. 177). The more severe the inflammation is, the more leucocytes the exudate contains, and the hypopyon consists almost exclusively of leucocytes.

In less severe cases in which we have to do with an exudate of a more fibrinous character, the latter may disappear without leaving a trace or may lead to only a partial adhesion of the iris to the lens capsule. In severe cases the exudate becomes organized into connective tissue. The iris is then covered by a connective-tissue pellicle (Fig. 166), which is often quite thick and not infrequently contains new-formed vessels, which have grown from the iris into the membrane. The surface of this pellicle is sometimes covered by a new-formed lamina vitrea which at the angle of the chamber becomes continuous with Descemet's membrane. From the surface of the iris the iritic membrane, crossing the pupillary margin which is solidly adherent to the lens capsule, passes into the pupil and closes it up (Figs. 166 and 167). The organized exudate often glues the periphery of the iris to the cornea and may also in places cover the posterior surface of the latter. The iris itself gets to be atrophic. It is thinner and its lax reticulate

tissue is transformed into a rigid fibrillary connective tissue. The gracefully branched chromatophores are in large part changed to thick round cells filled with coarse pigment granules (Fig. 164). The blood-vessels have thickened walls of a hyaline appearance, and many are quite obliterated. The sphincter pupillæ and the retinal pigment offer the longest resistance to the atrophy.

As an addendum to the description of the exudation in the region of the anterior chamber, mention may be made here of the *precipitates*, although these do not occur at

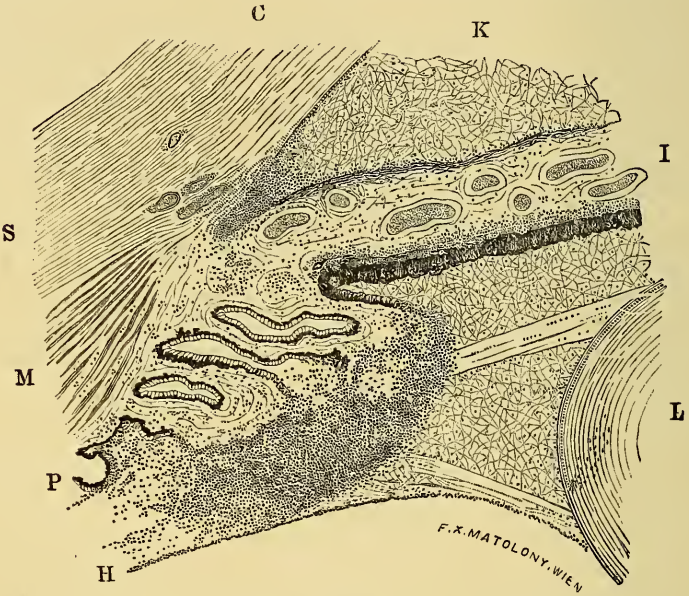


FIG. 177.—IRIDO-CYCLITIS AFTER PERFORATING INJURY. Magnified 30 × 1.

The cornea, *C*, and the sclera, *S*, are unchanged. Owing to the fact that the aqueous is rich in fibrin, and hence is coagulated by the hardening fluids used, the anterior chamber, *K*, is filled with a dense network of fibrin, in which lie isolated round cells (emigrated white blood corpuscles). These round cells are particularly numerous upon the surface of the iris, and above all in the sinus of the chamber; in fact, they fill the sinus completely, and form in it a low hypopyon which was visible in the living eye. A marked cellular infiltration can be seen surrounding Schlemm's canal and the cross sections of the anterior ciliary veins situated above the canal. The iris, *I*, appears broadened, as seen in cross section, this appearance corresponding with the swelling which existed in the living eye. Its vessels are dilated, and are distended with blood. In its stroma, particularly in the posterior layers, there are cells visible in great numbers; its retinal pigment layer is thickened and loosened. The greatest emigration of cells has taken place from the blood-vessels of the ciliary body, the direction pursued being a centripetal one—i. e., toward the interior of the eye. Hence the infiltration in the ciliary muscle, *M*, is but slight, in the ciliary processes, *P*, it is rather more pronounced, and upon the surface of the latter it is the most marked of all, so that the outer limits of the processes are concealed by the dense masses of cells. The hyaloid membrane, *H*, is pressed away by the exudate from the surface of the ciliary body. The contents of the posterior chamber, like those of the anterior, are formed of a fine network of fibrin with leucocytes imbedded in it, these latter being formed in specially large numbers along the hyaloid membrane. Two of the groups of fibers composing the zonule of Zinn are seen under the form of light-colored bands, passing through the coagulated contents of the posterior chamber to the lens *L*. The latter displays the epithelium of its anterior capsule and the nuclear ring and shows no morbid change.

all in acute inflammation due to a perforating injury, and at most occur only when such an inflammation runs a chronic course (iritis traumatica serosa). On the other hand, they are an almost constant occurrence in chronic non-traumatic inflammations of the iris and ciliary body. Hence precipitates and hypopyon, which is an accompaniment of acute inflammation, are not usually found together. The precipitates are conglomerates of round cells, many of which contain pigment granules, thus showing their origin from the uvea (Fig. 170, *P*). The precipitates lie on the endothelium of Descemet's mem-

brane, which at the outset is perfectly normal, and not till later on undergoes destruction beneath the accumulations of cells formed by the precipitate (Fig. 170, *e*). By many authors the term *iritis serosa* is used for the cases of uveitis that are associated with precipitates. But this name ought to be rejected, for the precipitates are certainly not a serous exudate, and those cases in which there is formation of precipitates and in which changes in the iris itself are not appreciable should not be called iritis at all, but cyclitis (page 407).

357. (*b*) *Changes in Posterior Chamber.*—The more severe the irido-cyclitis, the more does the tissue that lines the posterior chamber share in the inflammation.

Then it is above all the ciliary processes and mainly the most superficial portions of their tissue that are infiltrated with emigrated leucocytes. The exudate that comes out on the surface detaches the two retinal layers of the ciliary processes and covers the surface of the processes (Fig. 177).

The exudate poured out into the posterior chamber afterwards becomes organized into the cyclitic membranes, which form solid adhesions between the lens on the one hand and the iris and ciliary processes on the other (Fig. 173). The ciliary processes which are imbedded in the membranes become atrophic; on their surface the unpigmented and the pigmented layer of the retinal lining undergo proliferation, and send long rows of cells into the firm connective tissue of the membrane, which, moreover, contains new-formed vessels emanating from the ciliary processes. The lens gradually becomes opaque. If the lens capsule is injured either as a result of the traumatism itself or later as a result of its destruction by the exudate that presses against it, the lens substance disintegrates and gradually disappears and the exudate enters the capsular sac. This exudate here, too, becomes organized to form connective tissue, and may ultimately become ossified, so that the picture—to be sure, only an apparent one—of ossification of the lens is produced. The cyclitic membrane is often extremely tough, so that it grates when cut through; not infrequently it contains small laminæ of bone. Owing to its contraction, the ciliary processes are elongated more and more and are drawn into the interior of the eye. This constant traction upon a tissue, so richly supplied with nerves, is one of the causes of the constantly present or constantly recurring pain that distresses the patient in so many cases of old irido-cyclitis.

358. (*c*) *Changes in the Vitreous.*—Since that surface of the ciliary body which looks backward and inward adjoins the vitreous and is separated from it only by fibers of the zonula, it is easy to understand that in case of severe inflammation leucocytes will pass from the ciliary body into the vitreous. So in many cases in which the infection at first has affected only the aqueous chamber, it happens that later the vitreous cavity is also attacked by the inflammation. In other cases the *inflammation begins in the vitreous cavity*, this occurring when the agents causing the inflammation have been introduced into the latter first.

These agents act primarily on the surface of the membranes that line the vitreous cavity, i. e., the ciliary body and retina. The superficial layers of the ciliary body are densely infiltrated, while the ciliary muscle which lies deeper is commonly but little changed. An exudate lies on the surface of the ciliary body (Fig. 178, *b b*) and from it extends into the saucer-shaped depression between the lens and vitreous. In the retina, too, it is primarily the inner layers that are infiltrated with emigrated leucocytes,

these being particularly aggregated about the retinal veins which they surround with dense envelopes consisting of cells (purulent retinitis). The optic nerve-head is also swollen and infiltrated. From the retina the exudate passes out upon its inner surface, which in severe cases is often covered to a large extent with a layer of pus (Fig. 178, *c*). Very soon the infiltrated retina wrinkles (in Fig. 178 at a point near the outer margin of the optic nerve) and becomes detached over a progressively larger area. The infiltration of the retina and of the papilla and the detachment of the retina account for the rapid diminution and ultimate extinction of light perception in the cases in which the inflammation gets a foothold in the vitreous cavity.[3]

The chorioid is protected primarily by the retina against the action of the inflammatory agents that are present in the vitreous. If the retina becomes detached early, so that now subretinal liquid separates the chorioid from the inflamed retina, the chorioid may remain exempt from severe inflammation. If, on the other hand, the retina remains for a long time lying on the chorioid, the inflammation passes over to the latter; and the same thing happens when in the case of an especially severe inflammation the retina in places undergoes necrotic disintegration and the inflammatory agents can then act directly on the chorioid. The inflamed chorioid is densely infiltrated with leucocytes, and a free exudate collects both on its inner and on its outer surface. The outer exudate lies in the subchorioidal space, and hence produces a detachment of the chorioid.

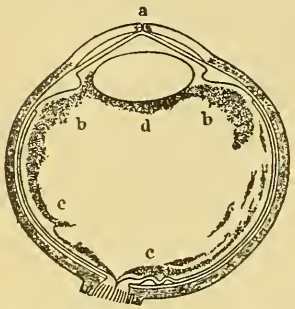


FIG. 178.—PURULENT INFLAMMATION OF THE INNER COATS OF THE EYE. HORIZONTAL SECTION.

Seven days previous to the examination a piece of iron had flown into the eye of an eighteen-year-old boy as he was watching a smith. In the cornea there is a perforating wound, *a*, to which runs the iris. A purulent exudate covers the inner surface of the membranes lining the vitreous cavity, namely, the ciliary body, *b b*, and the retina, *c c*. From the ciliary body the exudate, *d*, extends over the whole posterior surface of the lens. The ciliary body and retina are densely infiltrated, while the chorioid is scarcely changed. Near the entrance of the optic nerve the retina is beginning to detach under the form of two folds.

359. *Results of Changes in the Vitreous.*—In the cases in which the inflammation has been confined to the anterior chamber, changes remain which indeed are grave but which for the most part can be relieved by operation (§ 386). But if the vitreous cavity has been the site of the inflammation, it is but exceptionally the case that the eye is preserved, this occurring when the quantity of exudate has been so small that it is absorbed entirely except for a few membranes left behind in the vitreous.

But, as a rule, owing to the organization of the exudate in the vitreous with the subsequent shrinking of the new-formed connective tissue, *atrophy of the eyeball* sets in. The first sign of this is softening of the eyeball, which then gradually becomes smaller. If as the result of the injury a pretty large scar is present in the cornea or sclera, the intraocular membrane commonly becomes connected with this and, when it contracts,

[3] Hence, the way in which the light perception behaves is our most important guide for estimating the changes in the vitreous cavity, since such changes ordinarily are not directly visible. It is also decisive with regard to the question of enucleation, the latter being indicated as soon as the light perception is extinct. It is true that when such enucleated eyes are opened it is often found that the retina and optic nerve do not display the grave changes that we expect to find. Even then, however, the microscope shows serious changes in the optic nerve, such as disintegration of the nerve fibers, which may occur even when the inflammation in the posterior segment of the eye is otherwise quite insignificant. In other cases of this sort the optic nerve, even under the microscope, is found to be nearly normal. This agrees with the clinical fact that in many cases in which enucleation is not performed the light perception which has been almost lost is restored. It is conjectured that in these cases the diminution of light perception is caused by paralysis of the retina and of the optic nerve, poisonous substances (toxins) penetrating into these parts by diffusion from the exudate in the vitreous.

causes an indrawing of the scar—a condition which is recognized as one of the first clinical manifestations of beginning atrophy of the eye. When the eye gets very much diminished in size the sclera becomes furrowed; one furrow in particular is formed running around the eye behind the attachments of the recti muscles (*S*, Fig. 176). The consequences entailed by the shrinking of the new connective tissue become noticeable in the interior of the eye even sooner than they do exteriorly. This connective tissue draws up to it first the retina, which is readily movable anyhow and is already detached, and afterwards the ciliary processes and through these the anterior portion of the chorioid. Ultimately there is formed in place of the vitreous a small-sized mass of tough connective tissue, bounded in front by the lens, which is usually opaque and shrunken, and by the iris, which is joined to the lens. These two latter structures are sometimes driven forward, sometimes drawn backward, the anterior chamber being thus either abnormally shallow or abnormally deep. The former condition results when the cyclitic membrane, which originally was concave anteriorly, is stretched into a plane by the process of shrinking; the latter condition develops when the shrinking makes its influence felt more in a direction from before backward. Laterally the cyclitic membrane is bounded by the ciliary processes, behind by the wrinkled retina. Beneath the latter is a transudate which is rich in albumin and often contains blood. A similar liquid is found beneath the chorioid between dissevered lamellæ of the suprachorioid. The chorioid itself is often but little changed. If, however, it has participated to a rather large extent in the inflammatory process, an organized exudate forms upon its surface. Subsequently ossification often occurs in the chorioid or in the exudate lying upon it, so that in atrophic eyes there are found here sometimes scattered laminæ of bone, sometimes a thin plate of bone of quite large size lining the posterior portion of the eyeball.

360. *Suppuration in the Vitreous in Ectogenous Inflammation.*—In the severe cases, the exudate consists simply of pus and is incapable of undergoing organization.

Here, too, there are different degrees of inflammation according as the suppuration is circumscribed or extends over the whole vitreous cavity. Thus in the case represented in Fig. 179 there is a circumscribed suppuration in the vicinity of a foreign body that had penetrated into the eye, while in the case shown in Fig. 180 the vitreous is completely replaced by pus. If the anterior division of the eye is still so transparent that a look into the deeper parts is possible, we can in making our clinical examination recognize a yellow reflex behind the lens (*"abscess of the vitreous"*). This afterward becomes enveloped in a connective-tissue membrane which is connected with the ciliary process and the retina, so that these structures are drawn more and more into the interior of the eye as this membrane progressively shrinks. In these cases too the outcome is as a rule atrophy of the eyeball.

The most severe cases in their course are those in which suppuration invades the sclera. This occurrence is clinically recognizable from the fact that by the inflammation of the sclera an inflammatory œdema of the adjacent cellular tissue of the orbit is produced which causes exophthalmus. Ultimately the pus-soaked sclera disintegrates altogether at one spot and the suppurating contents of the eyeball are discharged exteriorly (*panophthalmitis*, § 423). The eyeball then rapidly shrinks until nothing is left of it but a small stump (*phthisis bulbi*). Phthisis is to be distinguished from atrophy, or the gradual diminution in size of the eyeball occurring after irido-cyclitis. In atrophy the diminution in size keeps within moderate bounds, while in phthisis the eyeball may be reduced to the size of a hazel nut or less. An atrophic eyeball is often the site of repeated inflammation and may also give rise to sympathetic disease of the other eye; a phthisical eyeball generally remains quiet and entails no danger for the

other eye. Hence an atrophic eyeball ought generally to be enucleated, while a phthisical stump can usually be left.

Both in atrophy and in phthisis the optic nerve becomes subsequently altogether atrophic, so that finally it forms a thin strand consisting simply of connective tissue. This takes place in obedience to the general law that nerve trunks atrophy when their terminal expansions are destroyed (ascending atrophy).

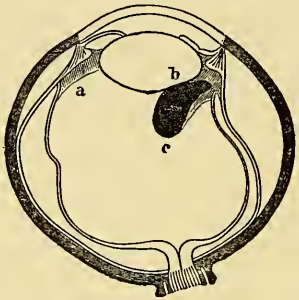


Fig. 179.

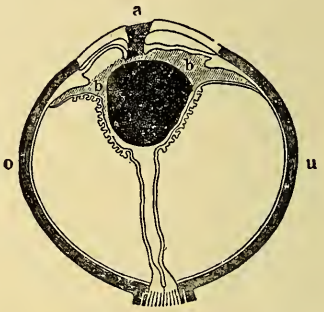


Fig. 180.

Fig. 179.—Abscess of the Vitreous. Horizontal Section. A perforating injury had been produced by a piece of iron in the upper and inner quadrant of the sclera. When the patient presented himself five days afterward there was irido-cyclitis with hypopyon and a gray reflex behind the lens, and the tension of the eye was somewhat elevated. In the course of the treatment the irido-cyclitis improved, but the deep reflex increased and became yellow. The tension of the eye sank below the normal and the perception of light was abolished. On this account the eye was enucleated five weeks after the injury. The site of the wound in the sclera is not included in the section here represented. As a result of the iritis there is on the temporal side an adhesion of the pupillary margin to the lens capsule, with a hump-like protrusion of the iris. On the nasal side the iris is adherent over its entire posterior surface to the lens capsule and to the exudation membrane which fills the posterior chamber. Hence it does not protrude. A cyclitic membrane, *a*, is also visible on the temporal side (really it runs all the way round, lying on the ciliary body), and on the temporal side it connects the ciliary body with the margin of the lens. By the traction produced by this membrane the ciliary body and in part also the chorioid have been detached from the sclera; on the nasal side, the detachment of the chorioid extends particularly far backwards. There is, furthermore, a detachment of the retina extending from the ora serrata to the papilla. The latter on account of its being markedly swollen extends far into the vitreous cavity. The lens is opaque and has a depression at *b* on its posterior surface, because the lens capsule had been injured in this spot by the foreign body and hence the posterior cortex of the lens had been to a certain extent absorbed in this situation. Close to this spot there lies in the vitreous the inspissated mass of pus, *c*. The latter is separated by a delicate membrane from the adjacent vitreous which is scarcely infiltrated at all. It is this abscess of the vitreous that gave the deep yellow reflex.

Fig. 180.—Abscess of the Vitreous. Vertical Section. The patient had suffered a rupture of the cornea from the thrust of a cow's horn. The rupture ran through the middle of the cornea. Inflammation set in and the eye became softer, lost its perception of light, and was, therefore, enucleated one month after the injury. The cornea at *a* has an indrawn depression, corresponding to the scar at the site of the rupture. The scar is continuous behind with an exudation membrane, *b b*. This membrane which is a product of the inflammation of the ciliary processes encloses the remains of the lens and forms a tough diaphragm separating the aqueous chamber from the vitreous cavity. At its upper side, *o*, the iris is adherent by its pupillary margin to the exudation membrane but on its posterior surface it is free and hence is driven forward by the accumulated aqueous. Below, at *u*, the iris is adherent by its whole posterior surface to the exudation membrane. Behind the membrane lies a large abscess of the vitreous which is shut in by the membrane in front, by the retina behind. The retina is everywhere detached and where it bounds the abscess is thrown into many folds. The chorioid remains attached everywhere

361. *Clinical Types of Ectogenous Inflammation.*—The clinical designations for the types of inflammation described above, which, moreover, present all possible combinations and transition forms, are as follows:—

The cases in which the main clinical feature is the inflammation of the anterior division of the eye, and which terminate in organization of the exudate, are known as plastic irido-cyclitis. When the exudate is purulent we speak of a purulent irido-cyclitis or, if the suppuration is in the vitreous cavity, of a purulent irido-chorioiditis. If the suppuration in the vitreous

becomes encapsulated, we call it an abscess of the vitreous or a hyalitis; if the pus breaks through we call the condition panophthalmitis.

These terms are not quite proper if we have regard to the anatomical findings, and it would accord best with the latter if we should call the inflammation of the inner coats of the eyes that is produced by infection an *endophthalmitis septica.*

362. *Changes in Sympathetic Inflammation.*—A special variety of ectogenous infection is that which underlies *sympathetic inflammation* (page 429). While in the ordinary cases of infection the exudation on the free surface of the coats of the eye plays the chief part, here the main thing is an infiltration of the tissue itself, which, moreover, is of a peculiar kind.

In the exciting[4] eye (that is the one which was injured and is the first affected) we find the uvea distended with densely crowded lymphocytes and plasma cells. In most cases there lie in the midst of this uniform infiltration focal collections of large (epithe-

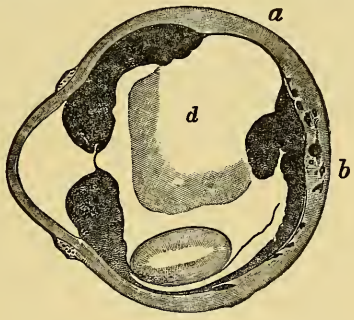


FIG. 181.—INFLAMMATION PRODUCING SYMPATHETIC OPHTHALMIA. Magnified 2 × 1.
A girl twenty years of age had a detachment of the retina, which developed in the right eye without any known cause. The eye gradually became blind but it was not till three years later that it was attacked suddenly by a very severe and painful inflammation, which, after four weeks, went over to the left eye. The latter presented the symptoms of a severe sympathetic irido-cyclitis and also became blind in spite of the immediate enucleation of the right eye. This was one of those rare cases in which a sarcoma, beginning in the chorioid, first produces blindness through detachment of the retina and afterwards becomes necrotic, and thus causes a severe inflammation in the eye. The vertical section of the eye shows the iris greatly thickened by an excessively dense infiltration. The pigment that is visible in it lies partly in the very numerous giant cells. The iris everywhere merges without any sharp line of demarcation into the also thickened ciliary body, and the latter again merges into the chorioid. Of the retina there is nothing to be seen. Below, at *c*, the lens lies in contact with the chorioid. At *a* the chorioid is replaced by a mass, *d*, without distinct structure, which extends thence into the posterior cavity of the eye and fills a large part of the latter. This is the necrotic sarcoma. At *c* the infiltration of the chorioid passes along a vena vorticosa into the sclera; further back at *b*, nodules of the same tissue, but unconnected with it, lie in the sclera. The pupil is closed by a pigmented membrane. The lens is not found in the pupil because it is dislocated into the vitreous.

lioid) cells, which not infrequently have giant cells between them (Fig. 182). Nodules are thus produced which often are like tuberculous nodules. This peculiar infiltration often is present on only a few spots, so that isolated nodules are seen here and there in the iris, ciliary body, or chorioid. In other cases the uvea is occupied by them either largely or wholly and thus often becomes extremely thickened so as to fill, more or less completely, the interior of the eye (Fig. 181). Sometimes the infiltration even makes its way into the sclera, which is permeated with scattered nodules (Fig. 181, *b*), and in this way perforation of the sclera and extra-ocular proliferation may result.

Up to the present time it has not been possible to demonstrate the presence of bacteria in the nodules. Nevertheless there is scarcely a doubt but that here too we

[4] [Dr. Fuchs uses the term "sympathizing," which by English authors is usually applied to the eye that is secondarily affected.—D.]

are dealing with an affection produced by bacteria, which, however, do not cause acute suppuration but, after the analogy of many other bacteria (for instance the tubercle bacilli), cause chronic proliferations. This inflammation has the property of being transmissible to the other eye. Of the sympathizing,[5] that is the secondarily affected eyes, only a few have so far been got for examination, but then generally the same peculiar changes have been found.

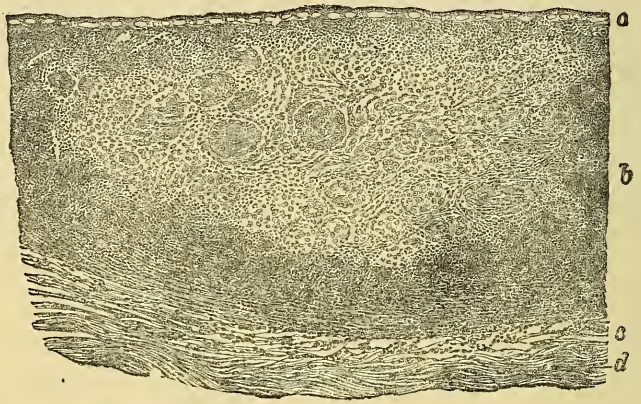


FIG. 182.—INFLAMMATION CAUSING SYMPATHETIC OPHTHALMIA.—DEGENERATED CHORIOID. Magnified 100 × 1.

Beneath the lamina vitrea can be recognized the lumen of the capillaries, *a*. The infiltration of the chorioid, *b*, begins beneath the chorio-capillaris and stops at the suprachorioid, *c*. *d*, innermost lamellæ of the sclera. The chorioid is very densely packed with lymphocytes. In the midst of this infiltration lies a large focus which on account of its lighter color can be seen even with the naked eye. It consists of large pale cells with a large nucleus (epithelioid cells) and of numerous large and small giant cells with the typical an ular arrangement of the nuclei. A few giant cells lie outside of the focus and in the midst of the infiltration of small cells.

363. (2) *Endogenous Infection.*—In the cases of endogenous infection the inflammatory agents act from the blood-vessels. In *metastatic ophthalmia* (§ 424) it is the pyogenic bacteria that so act. The inflammation that they produce is allied both clinically and anatomically to the ordinary form produced by ectogenous infection.

A distinction is found in the fact that in the latter case the inflammatory agents act first on the inner surface of the coats of the eye, while in metastatic inflammation they start from the blood-vessels, into which they have been carried by embolism, and act upon the surrounding tissue. About the embolic spot in the retina or chorioid a focus of inflammation is formed which rapidly leads to the formation of pus and to necrotic disintegration of the tissue. From this primary abscess the inflammation extends with great rapidity so that in the shortest possible time the whole interior of the eye is involved in the suppuration, and the pus soon breaks through to the outside of the eye (panophthalmitis). In exceptional cases it happens that the inflammation remains confined, the abscess which develops in the retina or chorioid becoming encapsulated, and the anterior division of the eye remaining entirely or in large part exempt from the inflammation. There can then be seen even on clinical examination the yellow reflex of the abscess in the depth of the eye. Such cases are found most often in children after infectious diseases (acute exanthemata, epidemic cerebrospinal meningitis); they may give rise to confusion with glioma (pseudo-glioma).

The emboli which produce a metastatic inflammation are as a rule of a capillary

[5] [In the original, "sympathiziertes Auge" ("sympathized eye").—D.]

character, and they affect the vessels of the retina more often than those of the uvea. The bacteria concerned are the streptococcus, which is the most common—more rarely the staphylococcus, pneumococcus, and other bacteria (cf. page 36).

Metastatic ophthalmia is the severest of the inflammations dependent on endogenous infection. Those inflammations of the eye which accompany acute and chronic infectious diseases (cf. § 364 et seq.) are without doubt in part also due to endogenous infection by bacteria, in part perhaps to the action of the toxins which are formed in the body. In any case the inflammations thus produced are not so violent and destructive as those due to infection by the pyogenic germs. The exudation develops more gradually and remains more confined to the tissue itself. The tissue is infiltrated chiefly with uninuclear leucocytes, which tend to accumulate about the blood-vessels under the form of scattered foci. Thus along with the diffuse infiltration there are formed nodular exudates which in the ordinary traumatic inflammations are only exceptionally met with. The nodules are generally so small that they are seen only on histological examination (Fig. 107), but they may be so large that they are visible clinically (papules, tuberculous nodules of the iris). In correspondence with the non-suppurative character of the inflammation is its outcome, which is also less serious. Panophthalmitis never results and even atrophy of the eyeball is a comparatively rare sequel, occurring only in particularly severe or in often recurring cases.

364. Etiology of Iritis and Cyclitis.—Iritis and cyclitis are either primary or secondary in their development. In the first case, the original site of the disease is in the iris or ciliary body itself; in the second case, there is an affection of neighboring parts, which has been transmitted to the iris and the ciliary body (e. g., in iritis with ulcer of the cornea).

We may classify the inflammations of the iris and the ciliary body with reference to their etiology according to the subjoined scheme. In it, as well as in the subsequent description, the expression "iritis" stands, for the sake of brevity, as the representative of iritis, cyclitis, irido-cyclitis, and irido-chorioiditis—that is, for all inflammations which affect predominantly the anterior part of the uvea:

- A. Primary iritis.
  - Iritis in consequence of general diseases.
    1. Iritis syphilitica.
    2. Iritis rheumatica.
    3. Iritis gonorrhoica.
    4. Iritis tuberculosa.
    5. Iritis in acute infectious diseases.
    6. Iritis in disorders of metabolism.
  - Iritis as a local affection.
    1. Iritis traumatica.
    2. Iritis sympathica.
- B. Secondary iritis.

This classification is evidently not an exhaustive one. There are many cases of iritis for which no cause can be discovered and which, therefore, cannot be placed under the classification given above. There is no doubt but that most of these cases, too, although called idiopathic depend on general diseases, especially on anomalies of metabolism, which at present are unknown to us [or on obscure infections]. Of the primary iritides the only ones that are certainly of a local nature are traumatic and sympathetic iritis.

[Iritis, whether primary or secondary, is due, in the great majority of cases, to *micro-organisms* or their toxins. A tentative classification may be made as follows:

A. Infectious iritis, due to the—
1. Spirochæta pallida.
2. Gonococcus.
3. Staphylococcus and streptococcus.
4. Tubercle bacillus.
5. Other organisms.

B. Metabolic iritis, due to—
Diabetes, gout, hypothyroidism.

The so-called rheumatic iritis is apparently due, in nearly all cases, to streptococci or gonococci. Moreover, many, and perhaps the majority of cases of iritis occurring in metabolic disorders, are probably due to micro-organisms or toxins, the altered metabolism simply lowering the resistance so that infection can take place. Traumatic iritis is undoubtedly almost always due to infection.

It must, however, be admitted that, while nearly all cases of iritis are due to infection, the *nature* of the infection often cannot be made out.—D.]

365. (*A*) **Primary Iritis.**—(*a*) *Due to General Diseases.* These are either infectious diseases (syphilis, etc.) or diseases of metabolism (gout, diabetes). In such cases both eyes, although not both at the same time, are often attacked by the iritis. In cases where no general disease is discoverable, a cold is often accused of being the cause. It is certain that in a man predisposed to iritis exposure to cold may start an attack, and recurrences of iritis are particularly apt to be caused by it. On the other hand, it is a question whether exposure to cold can by itself produce iritis.

Primary iritis is found mainly in adults; in childhood primary iritis is rare. Acute iritis (especially the syphilitic, gonorrhœal, and rheumatic forms) more often affects men, chronic iritis more often affects women. The forms of iritis that are based on general diseases are:—

366. (1) *Syphilitic Iritis.*—Syphilis is by far the most frequent cause of iritis. For the most part, it is *acquired* syphilis that we have to do with. The diagnosis of syphilitic iritis is very easy in those cases in which the characteristic formation of nodules (iritis papulosa, Fig. 183) is present. The nodules have a yellowish-red color, are of the size of a pin's head or larger, and are situated either on the ciliary or the pupillary margin of the iris, but never between the two zones in the mid-breadth of the iris. The nodules afterward disappear again by resorption without any purulent disintegration taking place. At the places where they were situated, broad and firm synechiæ are left, and there is often also a circumscribed atrophy of the tissue of the iris. At other times no distinct nodules are found, these being so small that they lie concealed within the tissue of the iris, but several portions of the pupillary margin are greatly swollen, or at all events there are unusually broad synechiæ which do not yield to atropine (*b*, Fig. 171). Finally, in many cases iritis syphilitica presents absolutely no characteristic

marks; the diagnosis, then, can be certainly established only by demonstrating the presence of syphilis, or by the favorable action of antisyphilitic remedies.

Syphilitic iritis generally belongs to the secondary stage of syphilis. It makes its appearance soon after the first eruptions upon the skin (macular or papular), for which reason we may compare the nodules in the iris to papules or to condylomata, and may designate the iritis as iritis papulosa.

Fig. 183.

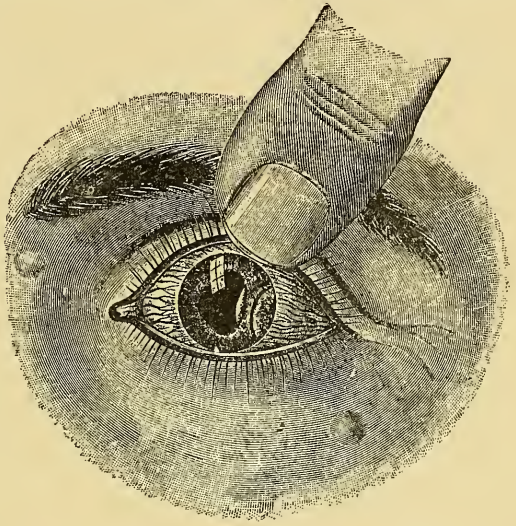


Fig. 184.

Fig. 183.—Iritis Papulosa. Magnified 2 × 1. In a woman 38 years of age the left eye had become inflamed eight days before. The woman was not conscious of having syphilis, and, except for some enlarged lymph glands, showed no symptoms of this disease, but her husband states that seven months previous he had had a hard chancre and soon after this an eruption. The eye is injected, and in the lower part of the cornea there are some minute precipitates. The pupil has dilated but little under atropine; the margin of the pupil in many places is adherent to the lens capsule and is hence indented. In the lower quadrant there is situated in the pupillary zone of the iris the yellowish-red, not very sharply defined papule which juts somewhat into the pupil. The dark spots at the periphery of the pupillary zone are crypts.

Fig. 184.—Syphilitic Tumor of the Ciliary Body. The patient, a man thirty years of age, acquired syphilis four months ago and got an exanthem one month later. The eye has been inflamed for one month. There are universal glandular swellings and a papular exanthem spread over the whole body. Of this some efflorescences are visible in the vicinity of the eye. The eye is markedly injected. The cornea has some precipitates on it. At its temporal side the margin of the pupil is contracted from an arc to a chord, because at this spot the iris is detached from its insertion and pushed towards the center. In the area occupied by this iridodialysis there is exposed to view a yellowish-red, vascular tumor which belongs to the ciliary body and whose light-colored apex is visible in the pupil.

In most cases, the time when the iritis first appears is comprised within the first year after the infection has taken place. More rarely the iritis breaks out in the later stages of syphilis, and is then not associated with the formation of nodules. In exceptional cases, however, nodules do show themselves in this late form also and they must then be regarded as gummata (iritis gummosa).

Syphilitic nodules occur in the ciliary body as well as in the iris. Of the little nodules that come to view on the ciliary border of the iris many certainly start from the ciliary body. Larger nodules in the ciliary body

push the iris away from the cornea-scleral margin and can also be seen behind the pupil (Fig. 184); and they may grow through the sclera, and produce perforation and atrophy of the eyeball.

Iritis also occurs in consequence of *hereditary* syphilis, although far less frequently than after acquired syphilis. The parenchymatous keratitis, which is dependent upon hereditary syphilis, is often associated with iritis. Sometimes it happens that the iritis becomes, comparatively speaking, very conspicuous, while the keratitis attains but a slight degree of prominence; and there may even be iritis without any keratitis whatever. Iritis due to hereditary syphilis is a disease of childhood and youth, while iritis resulting from acquired syphilis is usually observed only in adults.

Syphilitic iritis is very frequently associated with diseases of the posterior section of the eye—that is, with inflammation of the chorioid, retina, and optic nerve. Moreover, it displays a great tendency to recur.

That form of *syphilitic iritis* which is associated with the formation of nodules was formerly designated under the name of iritis gummosa. For it was believed that these nodular exudates were necessarily to be regarded as gummy tumors on account of their sharply circumscribed form, which gives them sometimes just the appearance of small neoplasms. If we agreed to this view we should have to ascribe this form of syphilitic iritis to the tertiary stage of syphilis, which is specially characterized by circumscribed exudates (gummata) resembling new growths. But in so doing we should be contradicting clinical observation, which shows that iritis with the formation of nodules always occurs simultaneously with the symptoms of the secondary stage. We are hence justified in comparing the nodules in the iris with the papules and condylomata, which also belong to this stage, and in designating the iritis as iritis papulosa or condylomatosa (Widder). Another argument against the gummy nature of the nodules in the iris is the fact that they never break down nor suppurate, as gummata are apt to do. True gummata of the iris and ciliary body do exist, however, but they are rare.

The diagnosis of syphilitic iritis will, of course, always have to be based upon the history of the case or upon the demonstration of the presence of syphilis in the patient [particularly by the Wassermann test]. But should we call every iritis which has no characteristic marks syphilitic, simply because it occurs in a syphilitic person? Often, to be sure, we should be right, since syphilis is, in fact, the most frequent cause of iritis; but still a syphilitic patient may also acquire an iritis from any other cause whatever. Among such causes one that requires special consideration is gonorrhœa, which almost all syphilitics have had at some time or other, and which is likewise a frequent cause of iritis. In doubtful cases, the indication in every case is to initiate an antisyphilitic treatment. This will, in most cases, cause rapid inprovement in iritis of syphilitic origin, while other kinds of cases are but little or not at all affected; from which fact a conclusion may be drawn as to the source of the iritis. The effect of antisyphilitic treatment is also of assistance in making the diagnosis in those cases in which we are in doubt as to whether a nodule in the iris is of syphilitic nature or must be regarded as a new formation (sarcoma, tubercle). [It must be noted that even in syphilitic iritis, the disease may be kept up or relapses caused by other infections (e. g., oral infections) (De Schweinitz). Cf. Dunn's theory of the causation of iritis in parenchymatous keratitis (page 297).—D.]

[Varying statistics indicate that from 55 to 70 per cent of all cases of primary iritis

are due to syphilis. Perhaps Jenning and Hill's estimate of 61 per cent is nearest the truth. The proportion must vary, though, in different countries. The significance of syphilis and gonorrhœa as a cause of iritis is proved by the fact that in Palestine, where the former diseases are almost unknown, iritis is rare (Butler).—D.]

Syphilitic iritis also occurs during *intra-uterine life;* the children then come into the world with the remains of it, such as synechiæ, occlusion of the pupil, atrophy of the iris, and even atrophy of the eyeball.

367. (2) *Iritis Rheumatica.*—This occurs in persons who have been ill with articular and muscular rheumatism. It is the form of iritis which is the most apt to recur. From the fact that in many cases recurrences of the iritis coincide with recurrences of rheumatism (attacks of swelling in individual joints), the connection between the two is demonstrated.

[This was formerly regarded as one of the most common types of iritis. Now the increasingly prevalent view is that it is not a morbid entity at all. Many of the cases that formerly would have been classed under this head have been traced to gonococcal infection (see § 368), others to infection by streptococci and similar organisms emanating from some focus about the tooth sockets, in the tonsils, in the accessory nasal sinuses, in the gastro-intestinal tract, etc. These infections are associated with persistent and recurring inflammations of the joints or with symptoms indicative of a general toxæmia (mental and physical depression, tendency to nervous exhaustion, etc.). Complement fixation tests may show the presence of the streptococcus viridens or similar organisms. The iritis may assume a chronic form (§§ 372–374) and it may complicate and prolong an iritis due to other factors (see page 424). Butler suggests that the term rheumatic iritis be replaced by *autotoxæmic iritis.*—D.]

368. (3) *Iritis Gonorrhoica* develops in cases in which gonorrhœa has caused a general infection. This latter runs a course like that of acute articular rheumatism, but generally milder. The knee-joint is regularly the part first attacked by inflammation, which may afterward migrate to the other joints also; associated cardiac complications may even occur. This disease is known as gonorrhœal gout [rheumatism]. Iritis generally sets in after an outbreak of arthritis, but there are cases in which gonorrhœa causes iritis alone and no joint disease. Just as the gonorrhœal joint lesion is very much like articular rheumatism, so gonorrhœal iritis resembles outwardly the rheumatic variety [under which, indeed, have been classed a number of cases really gonorrhœal (see § 367)—D.]. In both—in contrast with other, especially syphilitic iritides—the permanent changes are strikingly slight in comparison with the inflammation, which is often exceedingly violent. Frequently not a single synechia is left. Gonorrhœal, like rheumatic iritis, also affects chiefly men and very often exhibits recurrences, with which is sometimes associated renewal of the urethral discharge or return of swelling in the affected joints.

[Gonorrhœal arthritis may run a course quite different from that above depicted.

Statistics differ as to the frequency of gonorrhœal iritis. According to some authors 8, according to others 20, per cent of all cases of iritis should be classed under this head. Complement fixation tests may reveal the presence of the gonococcus when other evi-

dence is lacking (latent gonococcæmia—Reber). In any event, it is probable that syphilis and gonorrhœa account for at least 75 per cent of all cases of iritis. The significance of these diseases as an etiologic factor is indicated by the fact that in countries where they are rare (Palestine) iritis also is uncommon.—D.]

369. (4) *Iritis Tuberculosa.*—This may appear in the iris under the guise of miliary nodules or of a rather large solitary tubercle. The latter will be considered in § 395. Frequently, however, tuberculous and scrofulous persons, especially the young, suffer from an iritis, which is not accompanied by tuberculous nodules, but still must probably be regarded as tuberculous. This is often marked by the presence of large, lardaceous-looking deposits or by lardaceous exudation masses, which appear to grow out from the sinus of the chamber. It is found in the ages of childhood and youth in scrofulous persons, and also in those suffering simply from anæmia.

370. (5) *Iritis in Acute Infectious Diseases.*—Among these, relapsing fever is the chief one in which iritis forms a frequent complication. It is usually protracted in its course, but ultimately goes on to a cure.

Other infectious diseases which exceptionally give rise to iritis are pneumonia, pertussis; intermittent fever, typhoid, dysentery, influenza, variola, erysipelas, purpura hæmorrhagica, peliosis rheumatica, angina, and mumps. The iritis, moreover, that sometimes accompanies herpes zoster should probably be put down under this head. Some few cases are known in women in whom a transient iritis with hypopyon has recurred regularly with menstruation.

371. (6) *Iritis in Metabolic Disorders.*—This occurs as a result of arthritis urica and arthritis deformans, and very rarely also in diabetes.

[Some cases, too, are undoubtedly due to gastro-intestinal autotoxæmia; others possibly occur in conjunction with hypothyroidism. But as already noted (§ 364), in iritis occurring with disorders of metabolism it is a question whether the connection is a direct or an indirect one. An iritis, for example, occurring with gastro-intestinal disturbance may be due not so much to absorption of the products of perverted metabolism as to the toxins of the infection that produced the metabolic disorder. Again, the iritis occurring with diabetes and hypothyroidism may be due to an infection, to which the system, because of the disorder of metabolism, offers less resistance. This, of course, does not mean that the metabolic disorder is not of considerable significance and that its removal does not exert a favorable effect on the iritis.—D.]

372. We give the name *chronic iritis* to those cases in which we see the products of exudation—the synechiæ, pupillary membrane, deposits, and opacity in the vitreous—develop and gradually increase, while the eye either shows no symptoms of irritation at all, or else there is now and again simply an insignificant injection and some sensitiveness to light. There is always a disturbance of sight, which, indeed, is the thing that attracts the patient's attention and takes him to the physician. Chronic iritis occurs in a light and in a severe form. The *light* form is characterized mainly by the precipitates, occurring either alone or in combination with opacities in the anterior portion of the vitreous. If at the same time no changes of any kind can be

observed in the iris, the disease must be called a sluggish cyclitis (cf. page 407). Often the posterior portion of the uvea is likewise affected, and foci that can be made out with the ophthalmoscope appear in the chorioid. The more the inflammation affects the whole uvea, the more closely this form approximates to the second, severe form, into which it may pass and from which it is not separated by any sharp line of distinction.

The most serious complication of the ordinary cases is the *increase of tension.* We then find the anterior chamber deep (a sign that the increase of tension is caused by a hindrance to the escape of the aqueous) and the pupil somewhat dilated. Often the increase of tension abates spontaneously in a short time, but if it remains for a long time without being attended to, it may cause blinding of the eye. Apart from this the disease usually runs a mild course. It often extends over a period of years, but finally disappears either without leaving any trace of its presence or else leaving a disturbance of sight due to vitreous opacities.

373. In the *severe* form of chronic iritis symptoms of irritation are absent just as they are in the light form, but, on the other hand, the exudation is much more extensive and affects all parts of the uvea, for which reason this form is called *chronic irido-chorioiditis*—an expression in which, obviously, it is implied that the ciliary body, which is situated between the iris and the chorioid, is likewise involved. The patients come on account of the steadily increasing disturbance of vision. Examination of the eye shows posterior synechiæ which slowly increase in numbers until finally seclusion of the pupil is developed. Almost always, too, there is a thin membrane present in the pupil. The iris becomes atrophic early, and afterward, when seclusion of the pupil develops, bulges forward in hump-shaped projections. Hypopyon is never present, but instead there are very frequently fine deposits which point to the implication of the ciliary body, an implication which also manifests itself through the presence of opacities in the vitreous. As these opacities continually increase in number and the vitreous at the same time becomes liquefied, the latter is finally converted into an opaque, mucilaginous liquid. Subsequently opacity of the lens is added, and atrophy of the chorioid and retina sets in. The interference with vision in these eyes is, therefore, always much greater than the optical obstructions in the anterior division of the eye (deposits, membrane in the pupil) would lead one to expect. This disease, which really affects all parts of the eyeball, in most cases terminates in complete blindness. In consequence of the seclusio pupillæ, increase of tension sets in with blindness from excavation of the optic nerve; afterward the eyeball may become ectatic. In other cases, blindness takes place under the guise of a gradually developing atrophy of the eyeball; the eye becomes softer, and the retina undergoes total detachment.

374. Chronic iritis (irido-cyclitis, irido-chorioiditis) almost always attacks both eyes. It runs a very chronic course, and the light form often

passes gradually into the grave form, so that at the outset of the disease one can never say what its duration and outcome will be. It occurs more frequently in female than in male patients. In the case of the young our first thought will be of tuberculosis, our next of hereditary syphilis, and our investigations will be directed accordingly. Some would refer a few cases of chronic iritis to digestive disturbances or to chronic suppuration in the accessory cavities of the nose, in the mouth (from bad teeth), in the tonsils, or in other cavities of the body, and the iritis would then be due either to a toxæmia or to a chronic infection with streptococci. [For the reality and probable frequency of such cases see § 367.] But some of the persons, and especially the elderly women, that suffer from chronic iritis are found, even after quite careful examination, to be perfectly healthy.

375. (*b*) *Iritis as a Local Affection.*

(1) *Iritis Traumatica.*—The causes of this are traumatisms of all kinds, especially if perforation of the eyeball has taken place, and particularly if a foreign body is left in the eye. Operations upon the eyeball are, of course, to be counted among the traumatisms, and of these the most dangerous with regard to iritis and irido-cyclitis are the cataract operations.

The irido-cyclitis that follows traumatism is frequently of the most serious nature, so that atrophy of the eyeball speedily ensues; in contradistinction to iritis due to constitutional causes, which even when the inflammation is a violent one often gets well without leaving any ill results worth mentioning.

The exciting cause of traumatic iritis in cases of the sort just adduced may be of three kinds; either mechanical injury (traction, contusion), chemical irritation (e. g., in the case of contact with swollen portions of the lens or in the case of a cysticercus); or, finally, infection from without. The last-named cause is without doubt the most frequent.

[In traumatic (including post-operative) irido-cyclitis, exacerbation of the symptoms may occur periodically and often at quite regular intervals (every 3, 5, or 7 days) (De Schweinitz).—D.]

376. (2) *Irido-cyclitis Sympathica.*—When an eye is affected with irido-cyclitis in consequence of an injury, either symptoms of irritation or else an actual inflammation may develop in the other eye.

Sympathetic irritation (irritatio sympathica) consists in photophobia, lachrymation, or actual pain. Sometimes also weakness of accommodation is present so that when the patient tries to do fine work the vision gets indistinct and the work has to be given up. Such symptoms, however, are to be called by the name of sympathetic irritation only when objective signs of inflammation are absent, for when once these make their appearance, it is a question, not of sympathetic irritation, but of sympathetic inflammation. A characteristic sign, furthermore, of sympathetic irritation is that it disappears at once and forever when the primarily affected eye is removed.

377. *Sympathetic inflammation* (ophthalmia sympathica) consists in the development of an irido-cyclitis in the second eye. The eye primarily affected is called the "exciting eye,"[6] that which is affected secondarily the "sympathizing eye."[7] Sympathetic inflammation develops sometimes in immediate conjunction with preceding symptoms of sympathetic irritation, sometimes without any intermediary symptoms at all and quite unforeseen.

The beginning is often insidious. In conjunction with an insignificant reddening of the eye, deposits—which are never wanting in the beginning of a sympathetic ophthalmia—appear upon the cornea. Then a few posterior synechiæ and fine opacities in the vitreous develop, and the ophthalmoscope shows hyperæmia of the retina and optic nerve. In favorable cases the disease reaches no higher pitch and at length gets well, leaving either no trace of its presence or a few posterior synechiæ. Unfortunately such mild cases are the exception. The rule is that the symptoms of inflammation slowly or quickly increase; more and more adhesions of the iris develop, and the visual power becomes more and more reduced. Hypopyon, however, ordinarily does nor occur even when the inflammation is severe. Although sometimes intervals in the inflammation with some improvement in sight occur, yet these are not lasting, and finally, owing to the constantly recurring inflammation, there ensue in spite of all treatment the formation of a pupillary membrane and an annular or total posterior synechia. Then, in consequence of the seclusion of the pupil, increase of tension may set in, but this is commonly not of long duration, since cyclitic membranes have also formed in the vitreous, and by their shrinkage the intra-ocular pressure is once more lowered and finally atrophy of the eyeball is produced. As a rule, therefore, the sympathizing eye is lost.

[In view of the great gravity of sympathetic ophthalmia, it is important to know its early danger signs, i. e., those that occur before the stage of actual exudation. According to Brownlie, these are a contraction of the visual field, a spindle-shaped enlargement (elongation of the vertical diameter) of the blind spot, congestion of the optic disk and retinal vessels, loss of visual acuity, paresis of accommodation, and changes in the blood count (marked increase in the large uninuclear leucocytes, some increase in the lymphocytes, decrease in the polymorphous cells). The blood changes may be absent, but when present are regarded as very ominous. Cases that may develop sympathetic ophthalmia should be examined periodically for these symptoms (Brownlie).

For blanching of the eyebrows in sympathetic ophthalmia, see page 406.—D.]

It is supposed that the sympathetic disease may appear not only in the form of an irido-cyclitis, but also under some other guise. The greatest variety of affections have been described as sympathetic. Among non-inflammatory affections, cases of paralysis of accommodation, of amblyopia, and of blepharospasm have been adduced as sympathetic; among inflammatory affections in the posterior division of the eye, neuritis, chorioiditis, and glaucoma; and in the anterior division of the eye, conjunctivitis and keratitis. However, the only conditions whose existence is surely demonstrated are sympathetic neuritis and chorioiditis. The former occurs very rarely and gives a com-

[6] [In German the "sympathizing" eye ("sympathisierendes" Auge).—D.]
[7] [In German the "sympathized" eye ("sympathisiertes" Auge).—D.]

paratively good prognosis. As regards the chorioiditis, this probably is present in all severe cases of sympathetic irido-cyclitis, but cannot be diagnosticated, because the cloudiness of the media prevents ophthalmoscopic examination. Hence we get the ophthalmoscopic picture of chorioiditis only in those comparatively mild cases, in which the anterior segment of the uvea is affected but little or not at all; and such cases are rare. Sympathetic chorioiditis is marked by the presence of numerous small yellow patches which occupy especially the periphery of the fundus. In other cases observers have often gone too far in taking the sympathetic nature of the disease for granted. The fact that an eye has been destroyed through traumatism by no means justifies us in regarding, without further proof, any subsequent disease of the other eye as sympathetic. This assumption should be made only when such disease presents the characteristic clinical picture of sympathetic irido-cyclitis, or when, upon the enucleation of the eye first diseased, the symptoms in the second eye recede too rapidly to be accounted for upon any other assumption than that the affection of the second eye was caused by that of the first. The converse of this inference does not hold good—that is, the fact that enucleation of the first eye does not influence the course of the disease in the second is no argument against the sympathetic nature of the lesion; indeed, it is a well-established fact that when sympathetic ophthalmia has once broken out, enucleation of the eye first diseased is not generally able to cause much change.

378. The affection of the *exciting* eye, which gives rise to a sympathetic inflammation, is always an irido-cyclitis, and is, in fact, almost without exception, an irido-cyclitis traumatica, due to a penetrating injury of the eyeball. In this category, of course, are to be reckoned the operations that are attended with opening of the eyeball, in case they are followed by inflammation.

As a matter of prognosis and treatment, it is important to know that, in spite of there being a violent inflammation of the primarily diseased eye, sympathetic ophthalmia occurs very rarely in (1) suppuration of the cornea (in ulcus serpens, after acute blennorrhœa, etc.) and in its sequelæ, phthisis corneæ or staphyloma of the cornea, and in (2) panophthalmitis and the phthisis bulbi that follows it; and it never occurs in (3) absolute glaucoma.

The *point of time* at which the greatest danger of the transmission of the inflammation exists is when the irido-cyclitis in the injured eye is at its height. Hence sympathetic inflammation makes its appearance, in most cases, from four to eight weeks after the injury to the first eye has taken place. Later than this, when the traumatic irido-cyclitis has subsided and the eye has fallen a prey to atrophy, there need be generally no fear of sympathetic inflammation, so long as the atrophic eye is free from inflammation and is not painful, either spontaneously or to the touch. The danger for the other eye does not develop again until the atrophic eye becomes once more the seat of inflammation and of pain—an event which, to be sure, very frequently occurs. In this way an eye which has been carried for many years in an atrophic state without causing trouble may suddenly become the cause of a sympathetic inflammation. While, therefore, the minimum period for the development of sympathetic irido-cyclitis is a few weeks (the shortest period hitherto observed is ten days), no limits can be set to the

maximum period; sympathetic inflammation has been seen to appear forty years and more after the injury of the first eye. An eye which has been destroyed in consequence of injury is therefore a constant source of danger to the other eye.

To excite sympathetic inflammation it is not necessary for the injured eye to be perfectly blind. Cases occur in which the eye has retained a remnant of visual power after the injury and the irido-cyclitis following it, and has yet given rise to sympathetic inflammation. In that case it may happen that the sympathetically affected eye undergoes complete destruction, while the injured eye is still used to see with.

Can an irido-cyclitis of *non-traumatic origin* be transmitted to the other eye? We very often see irido-cyclitis develop spontaneously first in one eye, then in the other. But we must not therefore at once conclude that the inflammation has been transmitted from one eye to the other. It may be that we have to do with a deeply seated common cause, generally of constitutional nature, which makes itself felt first in one eye, then in the other. We should therefore regard an irido-cyclitis occurring in the fellow eye as sympathetic, only in case the inflammation in the first eye is certainly a purely local one and not dependent on constitutional causes. This is true of two varieties besides the traumatic, i. e., of the irido-cyclitis which sometimes occurs after the perforation of a corneal ulcer, and of that which develops in eyes with an intra-ocular tumor. If the latter becomes necrotic, a violent inflammation of the inner coats of the eye results. In these two cases a genuine sympathetic inflammation of the second eye is observed, even though it is rare.

379. The way in which the inflammation is *transmitted* from one eye to the other is as yet unknown. Not every severe traumatic inflammation of the eye leads to sympathetic disease of the other; whether this develops or not depends upon two conditions:

1. Upon the character of the inflammation of the injured eye. To a certain extent this has been known for a long time; thus we are aware that in cases in which the injury produces ulcus serpens or panophthalmitis, sympathetic inflammation usually does not set in. To excite this an irido-cyclitis is required. But again it is not every traumatic irido-cyclitis, even when destructive, that causes sympathetic inflammation, but only an irido-cyclitis of a quite well defined sort, which is marked by a peculiar, very characteristic anatomical condition (compare page 419). Unfortunately, we have not as yet advanced so far as to be able to conclude from the clinical picture itself whether in a given case we are dealing with this particular sort of irido-cyclitis.

2. If now this particular kind of irido-cyclitis does really develop after an injury, a possibility is then afforded that this inflammation may pass to the other eye, but such passage does not necessarily follow. For it to occur, a series of conditions must be involved, affecting the paths by which the passage takes place. On the discovery of these paths the endeavors of investigators have been mainly expended. Mackenzie was the first who taught us to know sympathetic ophthalmia. He regarded the optic nerves as the paths of transmission. This was the most obvious thing to do, since the optic nerves of the two eyes are connected directly at the chiasm. It was thought that the inflammation travels back little by little from the exciting eye along the optic nerve to the chiasm, thence to the optic nerve of the other side, and then on to the other eye. But then the objection was soon made that in this case the inflammation in the sympathizing eye should necessarily begin with the clinical picture of papillitis, and not

as an irido-cyclitis. Mackenzie's view therefore was abandoned, and it was believed that we should find the path of transmission to be in the ciliary nerves, since these supply the uvea. But since the ciliary nerves of the two sides are nowhere in direct connection, this could not be a case of direct migration but only one of reflex transmission. As a matter of fact it is scarcely to be doubted that sympathetic irritation is effected by way of the ciliary nerves. In fact even in slight changes occurring in one eye (for example a foreign body in the cornea) we observe lachrymation and photophobia in the other. But that an actual inflammation with serious anatomical changes should develop in a reflex way is in our present state of knowledge inadmissible. Hence, we have returned to the optic nerves as the path of transmission. Leber first pointed out that such transmission might be effected by bacteria which, being brought into the injured eye, first produced an inflammation here and then migrated by way of the optic nerves into the other eye. But since a demonstration of the presence of bacteria in the sympathizing eye could not be made, others assume that it was perhaps only the toxins of the bacteria that got from one eye into the other by way of the optic nerves (Bellarminoff).

There are weighty considerations, however, which oppose the idea of the disease being conveyed by the optic nerves. It has already been mentioned that in that case the affection of the sympathizing eye ought to begin under the form of a papillitis, which, as a matter of fact, is not the case. It is true that on ophthalmoscopic examination of sympathizing eyes we can make out slight inflammatory changes in the head of the optic nerve, but no more than in other cases of severe irido-cyclitis. Furthermore, it has been found in the cases in which the sympathizing eye could be examined anatomically that these changes in the optic nerve-head diminished the further back one went, instead of increasing, as necessarily would have been the case if the disease had entered the sympathizing eye from behind. Moreover, an inflammation that makes its way along the optic nerve would probably produce changes at the spot where through the chiasm it passes from one side to the other, that is within the cranial cavity. But nothing of the sort is known. A further argument against the transmission by the optic nerves is afforded by those cases in which sympathetic inflammation sets in, in spite of the fact that the optic nerve on the side of the injured eye was torn or cut through. These and other considerations against the optic-nerve pathway have led to the view that perhaps the transfer of the noxious influence, which presumably consists of bacteria, takes place by means of the blood current in a way analogous to that of metastases (Berlin). We must then of course, make the further assumption that these bacteria are not pathogenic for the other tissues of the body, since sympathetic ophthalmia is not complicated with disease of other organs besides the eye.

Another view is that sympathetic inflammation is an *anaphylactic* disease caused by the disintegration and resorption of uveal tissue [especially the pigment] in the injured eye. So far, however, it has not been possible to adduce proof of the truth of this view, and besides the character of sympathetic inflammation is essentially different from that of the inflammations that are positively known to be anaphylactic.

The reason for the lack of success hitherto in determining with certainty the path of transmission consists in the great difficulties that oppose investigations made with this end in view. So far, we have not been able to demonstrate either by staining within the diseased tissue or by culture methods the presence of the bacteria that are supposed to excite the sympathetic inflammation. As yet we have had no opportunities of examining a sympathizing eye in the first stages of the disease, nor is experimentation on animals of any help to us either. Not only does sympathetic ophthalmia fail to occur spontaneously in animals but the attempt to produce in animals an undoubted sympathetic inflammation experimentally has also failed of success.

[Brownlie and others have pointed out the analogy that sympathetic ophthalmia shows in various ways to the protozoal diseases (syphilis, malaria, trypanosomiasis). This analogy is somewhat supported by the fact that salvarsan exerts a favorable effect on the disease.—D.]

380. (*B*) **Secondary Iritis and Irido-cyclitis** are the varieties which develop through transfer of inflammation from neighboring organs to the iris and ciliary body. Inflammations of the cornea are the lesions that most frequently give rise to it; above all the suppurative keratitides, which are very frequently indeed complicated with iritis. Old scars with incarceration of the iris may through a late infection excite a dangerous irido-cyclitis (page 318). Of the varieties of scleritis, it is the deep form that leads to inflammation of the iris and the ciliary body. The lens may cause iritis, in case of its luxation and also when after the capsule has been opened, swelling masses of lens lie upon the iris. In these cases the iritis is not usually severe.

Of changes in the posterior division of the eye, which may lead to iritis, the chief one to be mentioned is detachment of the retina. When this has lasted a long time, irido-cyclitis very frequently ensues, sometimes insidiously, sometimes with such violence as to cause great pain and ultimately atrophy of the eye. Likewise very severe and terminating in atrophy, is the course run by the irido-cyclitis that sometimes occurs in intra-ocular neoplasms or in intra-ocular cysticercus.

381. Treatment of Iritis and Cyclitis.—In every case of iritis and cyclitis it is incumbent upon us, on the one hand, to combat the local symptoms (*indicatio morbi*); on the other, to remove the causes lying at their foundation (*indicatio causalis*). In those cases in which an etiological factor is not demonstrable, we are thrown back solely upon the symptomatic treatment

382. (1) *Symptomatic Treatment.—Atropine* is the most important remedy in iritis. Inasmuch as it contracts the iris, it necessarily diminishes the amount of blood in its vessels, and hence directly counteracts the hyperæmia. By paralyzing the sphincter it fulfills a second indication which requires every inflamed organ to be put at rest; the constant to-and-fro movement of the pupil is completely arrested by atropine. The third action of atropine consists in its rupturing posterior synechiæ which already exist and in its counteracting the formation of new ones, through the dilatation of the pupil that it causes. The amount of atropine administered must be carefully regulated according to the degree of the intensity of the iritis. During the period of increase of the inflammation it is usually difficult to obtain dilatation of the pupil, because a spasm of the sphincter exists. In this case we must instil atropine several times a day. If this does no good, we place a little granule of atropine in substance in the conjunctival sac (for the necessary precautions, see page 61); this is better

than to instil the solution too frequently, by doing which irritation of the conjunctiva (atropine catarrh) is readily set up. By the simultaneous employment of cocaine the action of atropine may be heightened (page 389). When the inflammation is abating, atropine is instilled just often enough to keep the pupil constantly dilated.

In cases of irido-cyclitis in which the implication of the ciliary body is particularly prominent, and also in cases of pure cyclitis, atropine is not always well borne. For, in proportion as the iris becomes narrower and its vessels can contain less blood, the vessels of the ciliary body are overdistended, since they have to take up the blood which finds no lodgment in the iris. Hence, in such cases, we must be very cautious in the use of atropine, and must suspend it whenever we find that the pain increases after the instillation. So also, when an irido-cyclitis is combined with elevation of tension, the atropine must be stopped and, if necessary, replaced by a miotic.

[Atropine should be used as early as possible in iritis in order to forestall the formation of synechiæ and put the inflamed iris at rest without delay. Hence, whenever the diagnostic application of homatropine (page 397) shows an imperfectly dilating pupil, atropine should be instilled immediately, and the instillations should be repeated until the pupil is thoroughly dilated.

For precautions in the use of atropine and the danger of poisoning see pages 61 and 388.—D.]

Atropine is, in iritis, useless if the pupillary margin is adherent to the capsule throughout, and the iris hence cannot retract.

383. In violent inflammation, *moist warm compresses* or poultices afford the best results, especially for the relief of pain. Cold compresses are generally not well borne, and are suitable only for recent cases of traumatic iritis (see page 56). *Dionin* introduced into the conjunctival sac in powder or in 5-per-cent solution, acts not only to relieve the violent ciliary pain, but also often exerts a favorable influence on the progress of the disease. Violent pain, moreover, is often ameliorated by the *faradaic current* (Reuss, see page 57). Of internal remedies the main ones that act to relieve pain are the *salicyl preparations* (e. g., aspirin).

Free *bloodletting* by means of six to ten leeches applied to the temple, or by a Heurteloup artificial leech, may greatly diminish the inflammatory symptoms in the bad cases; not infrequently, directly after such a bloodletting the pupil for the first time yields to the action of atropine, while before this it had remained in a constant state of spasmodic contraction. If the disease lasts a long time, the bloodletting may, if needed, be repeated once or twice.

One of the most efficient means both of combating the inflammation and of absorbing the exudate is energetic *diaphoresis* (see page 58). For absorbing the exudate we may also try *mercurial* treatment (even in non-

syphilitic cases) or *subconjunctival injections* of salt [or mercury]—these, however, only when there is no marked inflammatory irritation, which might be increased by the injections.

384. The *hygienic regulations* to be observed in iritis require first of all protection from the light, not only on account of the photophobia that is generally present, but also because light excites the pupil to contraction. For the latter reason the protection against light must be applied to both eyes, since with the contraction of the pupil of one eye the pupil of the other also tends to contract. The patient is kept in a moderately darkened room, or is made to wear dark goggles. This is better than a bandage, which it would be difficult to apply to both eyes. The patient should be moderate in eating, and should abstain from spirituous beverages. Care should also be taken to secure bodily rest by the avoidance of all physical exertion and in severe cases by rest in bed. The healthy eye should not be submitted to strain of any kind, i e., reading, etc. Moreover, it is very important to make sure that the bowels of the patient move easily.

385. (2) *Causal Indication.*—[This requires a searching *preliminary examination* to discover the cause or causes of the iritis. In making this examination we should, when the diagnosis is not otherwise certain, determine the condition of the gastro-intestinal tract, take skiagrams of the teeth and the nasal sinuses, use diagnostic injections of tuberculin, and make complement fixation tests of the blood for the spirochæta (Wassermann test), streptococci, gonococci, and other organisms. According to the findings, we apply dietetic and eliminative measures for the relief of gastro-intestinal disturbances, remove foci of infection in the teeth, etc., and if the complement fixation tests show systemic infection give vaccines by injection.—D.]

With regard to the etiological factor, *syphilitic iritis* gives the most favorable prognosis, since it generally yields promptly to an energetic antisyphilitic treatment. The chief requisite here is promptness of action, since we are dealing with a lesion in which a few days may produce great and lasting damage (by forming a seclusio or occlusio pupillæ). Hence salvarsan or mercury is selected, and the latter is best applied by inunction (from 2 to 4 gm. of blue ointment being rubbed in daily) [or by intramuscular injections of an aqueous solution of mercury bichloride or an albolene emulsion of mercury salicylate.—D.] The inunctions [or injections] should be kept up until the diseased eye has become perfectly free from discoloration and then potassium iodide (up to 3 gm. a day) may be used for after-treatment. In iritis due to hereditary syphilis, besides the specific antisyphilitic treatment special value is to be attributed to measures for strengthening the organism as a whole. [In a case of syphilitic iritis we must not forget that there may be other causal factors besides the syphilis that will require treatment (cf. page 424).]

In *iritis rheumatica* sodium salicylate or aspirin is administered. These drugs also do good service sometimes in other forms of iritis, especially in *iritis gonorrhoica*. For the latter, vaccines made of dead gonococci have been used, sometimes with good results. [In so-called rheumatic iritis removal of the probable source of infection (see page 425), followed by appropriate vaccine treatment (if possible with an autogenous vaccine), is indicated. The strength of the vaccine injections and the interval between them are regulated by the opsonic index, the local reaction at the site of injection, and the focal reaction in the eye.—D.]

For *iritis tuberculosa* the main thing is a correct and sufficiently long continued treatment with tuberculin injections (see pages 70 and 446). and, added to this, general hygienic measures for invigorating the patient.

[In iritis due to *metabolic disorders* treatment of the underlying cause (gout, diabetes) must be instituted. In stubborn cases of iritis administration of the thyroid extract sometimes does good service, even though hypothyroidism is only an indirect cause of the iritis (see page 426).—D.]

In *chronic iritis* the main thing to do is to determine the cause. For this purpose it is often necessary to add to a careful internal examination a diagnostic injection of tuberculin, a Wassermann test [and examination for other sources of infection (see page 428).—D.]. We should inaugurate a causal treatment based on the findings thus obtained. Such treatment, however, is often unable to affect materially the course of the disease, so that we have to depend mainly on general hygienic measures and local and symptomatic treatment. [The administration of thyroid extract is sometimes useful.—D.]

In *iritis traumatica* the causal factor is first of all to be eliminated, in case it still continues to act. Foreign bodies are to be removed from the iris; portions of the iris that are much contused or are incarcerated are to be excised; a swelling or luxated lens causing iritis must be removed from the eye. To combat the inflammation of the iris, iced compresses are employed in very recent cases in addition to the atropine. With respect to those traumatic iritides which follow an operation, the most important part belongs to prophylaxis. This consists in strict antisepsis during the operation; and, as a matter of fact, since this has been employed such iritides have become much more rare. [In traumatic (including post-operative) iritis mercury, either by inunction or in the form of calomel, kept up for a number of days, and the salicylates in doses of 3 to 6 gm. a day, are helpful (De Schweinitz).—D.]

386. (3) *Operative procedures* are for the most part indicated more in the sequelæ of iritis than in recent inflammation. *Paracentesis* is performed whenever increase of tension sets in. It may also be tried in cases of long-persisting inflammation which will not yield to other remedies. As the aqueous escapes, the deposits upon the cornea are often washed out

with it, and their escape can be facilitated by rubbing the cornea; but the removal of such deposits is not the proper aim of paracentesis.

Iridectomy is not performed while inflammation still exists, save in very exceptional cases, since, if we perform it then we should have reason to fear that the newly made pupil would be closed up again owing to a continuance of the exudative process. Hence we undertake an iridectomy only when we are compelled to do so because of the development of an increase of tension, or as a last resort in order to put an end to the iritis when all other means have failed. Otherwise we wait until the inflammation has run its course, and then perform an iridectomy, either because a seclusio pupillæ has developed, or as a prophylactic in relapsing iritis to prevent further recurrences. And, in fact, in many cases, an end is put once and for all to recurrences by this means; in other cases, the subsequent recurrences are, at all events, rendered lighter.

In cases of chronic irido-chorioiditis, iridectomy is not only mechanically efficient by removing the seclusio pupillæ, but it also has a favorable influence upon the entire nutritive condition of the eye. The vitreous clears up and the sight inproves, often for quite a long time. If an operation is done upon eyes which have already begun to grow softer—that is, are on the road to atrophy—the eye in favorable cases fills out again, and the ocular tension becomes normal.

387. *Enucleation* in general comes up for consideration only in those cases in which incurable blindness has already set in. It is then indicated: (1) If the eye is permanently inflamed and painful; (2) if we suspect that there is a neoplasm in the inflamed eye (§ 430); (3) in case of sympathetic irritation, this being surely and permanently relieved by the enucleation; (4) if sympathetic inflammation of the other eye threatens to occur. In the last case, enucleation has this great prophylactic value that it prevents with almost absolute certainty the outbreak of sympathetic inflammation. But if we wait to perform enucleation until the first signs of sympathetic disease show themselves, we are generally too late. Hence, to advise enucleation at the right moment is one of the most important tasks that the physician has to perform. To do this properly we must bear in mind the fact that the danger of sympathetic inflammation is almost confined to the traumatic cases. If it is a case of recent inflammation, enucleation is indicated as soon as we see that blinding of the injured eye is inevitable. We recognize that this is the case by the increasing deficiency in the quantitative perception of light (see page 416). If the traumatic inflammation has already run its course, and a greater or less degree of atrophy of the eyeball has supervened, enucleation is still indicated if the eye is sensitive to pressure or if it gets inflamed often. It is only when the eyeball is entirely and permanently free from irritation that enucleation is not imperative. But the patient ought to be warned that he should report immediately for enucleation, if pain or

inflammation happen to set in anew. [He should be kept under periodic observation. Cf. remarks on page 429.]

In phthisis bulbi enucleation is not generally required. Phthisis represents the outcome of panophthalmitis of which, as experience shows, sympathetic inflammation is only very exceptionally a sequel. Moreover, phthisical eyes are usually free from irritation.

When sympathetic ophthalmia has already broken out, the effect of enucleation is uncertain. In the lighter cases it appears to exert a favorable influence upon the course of the sympathetic inflammation; in severe cases, on the contrary, it is often of no avail.

Although *enucleation* generally affords a sure safeguard against sympathetic inflammation of the other eye, nevertheless a series of cases is known in which in spite of enucleation, inflammation has subsequently made its appearance. In every instance it has set in within a short time—from a few days to a few weeks—after the enucleation. The longest interval so far observed is forty-seven days (Shaw), [fifty-three days in a case of Stephenson's cited by De Schweinitz.—D.] Yet even in this case enucleation does not fail to exert a favorable effect, since in the great majority of these cases the sympathetic inflammation runs an unusually favorable course, probably because the removal of the first eye prevents the constant emission from it of new impulses for the production of inflammation.

388. *Sympathetic inflammation* itself is to be treated according to general rules. Salvarsan has been used in several cases with distinct success. Of good effect also is treatment with mercury and with salicyl preparations in the largest possible doses. [Treatment, to be successful, must be persistent and prolonged, and varied according to conditions. The patient, therefore, should be kept under constant observation. Atropine may have to be used for many months at a time. This may be combined with pilocarpine injections and potassium iodide may be combined with the mercury (Burnham).—D.]

Operations generally give a bad result, since they start up the inflammation again, so that the newly formed pupil is once more closed by fresh exudate. Hence operations are done only when it is absolutely requisite (e. g., when done on account of increase of tension); other operations, such as, for example, an iridectomy for optical purposes, are put off as long as possible, preferably for years.

389. Treatment of the Sequelæ of Iritis and Irido-cyclitis.—*Isolated posterior synechiæ* can often be ruptured by the employment of atropine either by itself or in combination with cocaine. Here it is not so much a long continued action as a very energetic one that is required, and this is most certainly secured by placing atropine in substance in the conjunctival sac after preliminary cocainization of the eye. [Some instil a solution of atropine every ten minutes during an hour. Others use strong solutions (4 per cent) and employ adrenaline as well as cocaine to promote absorption

of the atropine. In all such attempts to secure a thoroughgoing effect, the precautions given on page 61 must be observed.—D.]

It is frequently possible to rupture synechiæ which are narrow and drawn out into a point, while broad synechiæ (such as occur after syphilitic or sympathetic iritis) withstand all attempts.

Annular posterior synechia (seclusio pupillæ) demands iridectomy unconditionally, the object being to restore the communication between the anterior and posterior chambers. The operation is often difficult on account of the shallowness of the anterior chamber (due to protrusion of the iris), and also on account of the atrophy of the iris. Accordingly, we must often be contented if we succeed in making a small opening in the iris. In hump-like protrusion of the iris this is best accomplished by transfixion (§ 858). Then the anterior chamber, in consequence of the restoration of the connection between the two chambers, regains its normal depth, so that a iridectomy can be performed later under more favorable conditions.

Total posterior synechia also requires iridectomy, which, however, is often void of result, as on account of the adhesion between the surfaces of the iris and the lens it is frequently impossible to excise a sufficiently large piece of the iris, or because the pigment layer of the iris, which has grown fast to the lens, remains attached to it. In such cases the only thing to be done is to remove the lens, too, even if it is still transparent (Wenzel's extraction, see remarks to § 874). If the lens is shrunken or absent, iridotomy is indicated (see § 858).

In *atrophy of the eyeball* enucleation is indicated subject to the conditions given on page 437. Sometimes enucleation of an atrophic eyeball is desired simply for cosmetic reasons, when the eye is disfiguring and, moreover, does not tolerate the wearing of an artificial eye over it.

If, after an iritis, one or two posterior synechiæ are left which do not rupture when atropine is energetically employed, we abstain from further treatment of them, since they generally do no harm to the eye. At the present time we have entirely desisted from releasing them by operation (corelysis). When, however, an annular synechia is present, we ought not to let it stay, but must make an *iridectomy*. This is also indicated in those cases in which the seclusion of the pupil is not yet indeed complete, but is on the verge of being so, only one small spot of the pupillary margin being free from it. For if we are dealing with a chronic irido-cyclitis, we can count upon this small spot soon becoming adherent too, and in that case it is better not to wait for the seclusion of the pupil to become complete. It is particularly advisable not to do so if the patient lives far from the physician, and might perhaps let the proper moment for the iridectomy pass by.

In seclusio pupillæ the iridectomy is made upward. If occlusio pupillæ is simultaneously present, we should, according to the rules laid down for an optical iridectomy in § 856, make the coloboma to the inner side and below. But even in these cases it is advisable to make the iridectomy upward, because it is so often the case that the lens afterward becomes opaque, and in that case a coloboma situated above will be of advantage in performing the extraction that will be required later.

II. Injuries of the Iris

390. In addition to what has been already said in the previous sections in regard to injuries of the iris and their consequences, the following special varieties of injury, which are most frequently observed after contusions of the eye, may be mentioned:

(1) Under the name of *iridodialysis*[8] is designated the separation of the iris from the ciliary body. We then find on one side, at the ciliary margin of the iris, a black crescent which is formed by the separation of the iris from its insertion at this spot, so that we can there look into the interior of the eye (Fig. 185). When the separation is pretty considerable, the edge of the lens (*l*), the ciliary processes (*p*), and the fibers of the zonula of Zinn stretching between the two can be recognized by means of lateral illumination in the gap that is thus produced. The pupil has lost its round form, owing to the fact that the pupillary margin toward the side of the iridodialysis has shortened so as to occupy the chord of an arc instead of the arc itself (Fig. 185, *a*). The cause of this inward displacement of the pupillary margin lies in the fact that the separated portion of the iris is stretched in a straight line by the contraction of the sphincter. By this means it is withdrawn from its insertion in the ciliary body, and its reunion with the latter is rendered forever impossible. The sight is but little affected by iridodialysis; the only thing being that, if the eye is not accurately focused, monocular diplopia may occur, owing to the formation upon the retina of an image both by means of the pupil and also of the peripheral opening (see § 650).

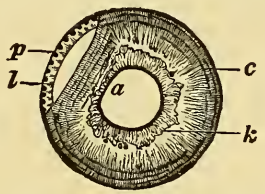


Fig. 185.—Iridodialysis. Magnified 2 × 1.

The pupillary margin at *a* is sloped off so as to form a straight line. At a point corresponding to this spot the iris is separated from the ciliary body and is narrower and somewhat wrinkled. In the interval between the iris and the margin of the cornea are seen the margin of the lens, *l*, and the apices of the ciliary processes, *p;* the fine radiating striation between the two structures represents the zonula of Zinn. *k*, circulus iridis minor; *c*, contraction furrows.

Iridodialysis may occur to any extent, from a scarcely perceptible tear to complete separation of the iris from its ciliary attachment. In the latter case the iris rolls up into a ball which sinks to the bottom of the chamber, and which by the next day has shrunken into an inconspicuous little gray mass. If a rupture of the sclera has been at the same time produced by the injury, the separated iris may be expelled altogether from the eye through the scleral wound. In both cases complete absence of the iris (*irideremia*[9] or *aniridia*[10] *traumatica*) is produced. Again, a partial expulsion of the iris from the eye may occur, especially in cases of rupture of the sclera, and thus a *traumatic coloboma* is produced.

Iridodialysis is sometimes unintentionally produced in operations upon the iris. If the eye that is being operated upon makes a violent movement at the moment when

[8] From ἶρις, iris, and διάλυσις, separation.
[9] From ἶρις and ἐρημία, loneliness, want. [10] From ἀ, privative, and ἶρις.

the operator grasps the iris with the forceps, the iris may by this means be separated from its insertion to a varying extent, or even in part be torn out of the eye. Great hæmorrhage into the anterior chamber is always the result of this unfortunate accident. In iridectomy done on account of occlusion of the pupil, iridodialysis may also be produced in the following way: The operator grasps the iris and tries to draw it out of the wound. To effect this it ought first to be set free from the pupillary membrane. But if the connection between the latter and the iris is very firm, the two do not separate, but the membrane and also the iris of the opposite side follow the course of the traction, so that an iridodialysis is produced upon the side opposite the iridectomy. Hence, in such cases, the iris ought always to be first released from the pupillary membrane by lateral movements of the forceps before it is drawn out of the wound.

Iridodialysis is produced in a non-traumatic way, when neoplasms of the ciliary body grow out into the anterior chamber so as gradually to push the iris away from its insertion (Figs. 184 and 192), and is also produced by traction on the iris (page 410).

391. (2) *Radiating lacerations* which start from the pupil. These may extend to the ciliary margin, so that the pupil at the site of laceration appears to be prolonged to the margin of the cornea in the form of a pointed

Fig. 186.

Fig. 187.

Fig. 186.—Large Radial Lacerations of the Iris. Above lies a large laceration, whose edges gape widely and whose upper extremity, which does not reach to the ciliary border, is rounded off. The smaller laceration, which lies close to the larger one on the inner side of the pupil, retains its original acute-angled shape. The pupil is dilated, and for obvious reasons lacks the black rim of retinal pigment in that part of its circumference occupied by the lacerations.

Fig. 187.—Small Radial Rupture of the Margin of the Pupil. Magnified 2 × 1. At the outside and above, the margin of the pupil is regular and is surrounded by the black line of the retinal pigment. At the inner side and below, this line is wanting; the margin of the pupil here is irregular, shows small indentations, and is retracted along its whole extent so that the pupil is dilated down and in.

Gothic arch (Fig. 186). Such large lacerations, however, are rare. Generally the pupillary margin is simply torn into a little, and the laceration gapes so slightly that it is discovered only upon careful examination, especially with the aid of a magnifying glass (Fig. 187). Such small lacerations are the most frequent cause of the dilatation of the pupil (*mydriasis traumatica*) which develops after contusions and which is founded upon the weakening or paralysis of the sphincter due to the laceration. In such cases, for the most part, a moderate dilatation of the pupil remains permanently.

The ciliary muscle, too, may be paralyzed by a contusion, as manifested by the diminution of the amplitude of accommodation (recession of the near point).

392. (3) *Inversion of the iris* (Ammon) consists in its being turned back so as to lie upon the surface of the ciliary body (Fig. 188, *o*). The iris then cannot be seen occupying its normal place, and looks as though

it were absent altogether. Total inversion of the iris is very rare. Partial dislocation backward is more frequently observed. At the spot where this takes place the iris seems to be wanting, and a coloboma appears to be present just as if an iridectomy had been made.

To *account for* the above-described traumatic changes in the iris, two factors have to be taken into consideration. The first is based upon the flattening which the cornea undergoes through the contusion, and because of which its circumference and consequently also the circle of insertion of the iris become larger. If this enlargement takes place suddenly, the iris cannot adapt itself to it and tears away in places from its insertion, so that iridodialysis is produced (Arlt). The second factor consists in the circumstance that the blow which strikes the cornea and flattens it out also pushes the aqueous backward. The latter tends to recede and pushes against the posterior wall of the anterior chamber, which in the area of the pupil is formed by the lens and in the rest of its extent by the iris. The latter, when pushed backward, finds a support in the lens except at the marginal portion of the iris which lies outside of the margin of the lens. Here the posterior chamber is deepest and is bounded posteriorly only by the feeble zonula of Zinn. The periphery of the iris, therefore, forms the most yielding spot in the posterior wall of the anterior chamber and the one which is the first to give way before the pressure of the receding aqueous. Hence the iris is bulged out backward by the aqueous so as to form a sac extending as far as the zonula, or, if this ruptures extending even into the vitreous (Fig. 189, *u*). The direct consequences of this dislocation of the iris are threefold: marked stretching of the fibers of the iris in a radial direction; dilatation of the pupil; and, finally, in extreme cases, rupture of the zonula. The first factor may result in iridodialysis. The sudden dilatation of the pupil may cause radiating lacerations of the sphincter and consequently paralysis of the latter. The rupture of the zonula causes tremulousness, subluxation, or luxation of the lens. If the backward dislocation of the iris and the consequent recession of its pupillary margin are of a sufficiently high degree to cause the latter to slip back over the edge of the lens, the lens being no longer held in place by the zonula will slip through the pupil into the anterior chamber, where it is then held captive by the iris, which again contracts behind it (luxation of the lens into the anterior chamber). Finally, the sac-like intrusion of the periphery of the iris may be so extensive that the iris at one spot is completely reversed and points straight backward (Fig. 189, *o*) and inversion of the iris is produced (Förster).

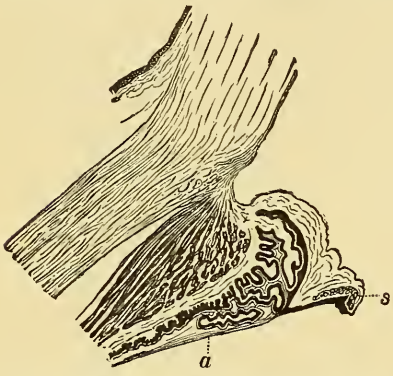


FIG. 188.—INVERSION OF THE IRIS. Magnified 11 × 1.

Fourteen days previous a piece of wood had flown into the patient's eye while he was splitting wood. The eye showed a rupture of the sclera running close to the limbus. The iris had been turned over backward and made to lie on the surface of the ciliary body. It is retained in this position by a slight exudation membrane, *a;* this is attached to a projection of iris, produced by the bending of the pupillary portion of the iris so as to form an acute angle with the ciliary portion. Near the free margin of the iris the pupillary portion makes another turn backward, and so does the sphincter pupillæ, which is seen in cross section at *s*.

A small sharp-edged *hole* may be made in the iris by foreign bodies, particularly fragments of metal, entering the eye. If such a hole is not blocked by blood or exuda-

tion it never grows together but remains unchanged all through life. Upon this fact depends the possibility of effecting a permanent communication between the anterior and posterior chambers in seclusio pupillæ by making a small hole in the iris (transfixion, § 858). For, provided it is not inflamed, the iris has no tendency to cover over raw surfaces by cicatrization as, for example, the cornea does. Thus in the iris stump which is produced by an iridectomy we find, even many years later, the tissue as smoothly divided and as bare as it was directly after the operation (Fig. 112).

After injuries, especially concussions with rupture of the sclera, there is often produced *laceration of the ciliary body* at its attachment or even an entire separation of it from the sclera. This sort of injury is found quite often in anatomical specimens, while clinically it cannot be diagnosticated, because we cannot see the ciliary body in the living eye. By laceration of this sort the anterior chamber is placed in direct communication with the space between the sclera on one hand and the ciliary body and chorioid on the other (perichorioidal space). It thus becomes possible for the aqueous to enter this space and detach the chorioid from the sclera (see § 427).

393. Injuries of the iris are generally accompanied by hæmorrhage into the anterior chamber. The blood which arises from the ruptured vessels of the iris sinks rapidly to the bottom of the chamber (*hyphœma*), and for the most part disappears by resorption within a few days. Then and not before, are we able to investigate completely the damage which the iris has suffered from the injury, and we find perhaps an iridodialysis or radiating lacerations. But often even then it is impossible to discover a solution of continuity in the iris, and so the source of the bleeding remains unknown. In many of these cases the blood is supposed to come from a laceration of Schlemm's canal (Czermak).

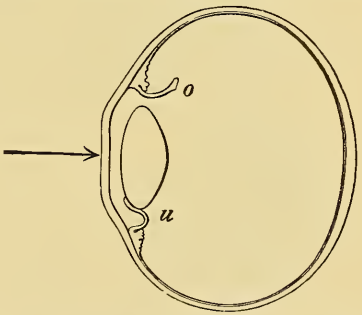


Fig. 189.—Inversion of the Iris. Schematic.

The cornea is flattened, and consequently the aqueous is pushed backward by the force which acts upon the center of the cornea. As a result of this, the iris in its lower part, *u*, bulges out in the form of a sac, but at its upper part, *o*, is completely reversed, so as to point straight backward.

Treatment.—If the symptoms of irritation after the injury are particularly marked, we apply iced compresses for several days; in other cases it is sufficient to keep the injured eye under a bandage and to insure quiet on the part of the patient, secured, if need be, by rest in bed. If an iridodialysis can be made out, we instil atropine in order that the contracting sphincter shall not draw the iris farther away from its point of attachment; in radiating lacerations, however, atropine is contraindicated, because it would make the lacerations gape still more. For the reabsorption of large quantities of blood diaphoretic treatment may be initiated. Iritis is generally not to be apprehended after injuries of the iris not associated with the perforation of the membranes of the eye. With reference to the treatment of perforating injuries, see page 351.

III. Tumors of the Iris and Ciliary Body

394. (1) *Cysts of the Iris.*—Serous cysts occur in the iris. These are filled with a clear substance, and develop within the stroma of the iris, so that their walls are formed by rarefied iris tissue (Fig. 190). Cysts develop after penetrating wounds of the eyeball, and grow very gradually until they reach the posterior surface of the cornea and fill half or more than half of the anterior chamber. Then elevation of tension supervenes, and as a result of this the eye becomes entirely blind. To prevent this, the cysts must be removed in season by an operation. This is performed by making an incision at the margin of the cornea at a point corresponding to the cyst; the forceps is entered through the incision, and the cyst, together with the adjoining iris, is drawn out and excised. Often complete removal is not possible, in which case a recurrence of the cyst is to be expected, which will require a new operation. [Cysts have also been destroyed by electrolysis.—D.]

Serous cysts of the iris are a very rare affection. They appear under the form of grayish, transparent vesicles whose anterior wall usually shows still one or two fibers of rarefied iris tissue, and also some pigment. When they have reached the posterior surface of the cornea, they flatten out upon it, while the cornea at the point of apposition becomes cloudy, as it always does when in contact with foreign tissue. Meanwhile the cyst has already reached the pupillary margin of the iris and pushes it forward into the pupil, so that the latter becomes kidney-shaped, and afterward even reduced to a slit. Moreover, the cyst keeps extending backward, too, and thus causes tilting and afterward opacity of the lens. All these factors give rise to disturbance of vision, which to be sure, is frequently unnoticed by the patient, whose vision has already been impaired by the injury.

Fig. 190.—Cyst of the Iris.

The cyst occupies the inner lower portion of the anterior chamber. It is round, gray, and translucent, and displays on its surface delicate, radiating fibers, which belong to the interior layers of the iris. Its upper and outer margin covers the somewhat distorted pupil, which appears as a dark oval spot showing through the cyst at a point about corresponding to the center of the cornea. Bordering the cyst on the outer side is a vertical, white, linear cicatrix in the cornea, originating from a perforating injury that was caused by a kick from a hoof thirty years before. From all sides the iris is drawn into the cicatrix, and, owing to the tension thus produced, the part of the iris that runs upward shows a partial dehiscence of its fibers.

Cases of congenital serous cysts have been observed; also cases of *pearl cysts*, which are distinguished from the serous cysts by their contents, which are pultaceous, tallowy, or like gruel; in rare cases hairs are also found in them. [Some congenital cysts have been observed which spring from the pars ciliaris retinæ and contain vitreous. They are therefore vitreous chamber cysts, although they project round the edge of the pupil into the anterior chamber and may even become detached and float free in the latter (Spicer).—D.]

Microscopical examination of the cysts has proved that their walls are formed by iris tissue, while their inner surface is lined with epithelium, which secretes the serous contents of the cyst; in the pearl cysts the pultaceous contents are formed by the epithelial cells which are constantly thrown off from the inner surface and undergo fatty disintegration.

We have as yet no certain explanation for the development of cysts except for the most ordinary kind, namely, the traumatic cysts. Normally there are nowhere in the

Iris either glands or epithelium so that retention cysts are not to be thought of. The epithelium must have gotten into the iris from the outside. How this is possible is clear to us when we remember that in the process of healing in wounds of the cornea the epithelium on the surface usually gows rapidly down into the deeper parts (epithelial indipping, Fig. 112). Sometimes it happens that the epithelium extends beyond the

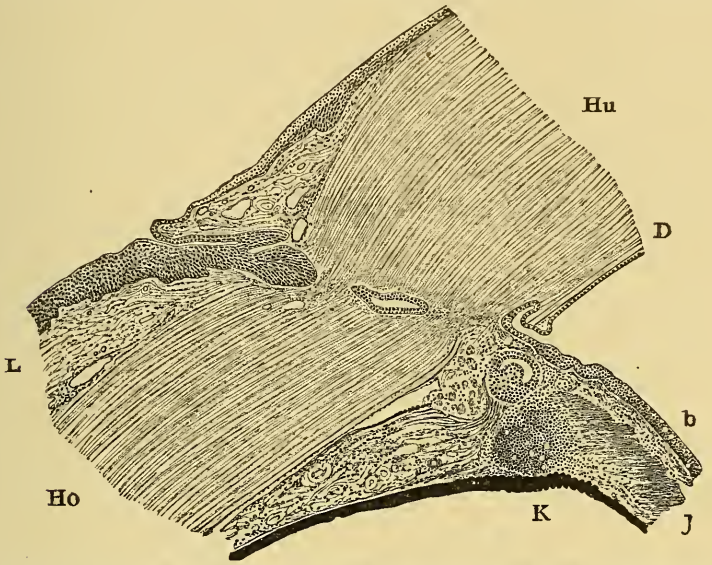


Fig. 191.—Migration of Epithelium into the Interior of the Eye. Magnified 30 × 1.

After an operation by flap incision for a senile cataract a good healing took place at first but after two years increase of tension set in. The eye became blind and had to be enucleated because of the constant pain. The figure shows the site of the scar which lies altogether in the corneal tissue, but, nevertheless, is covered by the limbus, *L*. *Ho* is the upper or peripheral, and *Hu*, the lower or central lip of the wound. The epithelium of the limbus grows over both lips of the wound down into the deeper parts and hence forms a hollow tube whose lumen, however, in many places has disappeared owing to the free surfaces being in apposition. Since the epithelial tube does not extend in a straight line in its passage to the deeper parts the section has not traversed it in its whole length but shows only separate portions of it. Thus we see the epithelial indipping on the surface; again, in the middle of the scar a portion of the epithelial tube; and lastly, far down, the opening of the tube into the anterior chamber at the spot where Descemet's membrane, *D*, was cut through. The epithelium winds over this cut edge and lines the posterior surface of Descemet's membrane in a single layer of cells. On the other side, the epithelium lines the surface of the iris, *J*, forming here a stratum consisting of several layers. The light spots, *b*, in the epithelium are produced by mucous degeneration of individual cells (beaker cells). Since the epithelium lines the anterior and posterior walls of the anterior chamber it forms an *anterior-chamber cyst* (Wintersteiner).

The upper edge of the cut in Descemet's membrane is turned somewhat forward and is separated by scar tissue from the lower edge. This scar tissue, on the one hand, juts somewhat into the wound canal and, on the other hand, it attaches the iris to the posterior end of the wound canal. In the iris we can recognize the muscular fibers of the sphincter pupillæ (at *J*), and behind this a nodular inflammatory infiltration, *K*. Between the latter and the scar tissue lies an islet of epithelium inclosing a small cavity. This epithelium is likewise derived from the epithelial tube which had entered the wound canal, but the connecting part between the latter and the epithelial vesicle has not been traversed by the section and hence this vesicle appears here like an islet of epithelium. The latter constitutes the first beginning of an *iris cyst*. If the eye had not been enucleated the cavity of the vesicle would probably have become progressively larger, so that the vesicle would have penetrated further and further into the tissue of the iris. The iris would thus have been divided into an anterior and a posterior layer of tissue which would have formed the anterior and posterior walls of the cyst. In time perhaps the epithelial tube lying within the corneal scar might have disappeared by a process of atrophy, so that the cyst of the iris would have appeared like an independent epithelial structure.

inner orifice of the wound and into the interior of the eye. In that case it grows on along the walls of the anterior chamber and covers both the posterior surface of the cornea and the anterior surface of the iris (Fig. 191, *D* and *b*). Such a formation of an epithelial lining for the anterior chamber, which may receive the name of an *anterior-chamber cyst*, cannot be diagnosticated clinically, since the epithelium is transparent; but it is

very destructive to the eye as it leads to increase of tension because the epithelial lining hinders filtration through the sinus of the chamber. If the iris abuts on the posterior orifice of the wound the epithelium as it grows into the deeper parts gets into it (Fig. 191); it pushes the layers of the iris further and further apart and becomes developed into an *iris cyst* (Stölting).

Cyst-like structures (*pseudocysts*) may be produced by the fact that portions of the anterior chamber become sacculated by adhesions of the iris to the cornea, and portions of the posterior chamber by adhesions between the iris and the lens—such sacculated portions afterward dilating from accumulation of the liquid contained in them.

Not to be confounded with the cysts of the iris are the *cysticercus vesicles*, which in very rare cases are observed in the anterior chamber. They either lie free in the latter or are attached to the anterior surface of the iris.

395. (2) *Tuberculosis of the Iris.*—This is observed in children and young people. It occurs as disseminated (miliary) tuberculosis and as conglobated (solitary) tubercle—i. e., either in the form of small nodules or as a larger growth resembling a neoplasm. In the milder cases recovery may take place, while in the severe cases the eye is usually lost. The treatment consists, on the one hand, in combating the local symptoms of iritis, and, on the other, in the general treatment of the tuberculosis by hygienic means, internal remedies, and injections of tuberculin. But if the disease, nevertheless, keeps on and blindness is impending, it is better to remove the eye by enucleation, that it may not be the source of a further extension of the tuberculosis.

There is no doubt, furthermore, that many cases of chronic iritis, in which real tubercle nodules are demonstrable neither clinically nor anatomically, are yet attributable to tuberculosis, and can be cured by treatment directed against it (tuberculin injections). It is unknown whether in such cases we have to do with an accumulation of (perhaps weakened) tubercle bacilli in the tissues of the iris or with a simple toxin action.

[In giving tuberculin injections in tuberculous iritis we must be careful to avoid such a dose as will produce a *focal reaction* in the iris, evidenced by ciliary injection, hyperæmia of the iris about the tuberculous foci and grayish deposits in the iris tissue (Gamble).—D.]

Tuberculosis of the iris is a disease that is well understood, as it can be produced experimentally. Cohnheim has shown that tuberculous iritis can be set up by the introduction of tuberculous masses into the anterior chamber. The tuberculous masses (fragments of excised tuberculous lymphatic glands, granulations removed from tuberculous joints by scraping, etc.) must be aseptic—i. e., free from pyogenic germs—as otherwise, upon their inoculation into the anterior chamber, violent irido-cyclitis or even panophthalmitis would be produced, by which the eye would be destroyed. It is safer, therefore, to employ pure cultures of tubercle bacilli for the inoculation. The fragments of tuberculous tissue, when introduced into the anterior chamber, excite there a slight irritation, which, after a few days, disappears again. As the fragments of tissue themselves are also rapidly absorbed, the eye soon appears perfectly normal again, as if the inoculation had remained without result. But in twenty or thirty days afterward the eye begins to become red once more, and the phenomena of iritis make their appearance; at the same time small gray nodules are noticed in the iris. These

increase in number, become confluent, afterward fill the anterior chamber, and finally break their way through to the outside. Generally the animal experimented on afterward perishes from the development of general tuberculosis due to infection starting from the eye. Tuberculous iritis is employed as a means of demonstrating the tuberculous nature of excised pieces of tissue, for which purpose we introduce the latter into the anterior chamber of a rabbit's eye, and see if tuberculous nodules develop after the usual period in the iris.

The tuberculosis of the iris experimentally induced in animals is a primary tuberculosis, but tuberculosis of the iris in man is secondary—i. e., has originated from another tuberculous focus somewhere in the body. As a matter of fact, in most cases of tuberculosis of the iris, signs of tuberculous disease in other organs (lungs, lymphatic glands, bones, etc.) are also found. In many cases, to be sure, the patients appear to have been perfectly healthy up to the time of their eye trouble, but even in these cases a primary tuberculous focus (e. g., caseous bronchial glands), although not demonstrable clinically, must be assumed to exist—in fact, the iris on account of its protected situation, cannot be infected by tubercle bacilli from without, as, for example, the conjunctiva can, in which primary tuberculosis is not so very rare.

Disseminated tuberculosis of the iris occurs in man under the form of an iritis, which presents as its characteristic feature the small, gray, transparent nodules spoken of. These keep changing slowly, some disappearing while others are being newly formed. Recovery may finally take place; not infrequently, however, atrophy of the eyeball occurs in consequence of plastic irido-cyclitis. The disease often develops in both eyes. Removal of the individual nodules through excision by an iridectomy of the portion of iris bearing them is generally useless, since new nodules form afterward in other parts of the iris. [Excision, moreover, is not free from danger, since it opens an avenue for the dissemination of the tuberculosis (Gamble).—D.]

The *solitary tubercle*, as so far observed, is confined to one eye. It either develops simultaneously with the miliary nodules, or, more frequently, without them and without any symptoms of iritis, so that it resembles a neoplasm; indeed, it was at first described as such by Von Graefe, under the name of *granuloma*, because Virchow, who made the anatomical examination of the tumor, described it as granulation tissue. Its subsequent course seems at first to confirm the diagnosis of a neoplasm, since the tumor keeps steadily enlarging, and finally, perforating the cornea near its margin, pushes out in the form of an exuberant growth. But then, instead of a larger tumor developing from this, which keeps growing on indefinitely, the growth breaks down, so that ultimately nothing is left of the eyeball but an atrophic stump. Haab was the first to bring proof of the fact that these tumors, formerly designated as granulomata, are tubercles. As far as treatment is concerned, however, this mistake in diagnosis would be of little significance, since enucleation is indicated alike in the case of a neoplasm and in that of a granuloma. The eye which harbors a granuloma is, in fact, lost for purposes of vision, and may become the source of a general tuberculous infection.

The name of granuloma of the iris has also been bestowed upon granulating prolapses of the iris when they develop into small, mushroom-shaped growths. It is better not to use this designation; it gives rise, on the one hand, to confusion with the tubercles called granuloma, and, on the other hand, to the incorrect assumption that the growth in question is a neoplasm.

Solitary tuberculous tumors have also been observed in the ciliary body.

Nodular Conditions Resembling Tuberculosis.—Besides occurring in tuberculosis, iritis with the formation of nodules also occurs in leuchæmia and pseudoleuchæmia. Under the name of *ophthalmia nodosa* is denoted the affection resembling tuberculosis produced by the entry of caterpillar hairs into the conjunctival sac. Some weeks or months

later nodules develop with violent inflammatory symptoms in the iris and often also in the conjunctiva and cornea. Examination of the excised nodules shows that they contain caterpillar hairs (Pagenstecher and others).

396. (3) *Melanomata.*—These are found under two different forms: The first consists in a blackish tumor which grows out from the stroma of the iris into the anterior chamber and arises from the proliferation of the chromatophores of the iris. The second kind of melanoma has its seat at the pupillary margin of the iris. It develops from the cells of the retinal pigment layer at the spot where it is reflected upon the anterior surface of the iris at the edge of the pupil. Here small blackish-brown nodules develop which project into the pupil.[11] Sometimes, in consequence of the alternating movements of the pupil, they become separated from the pupillary margin and then lie free in the anterior chamber.

A feature common to both kinds of melanoma is that they are benign tumors which reach only a certain size. Nevertheless, cases are known in which pigmented sarcomata have afterward developed from melanomata of the first kind.

397. (4) *Sarcomata.*—Sarcomata of the *iris* are for the most part pigmented, brown tumors, which grow at first slowly, afterward more rapidly, until they fill the anterior chamber, and finally, breaking through the envelopes of the eye, extend their growth exteriorly.

Sarcomata of the *ciliary body* remain for a long time unnoticed, since they are covered by the iris. It is not until they have reached a certain size that they are seen as a brown projection behind the iris, or are recognized by their growing forward into the anterior chamber (Fig. 192). This latter process takes place at the angle of the chamber, where they push the iris away from its insertion (iridodialysis). With regard to their subsequent course, sarcomata of the iris and the ciliary body resemble those which spring from the chorioid, to which reference must be made for particulars (page 475).

The only treatment for these tumors is radical removal, which should be performed as early as possible. Very small sarcomata of the iris can be removed by iridectomy, those parts of the iris which bear the nodules of the tumor being excised. Larger sarcomata of the iris, and also sarcomata of the ciliary body, require the enucleation of the eye without delay.

398. Differential Diagnosis of Iris Tumors.—A *non-pigmented* nodular tumor in the iris may be a syphilitic growth (papule or gumma), a solitary tubercle, an unpigmented sarcoma, or a granulation tumor which has formed about a foreign body imbedded in the iris. The distinguishing marks are as follows:

1. The granulation tumors contain most vessels and are hence usually reddish. The vascularity of the sarcomata varies, but is often also quite considerable; the syphilitic growths have fewer, the tuberculous masses scarcely any vessels passing through them. In the case of the tubercles, small gray tuberculous nodules of characteristic appearance are sometimes found in the neighborhood of the large tumor.

[11] Such pigmentary outgrowths occur normally and greatly developed in the iris of the horse, forming the so-called grape kernels.

2. Papules of the iris are situated only at its pupillary and ciliary margins—never at other spots—while other tumors may take their origin from any point whatever of the surface of the iris.

3. With syphilitic and tuberculous tumors, iritis appears earlier than with sarcomata.

4. Tubercle is found, as a rule, only in people under twenty, while both the other kinds of tumors usually occur after that age.

5. Particular importance must be attached to the general examination of the patient, with the purpose of determining whether there is any evidence pointing to the presence of a foreign body in the iris, or symptoms of syphilis or tuberculosis are found in other organs, and for this purpose the diagnostic injection of tuberculin and the deviation of the complement by Wassermann's test may be employed as an aid. In doubtful cases it is justifiable to initiate an energetic mercurial treatment, from the result of which a conclusion may be drawn as to the nature of the tumor.

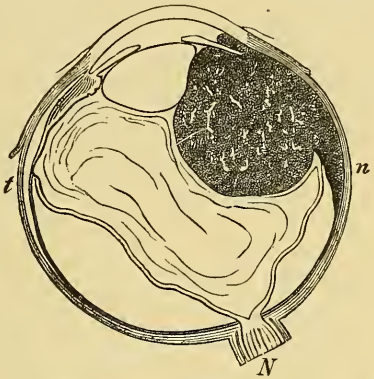


FIG. 192.—MELANO-SARCOMA OF THE CILIARY BODY. HORIZONTAL SECTION THROUGH THE LEFT EYEBALL OF A THIRTY-EIGHT-YEAR-OLD WOMAN. Magnified 2 × 1.

The tumor starts from the nasal side, *n*, of the ciliary body, and the most anterior portions of the chorioid, and forms a hemisphere extending into the interior of the eye. The bright spots and striæ in the tumor represent cross sections of the numerous broad and thin-walled blood-vessels. At its anterior border the new growth, which has perforated the root of the iris, has pushed its way into the anterior chamber, where it was visible in the living eye as a brownish mass filling the sinus of the chamber. The iris is detached from its insertion by the tumor (iridodialysis). Behind the iris the sarcoma bulges forward nearly to the axis of the eye, the nasal portion of the lens having disappeared by erosion, and so given way to the advancing tumor. The lens has consequently suffered but an inconsiderable displacement to the temporal side—enough, nevertheless, to make its edge abut against the apices of the ciliary processes there and flatten them out somewhat. The lens is transparent, and the lens capsule uninjured. The retina is adherent to the surface of the new growth, but elsewhere is detached. There was no detachment, however, in the living eye, this condition having been caused by the shrinking of the vitreous in the hardening fluid. Before enucleation the eye had normal tension and a visual acuity of $\frac{1}{75}$.

Among the *pigmented* tumors, pigmented sarcomata and melanomata (of the first variety) resemble each other exceedingly. They can be distinguished with certainty only by determining, from the previous history or from observation, whether a process of growth is going on or not.

As tumors of very rare occurrence may be mentioned: Vascular tumors (Mooren, Schirmer); myomata (Lagrange), myo-sarcomata (De Wecker and Iwanoff, Dreschfeld, Deutschmann), and endotheliomata (Franke, Wintersteiner) springing from the ciliary muscle; epithelial growths resembling adenoma and carcinoma, originating from the cylindrical cells of the pars ciliaris retinæ (Badal, Lagrange, Lawford, etc.); metastatic carcinoma (Uhthoff); hypernephroma (Chana); and, lastly, lepra nodules (Bull and Hansen).

IV. Disorders of Motility of the Iris

399. Disorders of motility of the iris manifest themselves in diminished reaction of the latter, but chiefly in an alteration of the diameter of the pupil. This alteration is particularly striking when the disease affects but one eye, so that a difference in the pupils (*anisocoria*[12]) results. This is always to be regarded as a pathological condition, since in the normal state the two pupils are of the same size under all circumstances [see, however, page 385]. The pathological alterations of the diameter of the pupil comprise dilatation (mydriasis), and contraction (miosis) of the pupil. Either of these conditions may be brought about by spasm (active or spastic processes) or by paralysis (passive or paralytic processes). Spastic mydriasis is produced by active contraction of the dilatator; paralytic mydriasis, on the contrary, by paralysis of the sphincter. The converse is true of miosis; spastic miosis being referable to contraction of the sphincter, and paralytic miosis to paralysis of the dilatator.

400. Mydriasis.—*Spastic* mydriasis accompanies conditions of cerebral irritation of the most widely differing kinds [and irritation of the cervical sympathetic (see page 451). It is produced artificially by cocaine. In spastic mydriasis the pupil usually still reacts (distinction from paralytic mydriasis).—D.]

Paralytic mydriasis is far more frequent. It is caused by paralysis of the fibers of the oculo-motor nerve, whose branches innervate the intrinsic muscles of the eye—i. e., the sphincter pupillæ and the ciliary muscle. These two muscles are hence generally found to be paralyzed simultaneously (ophthalmoplegia interna [or interior]). The oculo-motor paralysis may be a diffused one—i. e., affect several or all of the branches of the nerve—or it may be confined to the pupil (either alone or in conjunction with the muscle of accommodation). Such isolated paralyses occur: 1. In syphilis. 2. In diseases of the central nervous system, and most frequently in tabes and progressive paralysis, which are also connected with syphilis. 3. Through the action of poisons. Among these belong, above all, the alkaloids known as mydriatics. Paralysis of the pupil and of accommodation also occurs in case of poisoning by the toxic principles of putrefaction (rotten meat, fish, sausages, etc.). 4. After diphtheria (cf. § 797).

Paralysis of the pupil and of accommodation, occurring after contusions and in case of increased tension, are accounted for by an entirely local lesion of the sphincter and the muscle of accommodation. In the case of contusions, besides the concussion, small lacerations and extravasations of blood into the muscles are also met with. In the case of increased tension the paralysis is produced by the pressure upon the nerves, with which afterward is associated an actual atrophy of the muscular fibers.

[12] From ἀ, privative: ἴσος, equal, and κόρη, pupil.

The dilatation of the pupil in complete blindness (amaurosis) is not to be regarded as a disorder of motility of the iris, but simply as a physiologic cessation of the pupillary reflex when the perception of light is absent.

[*Irritation of the cervical sympathetic* is characterized by pallor of the face and increased secretion of sweat, dilatation of the pupil, moderate dilatation of the palpebral fissure (from spasm of Müller's muscle), and very slight exophthalmus. It is produced by the instillation of cocaine, and may be caused by irritative lesions in the neck and cervical cord (in syringomyelia and as a prodrome of paralysis of the sympathetic). (Wilbrand and Saenger.)—D.]

401. Miosis.—*Spastic* miosis is observed in beginning meningitis. The greatest degree of spastic miosis is produced by the alkaloids which contract the pupil (miotics); other poisons, such as opium and morphine, chloral, and nicotine, also cause contraction of the pupil, though in less degree.

Paralytic miosis is one of the most important symptoms of paralysis of the cervical sympathetic. Moreover, it very frequently accompanies spinal lesions, especially tabes dorsalis, and also occurs as a spinal symptom in progressive paresis. This *spinal miosis* is very often distinguished by the fact that the pupil has ceased to react to light, while it still contracts synchronously with accommodation and convergence (Argyll-Robertson's symptom; see page 386). [Spinal miosis may be spastic. Paralytic is distinguished from spastic miosis by the fact that in the former the pupil does not and in the latter it usually does dilate under cocaine.—D.]

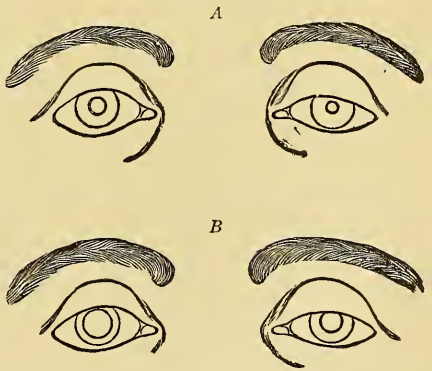


Fig. 193.—Left-sided Sympathetic Paralysis.

A, eyes when turned toward the light, *B*, eyes when turned away from the light. In both cases the left pupil is narrower than the right, but the difference is more pronounced in the second case when the right pupil is widely dilated because of the darkness. The upper lid on the left side hangs somewhat lower (ptosis), than on the right side, and moreover the lower lid rises somewhat higher, as can be made out from the fact that in this eye the distance between the border of the lid and the lower margin of the cornea is smaller. The greater elevation of the lower lid is caused partly by the paralysis of the musculus tarsalis inferior, partly by the enophthalmus, which, however, in this case amounted to no more than a millimetre.

Paralysis of the sympathetic is characterized by a series of symptoms which Horner was the first to describe fully. The pupil is contracted through paralysis of the fibers which dilate it—a fact which is particularly manifested in the non-dilatation of the pupil when the eye is shaded. The difference between the diameter of the pupil in the two eyes is hence more striking in a feeble than in a bright light (Fig. 193). The palpebral fissure is smaller in consequence of drooping of the upper lid. This moderate ptosis is caused by the paralysis of the smooth muscular fibers in the upper lid described by Müller (the musculus tarsalis superior), which are supplied by the sympathetic. The eyeball itself often seems to have sunk back into the orbit and to be less tense. An important symptom is the difference in the fullness of the vessels on the two sides of the face. In a recent paralysis the face is redder and warmer on the paralyzed side; afterward, the oppo-

site is the case, the paralyzed side being paler, cooler, and no longer sweating (a thing easily made out in men by the hat lining, which is stained with sweat on one side and not on the other). The causes of sympathetic paralysis are often the coarser lesions, and most frequently pressure on the nerve from tumors in the neck, such as goiter or enlarged lymphatic glands. More rarely it is caused by traumatisms (among which are fractures of the clavicle) and by operations, e. g., the extirpation of tumors [and sympathectomy]. Furthermore, tuberculous affections of the apex of a lung, lesions of the spinal cord, such as tabes, syringomyelia, or injury of the uppermost part of the cervical cord, and neuritis of the cervical plexus, have been observed as the cause of sympathetic paralysis. In most cases it is impossible to find a cause. The paralysis, in addition to the inconsiderable disfigurement due to the slight ptosis, causes no annoyance, and not infrequently is first discovered accidentally by the physician. It is commonly incurable.

402. Enlargement and diminution of the pupil in themselves cause no considerable interference with vision if they are not combined with paralysis of accommodation. Hence they are but seldom, in themselves, the subject of treatment; their chief significance lies in their being an important symptom of a deeply seated and widespread disorder. This latter, therefore, as a rule, is alone the object of treatment. Paralytic mydriasis may be treated symptomatically by miotics and electricity.

If the inequality of the pupils is slight and neither pupil shows any great variations from the average pupillary width, we may be in doubt as to which of the two pupils is to be regarded as the abnormal one. If for example the right pupil is somewhat larger than the left, it is a question whether a mydriasis of the right eye or a miosis of the left is present. The distinction is to be made by carefully testing the reaction of each pupil, this being done by alternately shading and illuminating both eyes at once. The pupil which shows the less marked reaction of the two is the diseased one.

Under the name of *hippus* is designated a pathological condition which consists in a constant and rapid change in the diameter of the pupil. Since even under physiological conditions the pupil never remains quite at rest, it is hard to draw the line between physiological and pathological inquietude of the pupil, and many believe that a genuine hippus does not exist. [A periodically alternating (*cyclic*) *contraction and dilatation* of the pupil has been noted (sometimes in conjunction with a simultaneous alternating dilatation and contraction of the palpebral fissure) in cases of oculo-motor paralysis (Von Hippel, Coats). The miosis seems to be spastic and occurs in association with contraction of the internal rectus in the same eye.—D.]

For disorders of motility of the ciliary body, see under the anomalies of accommodation (§§ 795 et seq.).

V. Congenital Anomalies of the Iris

403. (1) *Membrana Pupillaris Perseverans* [persistent pupillary membrane].—This consists of a gray or brown tissue which lies upon the anterior capsule of the lens in the region of the pupil, and is usually connected with the iris by brown filaments. Very frequently, however, there are simply a few brown dots present on the lens capsule or only one or two filaments that run from one portion of the pupillary margin to the opposite, and thus bridge over the pupil, or that pass from the iris to the capsule of the lens. They

bear a great resemblance to the posterior synechiæ remaining after iritis, but do not, like the latter, rise from the pupillary margin itself, but outside of it, from the circulus iridis minor, which lies on the anterior surface of the iris (Fig. 194, *c*), and which, as embryology shows, gives off the vessels for the pupillary membrane.

Persistent pupillary membrane is of comparatively frequent occurrence in newborn infants, but afterward disappears, except in a few cases in which remnants of it persist during the whole life. The brown filaments stretching from the pupil to the capsule of the lens are blood-vessels which have been obliterated and enveloped in pigment. Where they are attached to the lens capsule, the latter frequently shows a punctate, densely white opacity (Fig. 194, *c*). These brown filaments do not hinder the free movement of the pupil. Moreover, under atropine the pupil dilates ad maximum without suffering any change in its roundness, because the filaments are so extremely extensible. This is another mark distinguishing them from the acquired synechiæ due to inflammation.

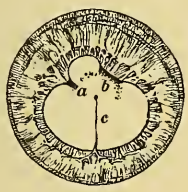


FIG. 194.

FIG. 195.

FIG. 194.—REMAINS OF THE PUPILLARY MEMBRANE. This rises under the form of a small filament, *c*, from the circulus iridis minor, and runs to the pupil, in the center of which it becomes attached to a small, round, white capsular opacity. In spite of the filament, the lower half of the iris has retracted widely under atropine, so that the filament is greatly elongated; but the upper part of the iris is prevented by two posterior synechiæ (*a* and *b*) from submitting readily to the action of atropine. (See explanation to Fig. 171.)

FIG. 195.—CONGENITAL COLOBOMA OF THE IRIS. Enlarged 2 × 1. The pupil as a whole is displaced downward, so that its upper border lies approximately behind the center of the cornea. It is continued below into the coloboma, and is hence pear-shaped. The circulus minor grows more and more narrow below, so that at the lowest part of the coloboma it is no longer visible; on the other hand, the black fringe belonging to the retinal pigment of the iris is here proportionately broader. The contraction furrows of the iris are present only in its upper part.

404. (2) *Coloboma*[13] *Iridis.*—Congenital coloboma of the iris is always situated below. The pupil is continued downward to the margin of the cornea, growing narrower all the time, so that it has the shape of a pear with its small end at the lower margin of the cornea (Fig. 195). The sphincter encompasses the pupil and the coloboma, too, as far as the apex of the latter. Congenital coloboma is thus distinguished from the artificial one made by iridectomy. In the latter the sphincter is wanting in the course of the coloboma, because it has been excised; it is seen to end with sharp edges at the dividing line between pupil and coloboma (cf. Fig. 402). Coloboma of the iris is very frequently combined with coloboma in the chorioid and in the ciliary body (see pages 479 and 482), and sometimes also with a small notching of the edge of the lens (coloboma of the lens) at the spot corresponding to the coloboma in the iris (§ 500).

Congenital coloboma of the iris occurs under different forms. Besides the pear-shaped colobomata above described there are occasionally observed some in which the

[13] From κολόβωμα, mutilation.

pupil has the shape of a keyhole, as in artificial coloboma. A special variety is the bridge coloboma. In this the pupil is separated from the coloboma by a narrow thread of iris tissue, which stretches like a bridge from one pillar of the coloboma to the other. Incomplete colobomata are of comparatively frequent occurrence; there is then simply a shallow notching of the pupillary margin, or the portion of the iris corresponding to the coloboma is distinguished by a different color, this being generally due to the absence of the anterior layers of the iris at this point.

With coloboma of the iris is often associated a pear-shaped appearance of the cornea, due to narrowing of the latter below.

For the way in which a coloboma develops see page 481.

405. (3) *Irideremia* (*Aniridia*).—The iris may be wanting either altogether or all except a small residual portion. This defect is frequently complicated with congenital opacities in the cornea or in the lens.

406. (4) *Ectopia Pupillæ*.[14]—Even in the normal eye the pupil is not precisely in the center, but is usually placed a little below and to the inner side of it. While usually this can be noticed only upon careful examination, there are cases in which the displacement is so great as to be obvious at once; indeed, the pupil may be situated quite eccentrically, in the neighborhood of the corneal margin. The displacement is oftenest observed to occur upward and outward, and is frequently complicated with a dislocation of the lens (ectopia lentis—Fig. 196).

Fig. 196.—Ectopia Pupillæ et Lentis. Right Eye.

The pupil is pretty wide (5 mm.), and not regularly circular, and is displaced temporally. The broader nasal portion of the iris displays the contraction furrows. The lens is slightly cloudy, smaller than normal, and displaced in a direction opposite to that of the pupil, that is, nasally.

407. [(5) *Ectropion Uveæ*.—As already stated (page 366), the retinal pigment epithelium forming the posterior layer of the iris, turns over the pupillary margin so as to be apparent in front as a narrow, black rim encircling the pupil. Occasionally a sector of it extends further so as to form a black apron on the anterior surface of the iris (Samuels).

This occurs not only as a congenital but also as an *acquired* condition; in the latter case in atrophy of the iris (page 411), glaucoma (see page 502), and other grave diseases of the posterior segment of the eye, especially those caused by traumatism (Stern).—D.]

408. The congenital anomalies above described are found, for the most part, in both eyes. They are apt to be inherited, so that they are frequently found in several members of the same family; they are often also met with in conjunction with other congenital anomalies. For the latter reason the disturbance of sight is often much more considerable than would be expected from the optical conditions. In such eyes there frequently exists very great myopia, hypermetropia, or astigmatism, or deficient development of the retina or even of the entire eye, the latter being considerably smaller than usual (microphthalmus). Such eyes, moreover, are more subject than normal ones to disease—(e. g., to irido-chorioiditis, glaucoma, and cataract).

[14] Also called *corectopia*, from κορή, pupil, ἐκ, out of, and τόπος, place.

CHAPTER VI

DISEASES OF THE CHORIOID

I. Inflammation of the Chorioid

409. Inflammation of the chorioid (chorioiditis), if it remains confined to this membrane, produces neither external evidences of inflammation nor pain. The eye looks normal externally, and the disease manifests itself to the patient only through the disturbance in sight—to the physician, only through ophthalmoscopic examination. But if the disease passes over the anterior portion of the uvea it becomes recognizable exteriorly through the symptoms of cyclitis and iritis (*irido-chorioiditis*).

Chorioiditis is distinguished as non-suppurative or as suppurative, according as the exudates disappear again by resorption or lead to the formation of pus. The ordinary variety of chorioiditis is the non-suppurative form, which is known as chorioiditis exudativa. Non-suppurative irido-chorioiditis generally runs a chronic course, being known as irido-chorioiditis chronica (see page 427). In suppurative chorioiditis it is only in rare cases that the inflammation remains confined to the posterior division of the eye. As a rule, the condition present is a suppurative inflammation of all the membranes lining the interior of the eye—an inflammation, therefore, which, in severe cases, rightly bears the name of panophthalmitis.

A. Chorioiditis Exudativa (Non-Suppurativa)

410. General Characters. Symptoms.—Exudative chorioiditis appears for the most part under the form of isolated foci of inflammation scattered over the chorioid (Figs. 197 and 199). While still recent they appear, when seen with the ophthalmoscope, as yellowish, indistinctly outlined spots which lie beneath the retinal vessels upon the red fundus oculi. The spots are produced by an infiltration of the chorioid with exudate which hides the red of the chorioidal vessels; furthermore, the overlying retina is clouded and gray, and covers the subjacent chorioidal mass as with a faint veil (Fig. 197). In proportion as the exudate disappears by resorption the chorioid again comes into view, but in an altered state; it is atrophic, deprived of its pigment, and in part converted into cicatricial connective tissue. Hence, after the disappearance of the exudate, the diseased spot is seen to grow lighter in color. Where the chorioid has become altogether atrophic, a white spot is formed, because the white sclera then shows through; in other cases remains of the vessels and of the pigment are recognized in the white cicatrix. Afterward the pigment often proliferates, so that the chorioiditic

spots appear lined with black pigment or covered with black spots (Fig. 199). The decolorized or pigmented spots remaining after chorioiditis may properly be characterized as cicatrices of the chorioid.

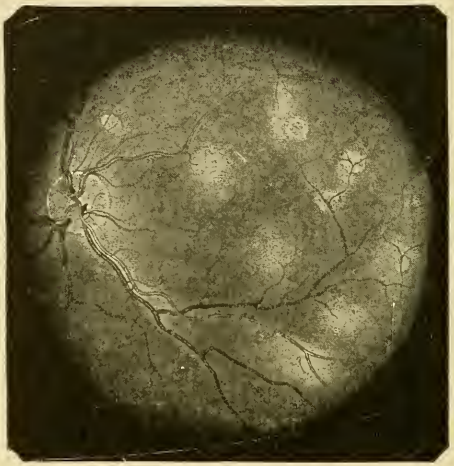
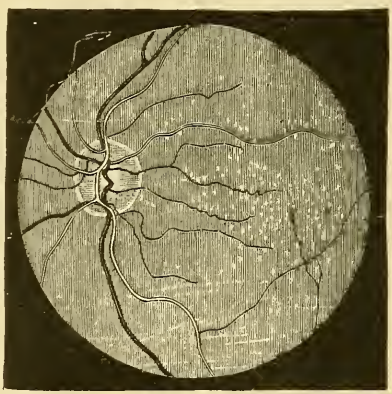


FIG. 197.—RECENT CHORIOIDITIS. (After Oeller.)

A man twenty-six years of age otherwise perfectly healthy had noticed for one or two weeks flashes of light and dark blue spots before the eye. Along the large retinal vessels starting from the papilla there is a whitish œdematous cloudiness of the retina. The chorioiditic spots are arranged irregularly about the macula lutea. The most recent spots are the ones that are most centrally situated; they are grayish-yellow with hazy outlines and without distinct markings. They represent an inflammatory opacity of the retina over the chorioiditic focus which is hence concealed. In the older spots, on the contrary, which are most numerous in the outer upper quadrant, these foci show through the cloudy retina under the form of more sharply outlined disks of about one-fourth the size of the papilla and of a gray color with a lighter center.



[FIG. 198.—SENILE GUTTATE CHORIOIDITIS (Tay and Hutchinson). (After Weeks).—D.]

The distinction between *recent exudates* and *old atrophic spots* in the chorioid is based upon the following signs: Exudates are of a yellow or yellowish-white color, do not have a sharp outline, and present to view no chorioidal vessels; retinal vessels,

which by chance run over them, show by their bending that a projection of the retina exists here, due to the prominence formed by the exudate. The atrophic spots are pure white, and have an irregular but sharp outline, often formed by a pigmented band. Pigment spots also lie in the white spot itself; sometimes, indeed, the growth of pigment becomes so excessive that the spots finally become entirely black. Moreover, remains of the chorioidal vessels are visible within the atrophic area. Such vessels not infrequently present thickened, white-looking walls, or are even quite obliterated and converted into light-colored cords.

In cases of old retino-chorioiditis the pigment often migrates from the chorioid *into the retina*. That the pigment is in the retina is obvious from the fact that the retinal vessels in the spots where this pigment lies are covered by it, whereas they pass over pigment that is situated in the chorioid and are hence not hidden by it.

There are cases in which the atrophy affects only the pigment epithelium, which gradually disappears. Then the stroma of the chorioid, with its vessels and pigmented intervascular spaces, is exposed to view, and there is developed the picture of a *tessellated fundus* resembling that observed as a physiological condition but much clearer cut (Fig. 201). This occurs both in diseases of the chorioid and also in diseases of the outer layers of the retina, to which the pigment epithelium belongs. Besides being found in certain forms of chorioiditis, this change is also found in glaucoma, in myopia of high degree, in retinitis pigmentosa, retinitis syphilitica, etc. In old people, quite small, yellowish white spots, often surrounded by a dark fringe, are sometimes found in the chorioid. They correspond to crystalloid outgrowths of the lamina vitrea of the chorioid, over which the pigment epithelium has undergone destruction. [These are the so-called "*druses*"[1] of the chorioid. Their true origin is probably in the pigment epithelium. They often project far into the retina. They seem rarely in themselves to cause any special impairment of vision, the latter when it occurs being due to degenerative changes independently present. In their typical form druses are discrete, small, whitish, or yellowish spots, unaccompanied by pigmentation or changes in the vessels or the papilla. Sometimes they look like scattered rice grains sown in the red background of the fundus or breaking their way through. Conglomerate and atypical forms occur. They are found especially in the periphery of the fundus and also massed in the maculo-papillary region. In the latter situation they have been called *chorioiditis guttata* (Fig. 198) and *Tay's chorioiditis* (although there is no real chorioiditis present) and *Hutchinson's disease*. The latter includes several cases of central retinal degeneration (see § 528). The white spots in retinitis punctata albescens (see § 527) have been regarded as druses (Leber). Although usually occurring in the aged, druses are found at times in the middle-aged and even in the young.—D.]

411. Discrete Chorioiditis.—Chorioiditis occurring in *isolated foci of inflammation* is distinguished, according to the location of the latter, into different forms:

1. *Chorioiditis centralis* is characterized by changes occurring directly in the region of the macula lutea. It thus causes a central scotoma. The most frequent cause of it is probably myopia, which, if of high degree, leads late in life, almost without exception, to changes in the yellow spot, which are mainly of an atrophic nature (Figs. 201 and 203). Inflammatory changes at this spot are often found in syphilis, in which the region of the macula is sometimes occupied by a large exudate, transformed later into a bluish-gray mass of connective tissue. Circumscribed disease of the macular region may also de-

[1 The German synonym "Druse" has nothing to do with Drüse, gland. Both the English "druse" and its German equivalent are derived from the Bohemian *druza* and denote a cavity in a rock lined with crystals.—D.]

velop as a result of injuries affecting the whole eyeball, such as contusions, the entry of foreign bodies into the vitreous, etc., so that the macula must be regarded as a particularly vulnerable spot in the background of the eye. Finally, there is observed in old people a disease of the macula which usually affects both eyes about equally, and is referable to senile changes. [This causes marked impairment of sight, especially for near work, even when but little change is discoverable by the ophthalmoscope.—D.]

2. *Chorioiditis disseminata* is characterized by numerous round or irregular spots which are scattered over the fundus (Figs. 197, 199). This is an eminently chronic variety, in which, in the course of time, new spots are being constantly formed. Often this takes place discontinuously; i. e., after a long interval suddenly a number of new foci develop, as if the morbific agent had been carried by the blood-current to different parts of the chorioid at the same time.

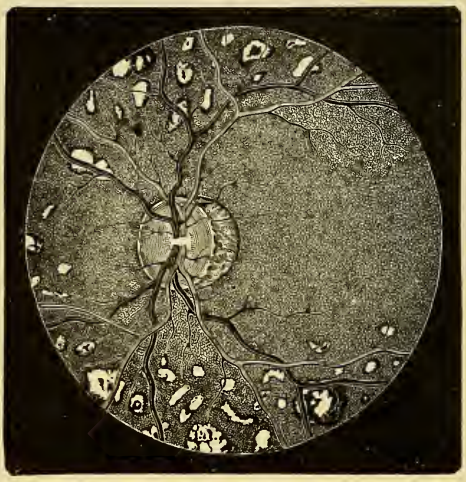


Fig. 199.—Chorioiditis Disseminata. (After De Wecker.)

That the chorioiditis has attacked a myopic eye can be recognized from the atrophic crescent which incloses the papilla upon the temporal side. The crescent is sharply defined by the scleral ring on the side next to the papilla, and by the pigment ring on its temporal side, and shows everywhere remains of the chorioidal vessels and also of pigment. The chorioiditic foci occupy chiefly the equatorial parts of the fundus. They are white, with a lining of pigment of greater or less width; many also have spots of pigment in their interior. The retinal vessels are perfectly distinct as they pass across the spots and their pigment—i. e., they lie in front of the latter.

In old cases, the chorioid is studded all over with spots, which in many places become confluent, so that the fundus looks spotted for a large part of its extent. The sight may, nevertheless, still be pretty good, if only the region of the macula lutea remains intact. In the beginning of the disease hyperæmia of the retina and optic nerve is present, but later both become atrophic. The papilla takes on a dirty grayish-red color and loses its sharp outline (chorioiditic atrophy), the retinal vessels are fewer and are greatly contracted.

A special form of chorioiditis disseminata is the *chorioiditis areolaris*, first described by Förster. In this the first foci develop in the vicinity of the yellow spot, while the subsequent ones make their appearance at a constantly increasing distance from the latter. The most recent foci, therefore, are always those situated at the periphery. The behavior of the individual spots is directly the reverse of the ordinary course pursued by chorioiditic patches; the most recent spots are entirely black, and afterward slowly enlarge, and at the same time become decolorized from the center toward the edge. They look then like black rings inclosing a white center, and at last they get to be almost entirely white.

3. *Chorioiditis anterior* deposits its foci of exudation at the periphery of the chorioid. These foci are therefore readily overlooked if we neglect to examine the most anterior portions of the fundus with the ophthalmoscope. Chorioiditis anterior occurs most frequently in eyes affected with excessive myopia. In young persons, chorioiditis anterior often occurs in consequence of hereditary syphilis; usually the periphery of the fundus is studded with roundish ink-black spots (see page 294). In old people, simple pigmentary changes are frequently found in the anterior portion of the chorioid.

412. Diffuse Chorioiditis.—The variety of chorioiditis which is spread diffusely over the whole chorioid is always combined with a coincident affection of the retina, and is therefore ordinarily known as retino-chorioiditis or chorio-retinitis.

In the typical fashion in which it was first described by Förster this occurs in *syphilis* [*E*, Fig. 255]. In recent cases the retina appears clouded, and, furthermore the entire fundus is veiled by a fine punctate haziness of vitreous; moreover, circumscribed exudates may also be present in the chorioid and the retina. These occupy mainly the region of the macula, and usually appear under the guise of large or small irregular patches, which are of a gray or dirty yellow color and are ill defined, so that they are discovered only upon careful examination with the erect image and with the pupil dilated. In the later stages the cloudiness of the retina disappears, to be replaced by atrophy; at the same time a migration of pigment under the form of numerous black spots takes place into the retina, especially at its periphery, so that there is produced a picture very similar to that of retinitis pigmentosa (see § 526). A similar finding is sometimes had in cases of chronic diseases (either inflammations or new growths) of the liver associated with jaundice. There then ensues in conjunction with the symptoms of hemeralopia a chronic inflammation of the chorioid and retina with pigmentation of the latter (*ophthalmia hepatica*).

413. Chorioidal Changes in Myopia.—Myopia is often complicated with changes in the chorioid, it being a rare exception to find the latter normal in high myopia (Figs. 201 and 203). The changes in the chorioid in this case are, to be sure, less those of a chorioiditis proper than of a primary atrophy caused by the stretching which the chorioid necessarily undergoes, when the entire posterior segment of the sclera bulges backward, as is the case in extreme myopia.

The changes consist in an atrophy of the chorioid close by the papilla and in foci of disease at other spots, particularly in the region of the macula lutea.

(*a*) Atrophy of the chorioid at the border of the optic disk begins at the temporal side of the latter in the form of a narrow light-colored crescent (*distraction crescent*, called conus by Jäger, Fig. 200 A, *ab*). This is accounted for by Stilling as being due to the displacement of the optic nerve-head toward the temporal side. As such crescents often occur in emmetropic eyes, their mode of development has already been considered on page 96. In near-sighted eyes the displacement of the optic nerve occurs much more regularly and to a much greater extent. As the wall of the sclerotico-chorioidal canal is pulled away from the optic nerve on the temporal side, the sclera and chorioid are pulled up over the nerve to a like degree on the nasal side (Fig. 200, *cd*). At the nasal side the optic nerve shows through the sclera and chorioid, and hence with the ophthalmoscope we see along the nasal border of the papilla a somewhat hazy yellowish crescent (*supertraction crescent*—Weiss). With the displacement of the walls of the

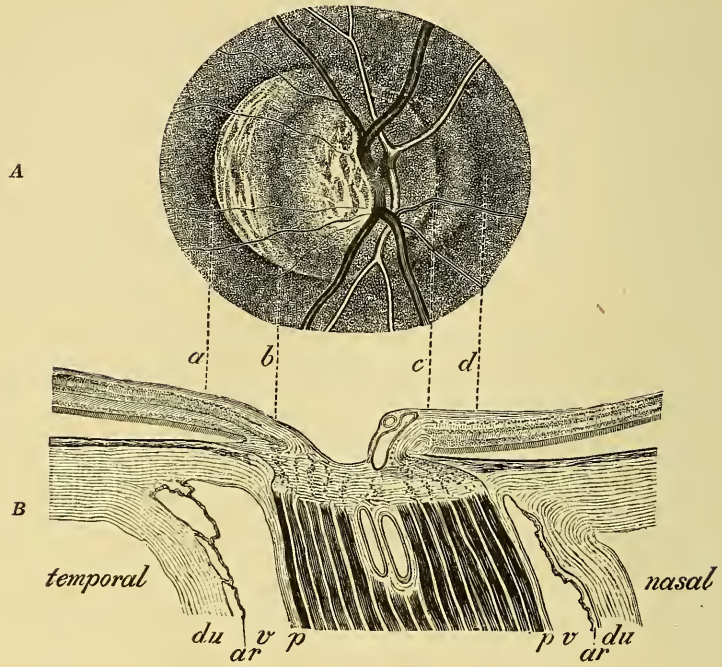


FIG. 200.—OPTIC-NERVE ENTRANCE IN MYOPIA.

A. OPHTHALMOSCOPIC IMAGE OF THE PAPILLA.—The papilla, *b–c*, is of the shape of an ellipse with its long axis vertical. In its outer half it shows the large physiological excavation, upon whose floor are visible the gray stipplings of the lamina cribrosa, while the central vessels ascend on the inner wall of the excavation. Adjoining the outer border of the papilla and not sharply separated from it is the bright crescent, *a–b* (distraction crescent). This is of a white color, while the papilla itself is reddish. The crescent is covered with brownish, elongated markings, representing remains of the stroma pigment of the chorioid. The temporal border of the crescent is sharply defined, and the chorioid adjoining it is somewhat more pigmented than usual. On the other hand, the chorioid in the vicinity of the nasal border of the papilla shows a somewhat lighter coloration in the space between *c* and *d*, so that a yellowish crescent, which, to be sure, is not much more than a suggestion of one, is formed on the nasal side of the disk (supertraction crescent).

B. LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE. Magnified 14 × 1.—The optic-nerve funiculi, wherever they consist of medullated fibers, are colored black by Weigert's hæmatoxylin stain; between them can be seen the septa, which remain unstained, and the longitudinal sections of the central artery and central vein. The black staining ceases abruptly at the lamina cribrosa. In front of the lamina cribrosa the head of the optic nerve presents the physiological excavation. This is a depression whose floor at its deepest part is formed by the lamina cribrosa. The temporal wall of the excavation slopes down quite gradually from the retina. The nasal wall declines abruptly, and shows the cross section of the central vessels. The trunk of the optic nerve is inserted obliquely into the eyeball and also passes obliquely through the coats of the eye; it looks as if these latter had been drawn to the temporal side, and the optic nerve to the nasal side, so that there has occurred a displacement of these two structures with reference to each other—a displacement which is most marked where the optic nerve passes through the sclera and chorioid—i. e., in the scleroticochorioidal canal. Consequently, the temporal wall of the latter is turned somewhat forward, and hence, since the overlying retina is transparent, comes into view when looked at from in front (with the ophthalmoscope), forming a bright crescent extending from *b* to the point *a*, where the pigment epithelium begins. The stroma pigment of the chorioid extends somewhat farther inward than does the pigment epithelium, and is consequently seen under the form of brown spots upon the light-colored crescent. The nasal wall of the scleral canal is turned partly backward, so that it has to pass in front of the most nasally situated portion of the optic nerve, *c–d*. As the displacement affects not only the aperture in the sclera but also the chorioid, the latter is also drawn up over the nasal border of the optic nerve as far as the point, *c*. Since now this nasal portion of the papilla, being covered by the sclera and chorioid, is not distinctly visible with the ophthalmoscope, the papilla appears contracted in its horizontal diameter. Nevertheless, the portion of the optic nerve that is thus concealed glimmers through its covering, so as to be distinguishable under the form of an ill-defined yellowish crescent at the nasal border of the papilla (*c–d* in Fig. A). The displacement of the optic nerve with reference to the sclera is shared in by the sheaths of the nerve. The dural sheath, *du*, and the adjoining arachnoid sheath, *ar*, are separated from the nerve, especially at its temporal side, and the intervaginal space, *v*, *v*, is consequently dilated. On the other hand, the pial sheath, *p*, lies in close apposition to the nerve.

orifice for the optic nerve there is afterward associated atrophy of the adjacent chorioid, which now allows the sclera to show white through it. In this way the white surface on the temporal side of the optic nerve grows constantly in circumference and then extends along the upper and lower borders of the nerve to the nasal side of the latter, until finally the papilla is surrounded on all sides by a white zone (*circumpapillary atrophy;* also called annular staphyloma—Fig. 201). The name staphyloma is properly applied to a bulging of the sclera, but is also applied to the atrophy of the chorioid which is the result of the bulging. The circumpapillary atrophy is generally broadest on the outer side, where it started. In the atrophic area the fundus is either a pure white, if the chorioid there has completely disappeared and the sclera comes into view; or remains of chorioidal tissue, such as vessels and pigment, are still present in it in varying amount.

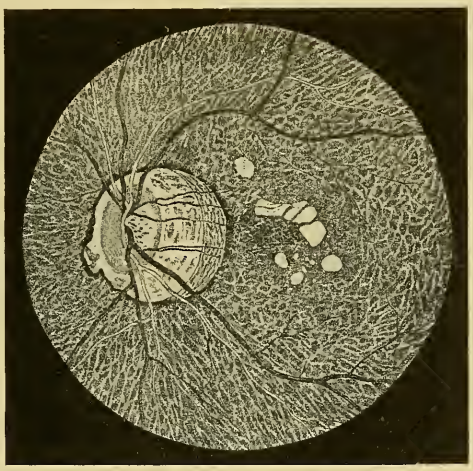


Fig. 201.—Fundus in Myopia of High Degree. (In part after De Wecker.)

The papilla is oblong-oval and has a physiological excavation to the outside of the point of entrance of the retinal vessels. It is surrounded by atrophic chorioid, the staphyloma posticum. This is very broad on the temporal side, and consists there of two divisions; the outer, more pigmented one, showing with especial distinctness the remains of the chorioidal vessels. On the nasal side, the staphyloma is narrower, is lined by a rim of pigment, and is of irregular contour. In one of the outlying portions is noticed a posterior ciliary artery which enters the chorioid from the sclera. The rest of the fundus is tessellated in such a way that the vascular network of the chorioid is clearly recognized. The region of the macula lutea is occupied by chorioiditic changes, consisting partly in an overgrowth of pigment (the small black specks), partly in atrophy (the white patches).

The beginner in ophthalmoscopy regularly falls into the mistake of regarding the atrophic zone that surrounds the papilla as comprised in the latter, and of considering the entire disk-shaped, white surface as a specially large papilla. The boundary between papilla and circumpapillary atrophy, in fact, is not well defined, the papilla being distinguished from the atrophic area mainly by its red color. Indeed, the papilla often appears so red by contrast with the white surface surrounding it, that one might regard it as hyperæmic.

When the atrophic areas are large the papilla displays an elliptical shape, the short axis of the ellipse coinciding with the greatest diameter of the atrophic zone. When, therefore, the latter is broadest outward, as is commonly the case, the papilla forms an upright ellipse (Fig. 201). This change of shape must in part be referred to the fact that the papilla, being pushed toward one side in consequence of the bulging of the sclera, is seen in perspective foreshortening. But it is also due in part to the fact that the sclera and chorioid, which on the outer side have moved away from the border of the papilla, have been drawn up over the border of the latter on its inner side (supertraction crescent).

The retinal vessels that emerge from the papilla are thin and are marked by their straight course—looking as if they had been put on the stretch.

Sometimes two or even three contrasting zones are present in the circumpapillary atrophy which differ from each other in their pigmentation,and often also lie at different levels, and which bear witness to the fact that the development and enlargement of the staphyloma have taken place at different periods (Fig. 201). By proliferation of the pigment, brown or even black crescents are formed.

The line separating the atrophic zone from the healthy chorioid is often sharply defined, especially if formed by a pigmented margin. In other cases a sharp border line is absent, a circumstance which indicates that the staphyloma is in process of growth so that an advance in the myopia is to be apprehended.

FIG. 202.—INFERIOR CRESCENT. ERECT IMAGE.

The bright disk which at first glance might be taken for an enlarged papilla consists of two divisions. The upper, which is darker and of reddish hue, is the papilla proper, which has the form of an irregular oval. Its upper border is semicircular, its lower almost rectilinear, while its two ends are somewhat pointed. The orifice of exit of the vessels lies close to the lower border of the oval, and the vessels as they emerge are at first all directed downward. Hence those that are going to supply the upper half of the retina have to make a sharp bend in order to take the proper direction. Thus the whole arrangement of the vessels on the papilla has a peculiar appearance differing from the normal. The lower division of the light-colored area is formed by the crescent, which is separated by a still brighter rim from the lower border of the papilla. The crescent is unusually large in this case, and, in contradistinction to the reddish papilla, is partly gray, partly white. It is lined by a delicate rim of pigment, and a small patch of pigment also lies close to its temporal border. The fundus shows the pigmentation of a tessellated background.

The size of the atrophic area is, broadly speaking, in direct proportion to the degree of myopia, but variations from this rule very often exist in the individual cases—e.g., great myopia without atrophy of the chorioid, and vice versa. Not infrequently also white crescents and even annular white zones are found in emmetropic and even in hypermetropic eyes.

What, ophthalmoscopically speaking, we call a staphyloma—i. e., the white ring that surrounds the papilla—is indeed caused by the protrusion of the sclera, but is not quite coincident with the latter, being, in fact, less extensive. In many cases of extreme myopia, however, it is also possible to recognize with the ophthalmoscope the limits of the actual *staphyloma in the sclera* under the form of a broad, dark, curved line, which

usually runs in the red fundus along the inner side of the white staphyloma and concentric with the latter. In well-marked cases we can see from the parallactic displacement, or from the bending of the retinal vessels as they pass over the dark curved line, that the latter corresponds to a sudden change of level in the fundus (staphyloma verum). [The line in this case is sometimes called Weiss's line.—D.]

White crescents are also observed at the *lower* border of the papilla (Fig. 202). These resemble in their appearance the acquired crescents due to myopia, but have an altogether different significance. They are congenital and are associated frequently with astigmatism amd almost always with incomplete acuity of vision [which, however, in many cases can be raised nearly or quite to the normal by proper glasses. Congenital and acquired crescents may occur in the same eye.—D.]

With the circumpapillary atrophy of the myopic eye should not be confounded cases of atrophy of the chorioid about the papilla from other causes. Under this latter head belong atrophy after chorioiditis, and also the atrophy in cases of glaucoma, the so-called halo glaucomatosus.

(*b*) The changes of the chorioid in the region of the *macula lutea* make their appearance when the myopia has reached a high degree. Both light-colored and pigmented spots are found (Fig. 201), and not infrequently also white branched lines. The spots gradually enlarge, and finally coalesce to form pretty large atrophic patches, which may even ultimately become united with the staphyloma surrounding the papilla. In such cases of extreme myopia, almost the entire posterior section of the interior of the eye is converted into a great white patch. Moreover, hæmorrhages occur in myopic eyes, and that, too, preferably at the site of the yellow spot. Another, though rare, alteration found in myopic eyes, consists in the formation right in the macula of a coal-black, round spot which gradually grows to about the size of the papilla (Fig. 203). In the subsequent course of this lesion the center of the spot grows continually lighter. It is the changes in the macula lutea that, besides detachment of the retina, represent the greatest danger for the extremely myopic eye. They do not, like detachment, threaten the eye with complete blindness, but they render it unserviceable for any sort of fine work. Besides, they are much more frequent than detachment of the retina, inasmuch as only a few of the excessively myopic attain any considerable age without being affected with these changes.

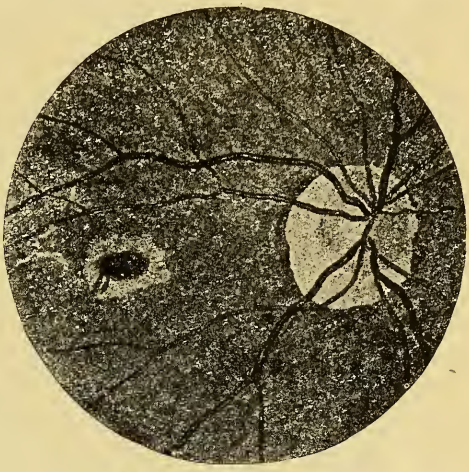


FIG. 203.—THE CENTRAL BLACK SPOT IN MYOPIA. Right eye, erect image.

Fourteen days previously a black spot had appeared before the eye which made reading impossible; the lines of the book seemed bowed. The papilla is surrounded by a zone of atrophic chorioid. At the site of the macula lutea there is a horizontally elliptical spot the marginal portions of which are deep black and the center a dark gray. It is sharply defined and is encompassed by an irregular zone of atrophic chorioid in which, to the outer side and below, lie two small streaky hæmorrhages. The eyeground, being but slightly pigmented, enables one to recognize the chorioidal vessels; between the papilla and macula lutea and out beyond the latter the pigmentation of the eyeground is irregularly maculate. Up and out from the macula lies a group of atrophic white spots, representing the results of stretching of the chorioid.

414. Anatomical Changes in Chorioiditis.—In exudative chorioiditis (Fig 204) there is first a cellular infiltration, which originates mainly from the innermost layers of the chorioid and spreads less toward the outer layers of the chorioid than it does in the direction of the retina. Within these two membranes, the retina and chorioid, the

infiltration is found chiefly along the vessels, which indeed may be said to be sheathed in the cells of the exudate. An exudate, often of considerable size, is deposited between the chorioid and the retina. This exudate is afterward transformed into a connective-

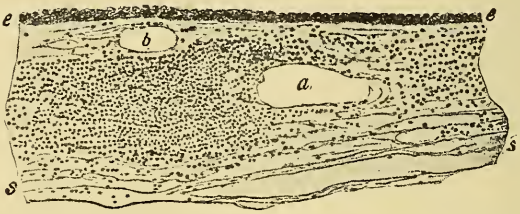


FIG. 204.—RECENT CHORIOIDITIC FOCUS. Magnified 85 × 1.

The cellular infiltration lies mainly in the layer of large vessels to which the vein *a* belongs while the vein *b* lies in the layer of medium sized vessels. The infiltration extends backward so far as to get between the lamellæ of the suprachorioid *s*. The pigment epithelium, *e*, over the focus is preserved so that the latter under the ophthalmoscope must have been indistinct and, because the leucocytes concealed the red chorioidal vessels, must have appeared under the form of a somewhat light-colored and not sharply defined patch.

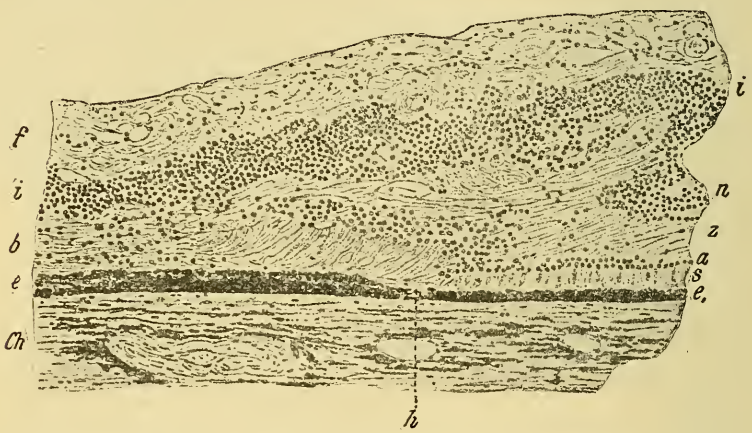


FIG. 205.—OLD FOCUS OF CENTRAL RETINO-CHORIOIDITIS. Magnified 136 × 1.

The inflammation has run its course in the chorioid and the outer layers of the retina in the region of the macula lutea; the figure represents only the edge of the focus which lies to the left of the drawing. In the focus itself the chorioid, *Ch*, is thickened and fibrous, and its vessels are to a large extent obliterated. Upon it lies the pigment epithelium, which at *e* has developed by proliferation into a double layer of cells. This corresponds to a black spot seen with the ophthalmoscope. The retina is adherent to the pigment epithelium and is degenerated. The fiber layer, *f*, is comparatively the best preserved but still it is filled with holes. The layer of ganglion cells has disappeared, the inner granular layer, *i i*, has been irregularly converted, and the layers of the retina that succeed it exteriorly have been entirely converted into a fibrous structure, *b*, composed of a new-formed connective tissue and of glia fibers, in which there still lie a few scattered exterior granules. The adjacent retina is drawn up by the shrinking of this tissue (cicatrix). This fact can be most distinctly made out from the wrinkling which the inner granular, *i*, has experienced because of the traction, with the result that it shows in the section, at *i* and *n*, a kinking which appears Z-shaped in the section. To a smaller extent also this wrinkling is present in the rarefied external granular layer, *a*. Here the wrinkle is situated at the margin of the focus, at *h*; from this point the outer passes forward to the inner granular layer and becomes amalgamated with it. The atrophic intermediate granular layer, *z*, consists of delicate oblong fibers (supporting fibers), which come into view because of the atrophy of the nerve tissue. The layer of rods and cones, *s*, and also the pigment epithelium, e_1, are fairly well preserved up to the edge of the focus, *h*. Here there is an interruption of the pigment epithelium and, from this point on, the rods and cones are obliquely placed and elongated up to the spot where they finally disappear altogether. Owing to their oblique position there was distorted vision (metamorphopsia) at the border of the blind spot or scotoma, that corresponded to the focus itself in which the visual cells had been entirely destroyed.

tissue membrane (cicatrix), binding the chorioid fast to the retina; and in this situation these two membranes themselves become atrophic (Fig. 205). From the chorioid the fine vessels and also the chromatophores in great part disappear; and the vessels that

are left either have their walls thickened and sclerosed, or are obliterated altogether. The retina is converted into a network of connective tissue and glia, in which can be seen the vessels with their walls greatly altered. Wherever the chorioid is adherent to the retina, the layer of rods and cones is absent, and so is the pigment epithelium, except for a few remnants. On the other hand, the pigment epithelium at the border of the scar proliferates; hence the black lining of the chorioiditic patch visible with the ophthalmoscope. In part the proliferating pigment epithelium migrates into the retina, where it is found particularly in the neighborhood of the vessels. The inner surface of the retina is sunken at the site of the cicatrix, and is frequently adherent to the surface of the vitreous (to the hyaloid membrane).

In *irido-chorioiditis chronica* the changes in the chorioid and retina are similar to those just described, with the addition of the evidences of chronic inflammation and afterwards of atrophy in the iris and ciliary body.

The changes in the *chorioid in myopia* consist mainly in atrophy with slight evidences of inflammation. From the stretching of the chorioid results a splitting of the lamina vitrea. This latter gapes and so later on does the underlying tissue of the chorioid. At these points the retina becomes adherent to the chorioid, the rods and cones being at the same time destroyed so that the sight is impaired (Salzmann).

415. Complications.—It is evident that the retina, since it directly adjoins the chorioid, must be sympathetically affected in those spots where the chorioid is diseased. If the implication of the retina is particularly prominent, we speak of retino-chorioiditis. Moreover, the exudates from the chorioid pass not only into the superimposed retina, but also through the latter into the vitreous. Opacities of the vitreous thus produced are hence an almost constant accompaniment of chorioiditis.

416. Symptoms.—It is the implication of the retina and vitreous which causes *disturbances of vision* of various kinds, and thus directs the patient's attention to the eye. The vision is diminished as a whole on account of the cloudiness of the vitreous and the hyperæmia of the retina. But in those spots in which inflammatory foci exist, sight may be entirely abolished, so that insular defects (scotomata) are present in the field of vision (see page 123). Owing to the fact that the retina over the focus of inflammation is pushed forward and its elements are displaced from their normal situation (Fig. 205), objects whose images fall on the retina may appear distorted (metamorphopsia); straight lines, for instance, appear bent in various directions. Frequently also objects appear smaller than they are (micropsia) [or in the atrophic stage look larger than they are (retinal macropsia). See page 126. —D.] As long as the inflammation is recent, symptoms of irritation of the retina manifest themselves; subjective sensations of light (photopsiæ) exist, such as spots before the eyes, sparks and balls of fire, etc. These phenomena cause the patient annoyance and anxiety, sometimes to an extreme degree. When, after the subsidence of the inflammation, atrophy of the chorioid and of the superimposed layers of the retina has taken place, signs of absence of function—i. e., gaps in the field of vision—take the place of the signs of irritation. The influence that the scotomata exert upon the sight in general

depends primarily upon the place they occupy in the fundus. Peripherally situated scotomata cause but little disturbance of vision, even when they are pretty numerous; and if they occur only in one or two places they usually escape the patient's notice altogether. On the other hand, when a scotoma occupies the site of the yellow spot the disturbance of vision is as great as in the preceding case it is trivial; direct vision is then destroyed, and the eye becomes unserviceable for fine work. The first case would be met with in the chorioiditis represented in Figs. 197 and 199, the second in Figs. 201 and 203.

417. Course.—The course of chorioiditis is chronic, it taking many weeks for the foci of exudation to be converted into atrophic spots. The opacities of the vitreous last even longer—often, in fact, permanently. But chorioiditis is chiefly dangerous because of its tendency to recur, in consequence of which new foci of inflammation are constantly developing in the chorioid, so that the latter is finally covered all over with old and recent spots. With this is ultimately associated atrophy of the retina and optic nerve, so that obstinate cases of chorioiditis terminate in partial or total blindness. When the affection of the chorioid is well advanced, clouding of the lens (cataracta complicata) is almost always associated with it.

418. Etiology.—Exudative chorioiditis is a frequent disease, which is observed at all ages. Among its most ordinary causes is syphilis, both acquired and hereditary. As a result of the latter, cases also of congenital chorioiditis have been observed. Chorioiditis may also be caused by general disorders of nutrition of various sorts, such as anæmia, chlorosis, tuberculosis (see page 479), scrofula [gastro-intestinal toxæmia, dental and tonsillar infection], etc. In many cases of chorioiditis the cause remains obscure.

419. Treatment.—The treatment of chorioiditis must regard mainly the *etiological* factor. When, as in syphilis, this is readily amenable to therapy, favorable results are promptly attained—in fact, syphilitic chorioiditis offers the best prognosis, as by energetic antisyphilitic treatment speedy improvement in most cases, and often even an entire cure, can be obtained. If there is evidence that the nature of the chorioiditis is tuberculous, tuberculin injections (see page 70) with invigorating general treatment are advisable. [Moreover, we must look out for other possible causes of chorioiditis, such as metabolic disorders, remote infections, etc., by a searching examination (cf. page 435), and if these are found must seek to remedy them.—D.] To be sure, we are not always able to prevent the frequent recurrences which may still lead ultimately to destruction of sight.

The treatment of the *local* changes should aim to produce rapid resorption of the exudate in the chorioid and the retina and also in the vitreous. Suitable remedies for this purpose are the iodide of potassium or, when necessary, a treatment by inunction, which, even in non-syphilitic cases, can do good service through its absorptive action; furthermore, diaphoresis and subconjunctival injections of salt solution. In marked hyperæmia of the

fundus, bloodletting may be performed by the application of six to ten leeches behind the mastoid process. Besides these measures we must enforce what may be called the hygiene of the eyes—that is, the avoidance of any straining of the eyes, and the protection of them from light by dark glasses, or when necessary, by rest in a darkened room.

B. Chorioiditis Suppurativa, Endophthalmitis

420. The feature which the diseases above named have in common is the purulent exudation into the vitreous. This is the analogue of the accumulation of pus in the anterior chamber, but with the difference that a hypopyon can disappear without entailing any lasting injury, while suppuration in the vitreous leads to permanent and serious changes and generally, in fact, to the destruction of the eye. Like hypopyon, the pus in the vitreous is thrown out by the membranes that line the cavity. The starting point of an inflammation of this sort may be in (1) the *chorioid.* This is the case if pyogenic bacteria have been carried by the blood current into the vessels of the chorioid and lodge there (metastatic inflammation). The inflammation then passes from the purulent foci in the chorioid to the other membranes lining the interior of the eye, and the pus thrown out by these membranes is poured into the vitreous. These cases are rather rare in comparison with the much more frequent instances in which the starting point of the inflammation is (2) the *vitreous* itself. In this case the bacteria are introduced from outside and most frequently by means of a perforating injury. The germs that excite the suppuration then, starting from the vitreous, act by means of the toxins that they produce upon the membranes that line the vitreous cavity—primarily upon the ciliary body and retina and secondarily upon the chorioid. In some cases of perforating injury and also after perforation of a corneal ulcer, the bacteria get into the (3) *aqueous chamber* first and then pass from this into the vitreous, or sometimes it is only their toxins that pass by diffusion from the aqueous into the vitreous chamber. In all cases, however, the purulent exudate that is thrown out by the inner coats of the eye is poured into the vitreous, and hence, although the starting point of the inflammation is different in the different cases, being in the vessels of the chorioid in some, in the vitreous in others, and in the aqueous chamber in other instances still, the ultimate result is the same in all, namely a deposition of a purulent exudate in the vitreous due to inflammation of the inner coats of the eye (*endophthalmitis*). We may distinguish three categories of endophthalmitis:

421. (1) That in which the purulent inflammation remains *confined to the posterior segment of the eye.* Since the posterior membranes of the eye, i. e., the chorioid and retina, have no sensory nerves, even a purulent inflammation of them runs its course without pain and without external evidences of inflammatory reaction. Hence exteriorly the eye appears normal, and we simply see deep in behind the lens the yellow exudate in the vitreous—

abscess of the vitreous (Figs. 179 and 180). Later on, this is transformed into a membrane, by the shrinking of which the retina is detached and the eyeball becomes softer (*atrophy of the eyeball*). From the start the sight is very much reduced by the exudation in the vitreous, and later is as a rule absolutely annihilated. It is only in exceptional cases that a portion of the retina remains attached, and that a residue of sight is preserved.

The cases of simple suppurative chorioiditis which run their course without producing an external inflammation are rare.

Cases of simple abscess of the vitreous may be confounded with neoplasms in the eyeball. This is possible because the inflammation in these cases runs so sluggish a course that all external signs of inflammation are wanting. The eye is not discolored, the iris is normal, the aqueous and lens are clear. The lens and iris are pressed forward by the exudation in the vitreous, and the anterior chamber is made shallower. The pupil is dilated, and permits the exudate, or abscess, to be seen deep down in the vitreous. Sometimes the exudate is visible even some distance off as a vivid, light-colored (whitish or yellow) reflex from the pupil (*amaurotic cat's eye* [Beer]). Just the same phenomena may be produced by new formations in the vitreous, and particularly by gliomata arising from the retina (see § 535), for which reason many cases of the sort described above have been designated as *pseudo-gliomata*. The most important distinctive mark lies in the tension of the eye. This, in genuine glioma, is normal in the beginning, and afterward is increased; in pseudo-glioma, on the contrary, diminution of tension soon sets in, which is followed by the shrinking of the eye. Then the subsequent course renders the true state of the case perfectly clear, inasmuch as glioma afterward breaks through the sclera and keeps on growing, while in pseudo-glioma the eye keeps constantly growing smaller.[2] But now in glioma it is requisite to remove the eye as early as possible, and it is therefore not right to wait a long time in doubtful cases until the diagnosis can be determined with certainty. In so doing, we should be putting our patient's life in jeopardy. Hence, in doubtful cases, we perform enucleation. Even if it should then turn out that the case was one of pseudo-glioma, the patient has not lost much by the enucleation, since the eye is already blind and would be much shrunken. In any case, the question of a confusion with glioma would come up only in cases of sluggish abscess of the vitreous occurring in children, since it is only in the children that glioma of the retina is found. The causes of pseudo-glioma are most frequently meningitis, also the acute exanthemata, and finally injuries, particularly those attended with the presence of a small foreign body in the interior of the eye. One or two cases of pseudo-glioma have turned out on dissection to be tuberculosis of the chorioid; [others are due to exudative retinitis]; and in some instances a mass of tissue of fetal origin situated behind the lens is the cause of the yellow reflex in the pupil.

422. (2) The purulent inflammation extends from the back part of the eye to the region of the anterior chamber; and in other cases, as stated above, it starts from the latter. There is then together with the suppuration in the vitreous a purulent iritis (*endophthalmitis septica*). In such cases the suppuration in the vitreous cannot be recognized with the same certainty as in the case of a simple abscess of the vitreous, because exudation into the anterior chamber (a turbidity in the aqueous, a hypopyon, a pupil-

[2] [In rare cases, however, a glioma or sarcoma may be associated with irido-cyclitis and come to be lodged in a phthisical eye. See remarks under Sarcoma of the Chorioid, page 476.—D.]

lary membrane) often interferes with a view into the deeper parts. And yet it is in these cases, which externally look like a violent suppurative iridocyclitis, that it is particularly important to determine whether the purulent inflammation is present in the posterior segment of the eye also, because upon this point depend both prognosis and treatment. Indeed, in case of suppuration of the vitreous the eye is lost, and if we are dealing with traumatic cases, enucleation is indicated. In cases in which the exudate in the vitreous cannot be seen directly, the diagnosis must be based on a test of the light perception (see § 754). If this is nearly or quite abolished, it is a proof that the retina is no longer functioning, which may be taken as evidence that there is a purulent inflammation of the latter (cf. page 416).

The outcome of endophthalmitis may be gradual shriveling of the eye just as in abscess of the vitreous. In the severest cases, on the other hand, the pus accumulated in the eye breaks through to the outside, and these cases are denoted as—

423. (3) *Panophthalmitis.*—The œdema of the lid increases so much that the physician can scarcely separate the lids. The appearance of the eyeball varies. If the suppuration originates in changes in the anterior division of the eyeball—e. g., from corneal ulcers, or from injuries—the symptoms of these conditions appear in the foreground, while those cases in which the inflammation has taken its origin in the deeper parts, show the picture, portrayed above, of a yellow exudate behind the lens. In any case, whatever the appearance of the eye, there is present as one of the chief symptoms of panophthalmitis marked protrusion of the eyeball (exophthalmus) with abolition of movement. The pain can scarcely be borne, and not infrequently annoying photopsiæ also are present. There is fever, and vomiting frequently occurs, especially at the beginning of the disease. These symptoms keep on until the purulent exudate in the interior of the eye makes an exit for itself by breaking through the sclera. Perforation takes place in the anterior division of the sclera. The conjunctiva is seen to bulge forward at some spot, showing the yellowish, discolored sclera through it, until finally sclera and conjunctiva are perforated and the purulent contents of the eyeball are slowly extruded. After perforation has occurred the pains soon cease, and the eye becomes softer and ultimately shrinks up to a small stump (*phthisis bulbi*). It takes at least six to eight weeks for this result to be attained and for the eye to become perfectly free from pain.

Panophthalmitis, therefore, is an endophthalmitis which goes on to the formation of an abscess, and is characterized by the development of two additional symptoms—protrusion of the eyeball and purulent perforation of the envelopes of the eye. The protrusion is due to the extension of the inflammation to the tissues behind the eye and above all to Tenon's capsule; and just as the lids and conjunctiva are œdematous, so also there develops a marked inflammatory œdema behind the eye and in consequence of this a protrusion of the ball.

Most cases of *panophthalmitis* are caused by injury. If the latter is of such a character that the eye is extensively opened, the purulent exudate may be discharged through the wound. This is true of the cases of panophthalmitis after extensive suppuration of the cornea. In all these cases the pus in the interior of the eye is not under any pressure, and hence the disease runs a mild course with but little pain and slight inflammatory symptoms. The total course, too, is comparatively quick, since the condition can proceed without waiting for the sclera to be perforated, which always requires a long time. Yet not infrequently even in these cases, in which the purulent exudate pushes out through the wound, perforation of the sclera by pus is also seen to take place as usual. Panophthalmitis after severe injuries must, from a certain point of view, be regarded as a more favorable outcome than a plastic irido-cyclitis. The former to be sure, causes more violent pain and leads to a greater degree of shrinking of the eye; but when it has run its course the patient has a lasting respite. Plastic irido-cyclitis, on the other hand, often for years produces after-attacks of inflammation, and may also give rise to sympathetic disease of the other eye, if the patient does not submit to enucleation at the proper time.

For the *anatomical changes* in irido-chorioiditis suppurativa, panophthalmitis, and ophthalmia metastatica, see page 417.

424. Etiology.—The three categories of purulent inflammation of the interior of the eye are essentially the same process and differ simply in extent (in one instance being confined to the posterior division of the eye, in the other affecting the whole interior of the ball) or else differ in the intensity of the inflammation (termination in one case being in gradual shriveling, in the other in suppuration). Hence the three categories are in no way sharply differentiated from one another, and there are transition forms between them.

So, too, the etiology of all three is alike. It is always a case of an infection of the interior of the eye by pyogenic germs. The infection may originate from the outside or have its source in the body itself.

Infection *from the outside* (*ectogenous infection*) occurs—

(*a*) Most frequently from penetrating injuries of all kinds. In this category belong unsuccessful operations.

(*b*) From the passage of suppuration from without inward, in the case of perforating ulcers of the cornea, and from prolapses of the iris that are covered with pus.

(*c*) As a process starting from a cicatrix of the cornea with [or even without] incarceration of the iris, when the cicatrix is thinned. Under this head belong the incarcerations of the periphery of the iris, that not infrequently remain after cataract operations. Infection takes place in these cases from the germs penetrating through the thin cicatrix into the tissue of the incarcerated iris, and then traveling in the latter backward into the eye. The starting point for the infection of the old cicatrix may be afforded by inconsiderable lesions of the epithelium covering the cicatrix, or by sudden stretching or bursting open of the latter. [See also page 318 .]

Infection by carriers of infection, which arise from the *organism itself* (*endogenous infection*), takes place—

1. Through embolism, septic substances from a focus of suppuration getting into the circulation and becoming arrested in the vessels of the chorioid. In this way *metastatic ophthalmia* develops. This is one of the symptoms of pyæmia, and most frequently of that form which makes its appearance in the puerperal period as puerperal fever. Moreover, we often find metastatic purulent chorioiditis in meningitis, and particularly in cerebrospinal meningitis. These cases are observed chiefly in children, and are distinguished by their comparatively mild course, so that in rare instances actually some small degree of sight is still retained.

2. By transfer of inflammation from behind forward in phlegmons in the orbit and in thrombo-phlebitis of the orbital veins.

3. By direct transmission of inflammation from the meninges to the eyeball along the sheaths of the optic nerve. This occurs in certain cases of meningitis. But most cases of suppurative chorioiditis due to meningitis are metastatic in origin (see above).

Metastatic ophthalmia occurs both as a unilateral and a bilateral affection. Cases of the former kind generally afford a better prognosis as far as the pyæmia is concerned, and particularly so when no metastases are observable, except the one in the eye. The prognosis that appertains to the bilateral cases, on the other hand, is extremely bad, even for life itself.

Many cases of panophthalmitis that develop suddenly, and to all appearances spontaneously, should perhaps be regarded as one of the symptoms of a pyæmia whose point of origin is undiscoverable. In children suppuration of the umbilical cord, and sometimes also vaccination, may give rise to pyæmia with metastatic ophthalmia. [Metastatic ophthalmia may also be caused by gonorrhœa (Von Hippel).—D.]

Besides occurring in pyæmia, a purulent chorioiditis, doubtless also of metastatic origin, occurs in rare instances in other acute infectious diseases, such as typhoid fever, variola, scarlet fever, anthrax, influenza, ulcerative endocarditis, diphtheria, erysipelas, pneumonia, and Weil's disease.

For the exciting germs of endophthalmitis, see pages 34 and 36.

425. Treatment.—Treatment cannot save the sight in suppurative chorioiditis. In cases in which the inflammation is not too violent, we confine ourselves to alleviating the pain by means of moist warm compresses and narcotics, until the shriveling eyeball becomes gradually destitute of sensation. If the pain is very severe, it is better to relieve the patient of a long and painful malady by early enucleation, especially when the disease threatens to go on to panophthalmitis. Enucleation is unconditionally indicated in all traumatic cases, since then it is a question not simply of cutting short the disease, but of preventing sympathetic ophthalmia. When there is a fully developed panophthalmitis, enucleation is not without risk (on account of the danger of a succeeding suppurative meningitis). We do better, therefore, if we content ourselves with opening the eye in its anterior

portion, either by abscising the cornea or by making a free incision in the sclera. In this way the eyeball, which is tightly distended by the exudation, is freed from tension, the discharge of the suppurating contents of the eye is accelerated, and thus the pain and the progress of the disease are cut short. When the eye at last becomes shrunken it usually remains quiescent and also admits of an artificial eye being worn over it. Should, however, secondary inflammatory phenomena set in, as they may exceptionally do in the shrunken eye, its enucleation is indicated.

Apart from simple incision of the sclera, different methods have been attempted for cutting short the course of panophthalmitis, particularly enucleation and the ablation of the anterior section of the eye, with scooping out of the contents of the eyeball. In fully developed panophthalmitis enucleation sometimes results in purulent meningitis with a fatal issue. It must be assumed that by the operation the blood and lymph passages in the orbit are freely opened, and thus made accessible to infection. Whether the scooping out (exenteration or evisceration) of the suppurating eye is a less dangerous procedure is questionable, since cases of death after this operation have also been observed (Schulek). It must be noted, however, that some cases have been known in which fatal meningitis has succeeded a panophthalmitis without any operative interference. [Cf. §§ 892, 894.]

[**426. Primary Chorioidal Sclerosis.**—This is a rare condition characterized by atrophy of the pigment epithelium and the chorio-capillaris, so that, owing to the exposure of the chorioidal stroma, the red fundus becomes browner in tint. The large chorioidal vessels stand out clearly and are accentuated by white lateral streaks which lie upon them and in advanced cases merge over them, so that the vessels appear of a creamy white. The process may be macular (occurring as a degenerative condition in old people), circumpapillary, or general. Syphilis, arteriosclerosis, nephritis, and menstrual disturbances have been assigned as causes, but the etiology is obscure. The condition is not inflammatory, but must be regarded as a primary degeneration of the chorioid (Wood).—D.]

427. Detachment of the Chorioid.—This is frequently found in the dissection of enucleated eyes. In shrunken eyes the chorioid—and the ciliary body, too—are very frequently found to be detached by the exudates which are present in the vitreous chamber, and which exert a centripetal traction in all directions (*a*, *a*, Fig. 176). Then owing to the negative pressure serous liquid collects beneath the chorioid.

A detachment of the retina, usually total, is never wanting in these cases. Since we are dealing with eyes which are already blind, the detachment of the chorioid has a practical interest only in so far as traction upon the ciliary body is produced by it; for this may induce conditions of irritation in the blinded eye.

Another mode of detachment is the active propulsion of the chorioid from the sclera by blood which is poured out beneath the chorioid. This takes place as a result of rupture of the ciliary arteries when in an eye (usually one affected with old increase of tension or with degenerated blood-vessels) the intra-ocular pressure sinks to zero in consequence of perforation (due to corneal ulcer, injury, or operation). The hæmorrhage may be so great that the inner coats of the eye are in large part expelled from the eyeball (expulsive hæmorrhage). In this way an eye that still retains sight may be destroyed.

Detachment may also be produced by exudation under the chorioid or by the development of a sarcoma in its outer layers.

It is a rare thing to see with the ophthalmoscope a detachment of the chorioid in an eye which still retains sight and in which the media are transparent. The detached chorioid appears under the guise of a dark prominence projecting into the vitreous. This sort of detachment is most frequently produced by serous fluid, and, in fact, by aqueous which percolates from the anterior chamber beneath the chorioid through a small rent in the attachment of the ciliary body. [According to Marshall, Hudson, and others, the detachment is due to serous exudation arising from the chorioidal vessels.—D.] Hence, we find, at the same time, that the anterior chamber is either shallower or quite obliterated. This serous detachment occurs not infrequently in the days immediately succeeding an extraction of cataract or an iridectomy for glaucoma [also quite frequently after Elliot's trephine operation and still more frequently after Lagrange's sclerectomy (Barkan).—D.] It gives a good prognosis, since usually the chorioid becomes reattached to the sclera.

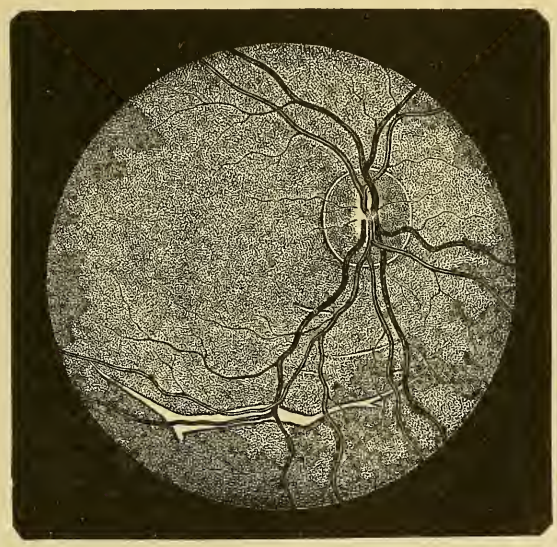


Fig. 206.—Rupture of the Chorioid. Right Eye. (After De Wecker.)

There are one large and four small ruptures in the lower part of the chorioid. The large rupture appears under the guise of a slightly curved white band with jagged, somewhat pigmented borders. The small ruptures lie concentric with the large one and between it and the papilla. Over all of them the retinal vessels run unaltered.

428. Rupture of the Chorioid.—This is produced through the action of some blunt body upon the eyeball (contusion). Immediately after the injury the blood extravasated into the vitreous usually prevents a clear view of the interior of the eye. After the absorption of the blood the rupture of the chorioid, which ordinarily lies in the neighborhood of the papilla, and most frequently to the outer side of it, is discovered. Sometimes there is only one, sometimes there are several lacerations. They form long, yellowish-white streaks, as the edges of the laceration in the chorioid gape and allow the white sclera to be seen between them (Fig. 206). The streaks generally have a curved shape, with the concavity toward the papilla; they are broadest in the center, and taper off to a sharp point at the ends. Their edges have an irregular black coloration, due to proliferation of the pigment. The retinal vessels run without any change over the streaks, a proof that the retina is unruptured.

II. Tumors of the Chorioid

429. Of malignant tumors the one [chiefly] occurring in the chorioid is *sarcoma*, which in most cases is pigmented (melano-sarcoma). The clinical symptoms that sarcoma of the chorioid presents change so during the development of the tumor that four stages can be distinguished in the course of the disease.

In the *first stage* the tumor is small, and manifests itself only in ophthalmoscopic examination by detachment of the retina at the site of the tumor. The patient notices a disturbance of vision in the shape of a defect in the visual field, corresponding to the site of the tumor. Afterward the detachment of the retina becomes total (Fig. 207, *N*), and thus the eye, which externally still looks normal, becomes completely blind.

Sarcomata of the chorioid consist either of round cells or of spindle cells, or are tumors made up of a mixture of both. They are almost always pigmented (melano-sarcomata); non-pigmented sarcomata (leuco-sarcomata) are among the rarities. Very often they contain many and wide blood-vessels. Sarcomata develop from the external layers of the chorioid (layer of large and of medium-sized vessels) and grow inward toward the vitreous space, pushing the retina before them. In the beginning the retina lies everywhere in contact with the surface of the tumor, so that with the ophthalmoscope a sharply circumscribed gibbous detachment of the retina is found, rising steeply from the fundus. This, for obvious reasons, does not fluctuate. Through the retina we can often recognize the vessels of the chorioid or of the tumor and also the yellow or brown color of the latter. In this case the *diagnosis of sarcoma* is easily made. But afterward, in consequence of the disturbance of circulation in the chorioid produced by the tumor, an accumulation of fluid takes place between the chorioid and the retina.

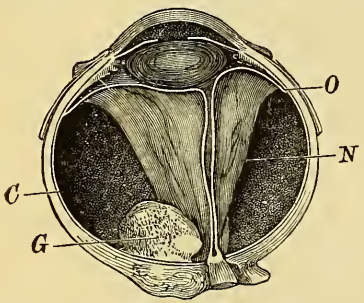


Fig. 207.—Sarcoma of the Chorioid. (After Leber.)

The tumor, *G*, rises from the chorioid, *C*, which everywhere lies in contact with the sclera. The retina, *N*, on the contrary, is detached entirely from its bed under the form of a folded funnel. It retains its connection only with the papilla behind, and with the chorioid along the ora serrata, *O*, in front.

The latter is thus detached over an area greater than that occupied by the tumor, and does not permit the latter to be seen through it any more; ultimately, the detachment becomes total (Fig. 207). In this stage, since the detachment of the retina has lost its characteristic appearance, the diagnosis cannot for the most part be made with certainty. The tension of the eye affords one diagnostic point, being usually diminished early in simple serous detachment of the retina, while in detachment due to a tumor it is at first normal and afterward increased (Von Graefe). It is an additional argument for the existence of sarcoma if the anterior ciliary veins are found markedly dilated upon one side or the other. These dilated veins indicate that the sarcoma is situated in the chorioid, and, in the area affected, prevents the blood in the forepart of the uvea from flowing out through the vasa vorticosa, so that it has to make its way through the anterior ciliary veins. As the intra-ocular pressure later on steadily increases, the detachment of the retina often assumes a characteristic appearance. The retinal protrusions are pushed against the posterior surface of the lens and against each other by

the increased pressure in the subretinal space; they flatten themselves upon each other and often include only a narrow Y-shaped furrow between them. There are, it is true, serous detachments which also show this appearance. These are those rare cases in which serous detachment is associated with increase of tension. Such cases are not distinguishable from tumor of the chorioid and might, in analogy with pseudo-glioma (see page 420) be called pseudo-sarcoma. To make the right diagnosis in such cases we may call in the aid of transillumination (see page 94). Or we may employ puncture. This is made with a fine needle, which is plunged deep in through the sclera at the spot where the tumor is thought to be. If the needle enters a tumor, it is not freely movable, as would be the case if its point lay in the vitreous or in subretinal fluid. If in spite of these diagnostic aids we are still in doubt, enucleation of the eye, which would be blind anyhow, is unconditionally indicated.

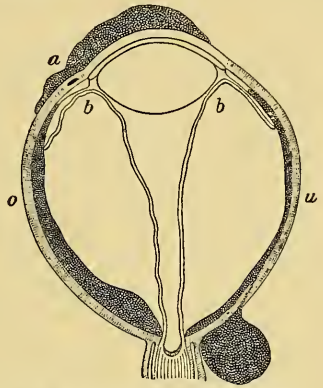


FIG. 208.—SARCOMA CHORIOIDEÆ DIFFUSUM. VERTICAL SECTION THROUGH THE RIGHT EYE.

The patient, who was fifty-four years old, had been blind in this eye for years from an opacity in the cornea. The eye became inflamed from time to time and half a year previous had begun to take on a black color. On the patient's admission the eye was found to be enlarged in all its parts and the cornea was covered by a granular brownish-black deposit which only at the nasal and lower side left a narrow marginal portion of the cornea free. In the section made through the eye we see that the latter as a whole is elongated and tapers off into a somewhat oval extremity behind—two signs of high myopia, which was also present in the patient's other eye. The cornea is covered with a flat dark pseudoplasm which, at the upper side, *o*, is continued on to the adjoining sclera, but at the lower side, *u*, still leaves some part of the cornea free. The cornea beneath the tumor is scarcely altered; only in a few spots have the sarcomatous masses destroyed Bowman's membrane, but nowhere have they penetrated into the deeper parts. Near the upper border of the cornea, at *a*, there lies in the thickness of the sclera a little black island of sarcomatous tissue representing the cross section of an anterior ciliary vein. This vein is filled with sarcoma masses and constitutes the way by which the intra-ocular pseudoplasm had grown through to the outside. In the interior of the eye the chorioid and the flat portion of the ciliary body are everywhere uniformly sarcomatous; a coating of black pseudoplasm also covers the floor of the excavation in the optic nerve. Adjoining the lower side of the latter lies a second extra-ocular nodule from which a filament of black sarcoma tissue runs through the sclera obliquely forward and inward as far as the chorioid. At this spot the intra-ocular sarcoma has grown out through the sclera along a ciliary nerve. In the interior of the eye, furthermore, are found the evidences of a long continued and very marked increase of tension—namely pushing of the iris and lens against the cornea, atrophy of the ciliary processes, deep and total excavation of the head of the optic nerve.

A rare form of sarcoma of the chorioid is the *diffuse* variety, in which the new growth does not begin as a circumscribed rounded tumor, but as uniform degeneration, which is diffused over wide areas and in which often the ciliary body and even the iris are likewise implicated (Fig. 208).

430. In the further growth of the tumor a time occurs when increase of tension suddenly sets in. The sarcoma thereupon enters the *second stage* of its development—that of increased tension. Externally the eye presents the appearances of inflammatory glaucoma (page 491). Marked injection of the

eyeball exists, the cornea is dull and clouded, the anterior chamber shallow, the iris discolored, the pupil dilated and immobile, and the tension of the eye to palpation is noticeably elevated. If the media are sufficiently clear, the gray reflex of the detached retina can be seen deep down behind the pupil. Later on, the lens becomes clouded, so that the clinical picture of glaucoma absolutum with cataracta glaucomatosa is produced. From the time when the symptoms of inflammatory glaucoma set in the patient suffers with pain; very frequently it is by this that he has his attention first called to his trouble. Since the picture presented by the affected eye corresponds completely to the complex of symptoms of inflammatory glaucoma, the correct diagnosis in this stage is to be made with difficulty, or not at all.

If the detachment of the retina is not already total, it becomes so now, and the eye becomes completely blind. The point of time at which the glaucomatous attack sets in does not depend directly upon the size of the intra-ocular tumor. The increase in tension does not arise from the fact that the tumor occupies a certain space in the interior of the eye, for this call for additional space is compensated for by a corresponding decrease of the vitreous. On the contrary, the increase in tension is based upon the congestion which the tumor produces in the veins of the chorioid, and by which increased transudation of fluid takes place into the interior of the eye. Elevated tension is, therefore, often seen with quite small tumors, while at another time the tumor may have already filled up a large part of the eye without exciting the symptoms of glaucoma. When the glaucomatous attack has set in, the eye looks as if it had been blinded by primary glaucoma, and the diagnosis cannot be made with certainty. The existence of a sarcoma will be suspected if the patient states that the eye was already entirely blind before the outbreak of the inflammation, for in primary glaucoma blindness usually does not precede the attack, but follows it. Besides, we examine the second eye; if one eye is completely blinded by primary glaucoma, the second eye will rarely be found quite normal.

In rare cases the second (inflammatory) stage of formation of the tumor does not present the symptoms of glaucoma, but of a severe *irido-cyclitis*, which because the inflammation is especially violent, is often accompanied by chemosis and exophthalmus. Such inflammation is apt to occur when the intra-ocular tumor owing to inadequate nutrition has become largely or wholly necrotic. As a result of the irido-cyclitis, the eye becomes softer and shrinks up so far as the tumor contained in it permits. The growth of the latter is thus retarded for some time—a fact which does not prevent epibulbar tumors and metastases from developing.

431. The *third stage* is that of the growth of the tumor upon the outside. The symptoms are different, according as the tumor breaks through the sclera in its anterior or in its posterior division. In the former case, dark, hard prominences are seen developing in the circumference of the cornea, and the diagnosis can readily be made. If, however, the tumor first grows through the sclera posteriorly, the nodules of the tumor are invisible, and do not give evidence of their existence until later, through the gradually increasing protrusion of the eyeball (exophthalmus). As soon as the tumor has broken through the envelopes of the eyeball to a sufficient extent, the pain usually abates, since the great tension in the eyeball then ceases. But, to make up for this, the extra-ocular masses of tumor, freed from the

intra-ocular pressure that constrained them, grow so much the quicker. First the orbit is entirely filled by the tumor, afterward the latter projects from the orbit, as big as an apple or as the fist. From the orbit the tumor is continued to the neighboring parts, particularly to the brain. At its exposed portions the tumor ulcerates and gives rise to frequent hæmorrhages.

Growth of the mass exteriorly occurs before the tumor has yet filled the whole interior of the eyeball, and is effected by the gradual growth of the cells of the tumor through the sclera, the cells usually following out preformed passages. We hence find the tumor growing out along the optic nerve and its sheaths, or utilizing the points where the blood-vessels (anterior or posterior ciliary vessels or the vasa vorticosa) and the ciliary nerves penetrate the sclera (Fig. 208).

432. The *fourth stage* is that of the generalization of the tumor by the development of metastatic nodules in the internal organs, most frequently in the liver.

Years usually pass before the sarcoma has run through the four stages just pictured. The first and second stages last a long time, while afterward the growth of the tumor becomes continually more rapid. The patient dies either from exhaustion, in consequence of the suppuration and hæmorrhage from the tumor, or succumbs to the extension of the latter into the brain or to the metastases in the internal organs.

The *metastases* in remote organs arise through embolism. The blood current detaches cells from the tumor and carries them into other parts of the body, where they develop into independent tumors. Local recurrences are scarcely to be apprehended if the growth was confined to the eyeball at the time of the operation. On the other hand metastases occur even in cases in which enucleation was performed very early. Such metastases remain unobserved at the outset; and sometimes it is years before they cause the patient's death.

433. The *prognosis* of sarcoma of the chorioid is absolutely unfavorable for the life of the patient if the eye is not removed early. But even then the prognosis is by no means to be regarded as perfectly favorable. Apart from the fact that the eye itself in every case is lost, both local recurrences in the orbit and also metastases may develop even after its removal, the germs for their development having been already scattered abroad earlier, although, at the time when the eye was removed, they were too small to be demonstrable. Sarcoma of the chorioid is hence to be regarded as one of the most malignant of diseases—one which, in very many cases, ends in death. Sarcomata of the iris and ciliary body behave, in respect to their course and ultimate outcome, like sarcomata of the chorioid.

Sarcoma of the chorioid is a rare disease. It is found most frequently between the fortieth and sixtieth year; in childhood it is of extremely rare occurrence. This gives a means of distinguishing it from the gliomata which spring from the retina, and which in part present symptoms like those of sarcomata, but which occur in childhood exclusively. A malignant neoplasm

developing in the eyeball will, therefore, have to be regarded in all probability as a glioma in a child and as a sarcoma in an adult.

The *treatment*, as long as the neoplasm is still confined to the eyeball, consists in enucleation, which should be performed as early as possible. In doing it, we cut off the optic nerve as far back as possible, to meet the contingency that the degeneration has already passed over upon it. If the neoplasm has already grown out of the eyeball, everything diseased must be removed according to surgical rules. The surest method in this case is exenteration of the orbit—i. e., the removal of the entire contents of the orbit, together with the periosteum.

Other Tumors of Chorioid.—Cavernous angioma, endothelioma, perithelioma [and melanoma] have been known to occur as primary tumors of the chorioid in a few cases. [Melanomata are benign tumors which greatly resemble small sarcomata. They can be distinguished from the latter by their uniform slaty color; their definite, somewhat feathered, outline; the absence of abnormities in the appearance of the overlying retina; the absence of pigmentary disturbances adjoining the tumor; and the absence of any symptoms of retinal involvement (Moore).—D.]

Carcinomata and also adenomata have been observed as a great rarity in the chorioid, but only as secondary tumors, as metastases from carcinoma in other organs (especially in the thoracic glands).

III. Tuberculosis of the Chorioid

434. In the chorioid, as in the iris, tuberculosis occurs under the two forms of disseminated and of solitary nodules. The diagnosis between them is made with the ophthalmoscope.

(*a*) *Disseminated* or *miliary* tuberculosis of the chorioid was first described by Jäger. Small ill-defined patches of yellowish or pale-reddish color are seen in the fundus. These even within a short period of examination—within a few days—grow larger without, however, attaining a size of more than one-third of the optic disk; and at the same time new patches may make their appearance in the fundus. By this rapidity of growth the affection is distinguished from chorioiditic spots, which change very slowly; besides the pigment changes, so frequent in chorioiditis, are wanting in tubercles of the chorioid. The tubercles occupy chiefly the posterior division of the chorioid. Ordinarily only a small number are present, although sometimes as many as twenty or thirty of them can be counted in the eye. Anatomical examination has proved that the spots seen with the ophthalmoscope correspond to nodules of a mean diameter of 1 mm., which possess the typical structure of tubercle nodules (Manz).

Miliary tuberculosis of the chorioid forms one of the symptoms of general miliary tuberculosis (Cohnheim). It has essentially a diagnostic interest; as in doubtful cases of acute miliary tuberculosis, it may assist in establishing the diagnosis. In chronic tuberculosis of the lungs, intestine, etc., it is not ordinarily observed. [According to Stephenson and Carpenter, whose views are corroborated by other observers, miliary tuberculosis of the chorioid may occur in any stage or kind of tuberculosis and is common in chronic tuberculosis. It generally forms a small exudate which is found near the optic nerve or macula, and which ultimately becomes absorbed, leaving an atrophic pigmented patch in the chorioid. Sometimes the extra-ocular tuberculous lesion that causes the chorioidal disease is not obvious, so that the eye condition becomes of diagnostic value in that it points to the existence of latent tuberculosis.—D.]

(*b*) *Solitary* or *conglobated* tubercle of the chorioid makes its appearance under the form of a neoplasm. With the ophthalmoscope a rather large, light-colored tumor is seen in the chorioid; it is an argument for its tuberculous nature if smaller light spots (tubercle nodules) are found in the chorioid in its vicinity. The tumor may afterward grow through the sclera to the outside, and there break down. Anatomical examination shows that it consists of a great number of smaller miliary nodules, which have coalesced to form one pretty large tumor. In the center of the latter caseation takes place. The solitary form of tuberculosis of the chorioid is a very rare disease, pre-eminently affecting young people. It runs a chronic course, and accompanies chronic tuberculosis of the internal organs, especially of the brain. There are, however, cases in which, beside the tuberculous nodule in the eye, no focus of tubercle can be clinically demonstrated to exist in the organism.

The prognosis of solitary tubercle of the chorioid is bad, since the eye in any case is lost, and in most cases also life is endangered through the presence of tuberculous diseases in other parts.

It is probable, too, that not a few cases of disseminated chorioiditis, especially in young persons, are a kind of attenuated tuberculosis of the chorioid, although a sure proof of this has not yet been produced.

[In suspected tuberculosis of the chorioid, we use the Von Pirquet test and also injections of tuberculin (see page 70) to establish the diagnosis.—D.]

For treatment we must first initiate general measures directed against tuberculosis [and especially tuberculin injections, and with these are not infrequently successful]; but if we see that the tuberculous focus is increasing in spite of these measures and that blindness is setting in, enucleation is indicated.

IV. Congenital Anomalies of the Chorioid

435. Coloboma of the Chorioid.—In this affection the ophthalmoscope shows a brilliant white area in the red fundus below the optic-nerve entrance (Fig. 209). This represents a circumscribed defect in the chorioid and retina, in the confines of which the sclera lies exposed, and is hence visible as a white surface. Coloboma of the chorioid is frequently found along with coloboma of the iris, and also with other congenital anomalies of the eye. Such eyes are often smaller than usual (microphthalmus). Sometimes, indeed, eyeballs are found which are only as large as a pea or a millet seed, and which lie entirely in the back part of the orbit, and are not discovered in an examination made upon the living subject. In this way absence of the eye (anophthalmus) is simulated (see page 482). Whether a true anophthalmus—i. e., a condition in which, while the orbit is present, there is not even a rudiment of the eyeball—does occur or not, has not so far been determined.

The eyesight suffers in coloboma of the chorioid because, in the first place, there is [usually] a defect in the visual field corresponding to the coloboma. Moreover, even the direct visual acuity is usually defective because the eye, as a whole, has its development deranged. In the higher degrees of microphthalmus the sight is reduced to the mere differentiation of light from darkness.

Coloboma of the chorioid is in a marked degree transmissible by inheritance, and that, too, not infrequently in conjunction with other congenital malformations of the body.

Coloboma of the chorioid has the shape either of an oval, whose long axis corresponds nearly to the vertical meridian, or of an obtuse-angled triangle whose apex is directed toward the papilla. The peripheral border of the coloboma not infrequently presents a tapering prolongation extending toward the ciliary body. Even the smallest colobomata are much larger than the optic disk; and the large colobomata are so extensive that their anterior border can no longer be seen with the ophthalmoscope, because

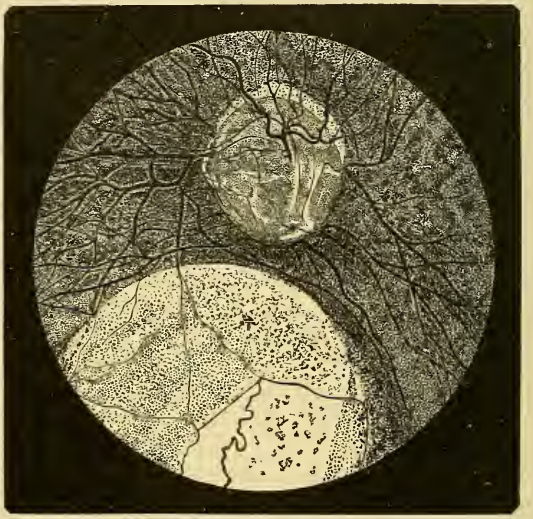


FIG. 209.—COLOBOMA OF THE OPTIC NERVE AND CHORIOID. FROM THE RIGHT EYE OF A FOURTEEN YEAR-OLD GIRL. ERECT IMAGE. (After Casper and Krüger.)

The papilla appears about nine times as large as in the normal state, and lies considerably below the level of the adjoining retina. An upper (yellowish) and a lower (gray-colored) portion can be made out in it. From the former rise the central vessels, which are abnormal in sending most of their branches upward. The lower (gray) portion of the papilla shows several light-colored, ridge-like projections and but a few blood-vessels, although numerous blood-vessels emerge at its overhanging border and run out into the retina, evading the coloboma. The enlarged papilla is bordered above by a narrow atrophic crescent. The coloboma of the chorioid lies below the papilla and somewhat to its temporal side. Its lower (anterior) border is not represented in the drawing. The coloboma is of brilliant white hue, is sharply defined and is placed somewhat deeper than the adjoining portions of the fundus. It shows a few blood-vessels and in spots a fine granular pigmentation.

it lies too far forward. So, too, they may extend so far backward that they involve the papilla. The latter, in that case, is generally changed in shape and appearance—sometimes so much so that we can scarcely tell where it lies except for the intimation of its existence afforded by the place of origin of the retinal vessels. [See page 481.]

The edge of the coloboma is sharply defined and commonly bordered by pigment. The coloboma itself is of a pure white or bluish-white hue, and displays here and there pigment spots and also vessels. The vessels are in part those that arise from the adjoining retina and chorioid, in part belong to the sclera, which lies exposed within the area of the coloboma, and in part are seen to originate from the coloboma itself. The latter set must be regarded as posterior ciliary vessels. In eyes of this sort the retinal vessels often display an irregular course; not infrequently it looks as if they were trying to evade the coloboma, since they run along its borders instead of passing over it.

The surface of the coloboma lies deeper than the rest of the fundus, and often presents channeled depressions or prominent ridges, as can be inferred both from the way in which the vessels bend and also from the parallactic displacement.

Cataract, generally of a complicated, inoperable character, frequently develops in eyes affected with coloboma of the chorioid. If I may be allowed to judge from one case whose course I myself observed, such a cataract is produced in the following way: The retina, being adherent to the margin of the coloboma, undergoes the same sort of traction that it does when adherent to a scleral cicatrix (see page 350). In consequence of this traction detachment of the retina takes place—at first only at the edge of the coloboma, but afterward over the whole extent of the retina. The clouding of the lens, then taking place, is to be regarded as the result ordinarily following total detachment of the retina.

In rare cases large, white, depressed areas have been observed not below, but to the outside of, the optic nerve, in the region of the yellow spot. These have likewise been looked upon as congenital malformations—*colobomata of the macula.*

[Colobomata of the chorioid may be associated with a complete *coloboma of the retina*—the retinal vessels in this case being cut short off at the margin of the affected area. In other cases functional tests show that the retina is at least in part preserved over the colobomatous chorioid.—D.]

The formation of colobomata occurs also in the *optic nerve.* Either a deep depression is found in the lower part of the latter or the entire optic-nerve entrance is enlarged to several times its usual size, and the vessels coming out from it are, as it were, forced apart (Fig. 209).

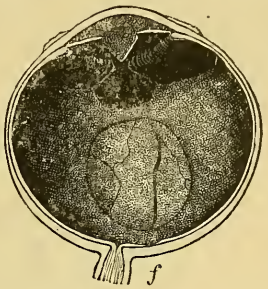


Fig. 210.—Lower Half of an Eye with Congenital Coloboma of the Iris, Ciliary Body, and Chorioid.

In the iris, whose posterior surface is seen in the figure, is recognized the prolongation of the pupil, running out in a sharp point to the lower ciliary margin. In the corresponding portion of the ciliary body, the ciliary processes are wanting; the processes immediately adjoining this gap are higher and longer than the rest, and, as they diverge backward, inclose a triangular, very darkly pigmented area. Still farther back there is found in the wall of the eyeball a deep excavation of oval form, whose edges are sharply defined and in part overhanging. Upon the floor of the excavation is seen the sclera, covered simply by a very thin, transparent pellicle, in which run several vessels. The posterior pole of the oval that is formed by the coloboma of the chorioid is directed toward the fovea centralis, *f*.

Anatomical examination of the eye affected with coloboma of the chorioid shows even upon an external view a protrusion of the sclera, situated below the optic nerve. This is the scleral protrusion first described by Ammon (see page 358). Corresponding to this, in the inner membrane of the eye, is the coloboma visible with the ophthalmoscope (Fig. 210). Within this, microscopical examination reveals, for the most part, only a thin pellicle composed of connective tissue, the remains of the fused chorioid and retina.

436. Origin of Colobomata.—The starting point of a coloboma we must regard as located in the fetal ocular cleft.

This is found at the lower side of the secondary ocular vesicle—the flask-shaped structure of the embryonic eye—and is designed for the admission of the blood-vessels into the interior of this structure (Figs. 160, 161, see also page 391). Later on, this cleft ought to close again without leaving any trace of its presence. But if the closure takes place incompletely, a coloboma is formed. The regular closure of the fissure is prevented, because the process of mesoderm which conveys the blood-vessels and which passes through the fissure into the interior of the eye does not undergo retrogression at the proper time. The layers of the ocular vesicle which grow toward each other do not in

that case come into contact (Fig. 211, A) and the interspace left between them, which afterwards becomes the coloboma, is closed simply by mesodermal tissue and so remains. This mesodermal tissue at the time when the ocular fissure ought to close is not yet differentiated into chorioid and sclera. Even later this differentiation fails to occur wherever in the course of the unclosed fissure the outer layer of the ocular vesicle is wanting. In this spot there develops from the mesodermal plug not chorioid but merely a simple fibrous tissue which later on becomes ectatic because of the intra-ocular pressure (scleral protuberance). The coloboma therefore is originally present in the retina and the pigment epithelium and then secondarily in the chorioid also. In many animals the persistence of the mesodermal plug and hence the patency of the ocular fissure are the rule. The pecten of the bird's eye and the falx of the fish's eye are nothing but a more highly developed mesodermal plug. In this respect we might consider the cause of a coloboma formation in man to be a reversion to earlier forms.

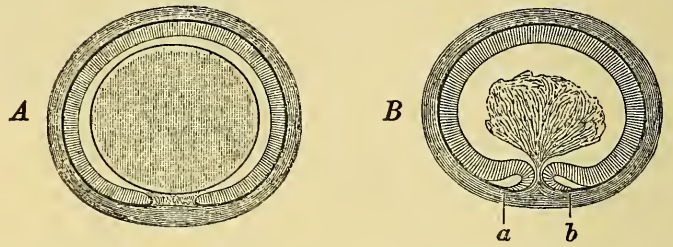


Fig. 211.—Development of Coloboma of the Chorioid. Schematic. (After Hippel, Junior.)

The layers from without inwards are the mesoderm (not yet differentiated into sclera and chorioid), and the outer layer (pigment epithelium) and the inner layer (retina) of the secondary ocular vesicle.

A. The interior of the ocular vesicle is entirely filled by the lens. At the lower side the ocular fissure is wide open, and the mesoderm passes through it into the interior of the eye as far as the lens. At a point exactly corresponding to the margin of the ocular fissure the outer layer of the ocular vesicle turns round the inner one and merges into it.

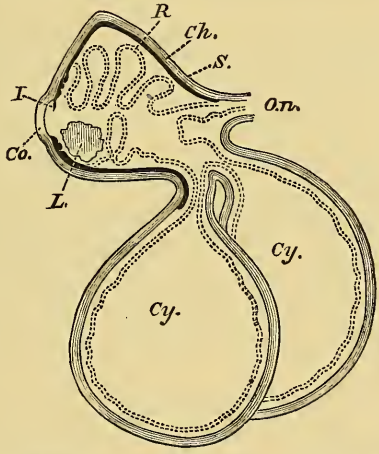
B. Here the ocular fissure is narrower and the process of mesoderm is more slender, but expands within the interior of the eye. The margins of the ocular fissure as they grow up towards the process of mesoderm have rotated in such a way that the inner layer is ectropionized outward and does not merge into the outer layer until at some distance from the fissure (at *a* and *b*). The fissure in the outer layer, therefore, is larger than the ocular fissure proper; hence, in the fully developed eye a comparatively large area (the area *a*, *b*) would be denuded of pigment and consequently appear white under the form of a coloboma, while as a matter of fact in the lateral portions of this area the retina, which is derived from the inner layer, might still be present. This is the reason why in many cases we find on anatomical examination that the coloboma is partially lined with rudimentary retina. If the mesoderm between *a* and *b* should yield and should protrude outward and the marginal folds of the ocular vesicle should grow into the cavity thus produced, the result would be the formation of a sac lined with retina at the lower side of the eye.

If the margins of the ocular vesicles grow up against the process of mesoderm and find in it an obstacle to their union they may undergo rotation (Fig. 211, B). They then grow on in a faulty direction, either into the interior of the eye or more frequently toward the outside. In the latter case there is formed at the lower side of the eyeball a sac-like pocket which contains retinal tissue (Hippel, Junior). In the more marked varieties of this anomaly of development, the eyeball itself remains quite small while the pocket develops into a pretty large sac. In this way develop the cases of *microphthalmus*, or of apparent *anophthalmus*, with the simultaneous presence of quite a large cyst situated in the lower lid, and glimmering with a bluish luster through the skin of the latter. The cyst is filled with a serous liquid, is lined with rudimentary retina, and is connected by a process with the dwarfed eyeball [Fig. 212].

The fetal ocular cleft is also continued as a furrow on the stalk of the ocular vesicle, which later becomes the optic nerve. Incomplete closure of this furrow causes colobomata of the optic nerve to develop.

Coloboma of the iris is likewise connected with the ocular cleft. The iris grows out from the anterior margin of the secondary ocular vesicle and the rudimentary chorioid, at a time when the fetal ocular cleft is already closed; hence the iris in no stage of its development has a fissure. But when the optic vesicle and the mesodermal tissue covering it suffer a derangement of development at the site of the retinal cleft, it is conceivable that derangements may also take place in the same meridian further forward and in that case may affect the iris. For example, the mesoderm at this spot (vascular lens capsule) may have an unusually solid connection with the mesoderm that forms the envelopes of the eye (Fig. 163). A firm band of this sort necessarily prevents the iris from growing forward. This may happen even when the cleft in the retina and chorioid has closed completely, so that then a coloboma of the iris is produced without a coexisting coloboma of the chorioid.

437. Albinism consists in the absence of the physiological pigment. Albinos have yellowish-white flaxen hair and also white eyebrows and lashes. The iris is light gray, and appears reddish by transmitted light, while the pupil has a vivid red luster.



[Fig. 212.—Diagrammatic Representation of Microphthalmic Eye with Two Cysts Attached. (After Lang and Collins in Norris and Oliver.)

Co, cornea; *L*, lens displaced and shrunken; *I*, iris; *S*, sclerotic; *Ch*, chorioid; *R*, retina much folded; *O.n.*, optic nerve; *Cy*, cysts lined by retina.—D.]

With the ophthalmoscope the blood-vessels of the retina and chorioid are seen with perfect distinctness running upon the almost white fundus, to which the papilla by its dark, grayish-red color offers a striking contrast (Figs 25 A, and 27). Albinotic eyes are photophobic, and hence see better in the dusk; their visual acuity is always reduced; and nystagmus, frequently combined with a pretty high degree of myopia or with strabismus, is constantly present. Albinism is congenital and often inherited. In albinotic eyes the pigmentiferous cells of the uvea and the retina are present, just the same as in the normal eye, only they contain [little or] no pigment. All sorts of transition forms exist between complete albinism and normal pigmentation. [In infants large areas of the fundus may be albinotic. Such areas may alternate with quite sharply defined islands of normally pigmented chorioid. The albinotic areas are found particularly in the periphery of the fundus, and this tendency to semi-albinism in the periphery may persist even in adult life.—D.]

CHAPTER VII

GLAUCOMA

General Considerations

438. Nature.—The essence of glaucoma lies in the *increase in the intraocular pressure*, from which all the other essential symptoms of glaucoma can be deduced (Von Graefe). In one series of cases the increase in pressure sets in without our being able to discover any reason for it in an antecedent disease of the eye (*primary glaucoma*). In other cases, on the contrary, the increase in pressure is the result of some other disease of the eye (*secondary glaucoma*). Primary glaucoma, accordingly, has increase in tension as its first and most important symptom, from which all the rest of its phenomena arise—it is glaucoma proper, the glaucoma par excellence. In secondary glaucoma, on the contrary, the increase in tension is only a consequence of other pathological conditions—is an accessory, as it were. The clinical picture of secondary glaucoma, therefore, is exceedingly polymorphous, varying according to the different affections which form the basis of it. While genuine or primary glaucoma always affects both eyes, although not always at the same time, secondary glaucoma remains confined to the eye which, by being diseased, has caused the increase in tension. The diagnosis of glaucoma is based mainly on the demonstration of its cardinal symptom, increase of tension, by palpation with the finger or by the tonometer (page 83).

Primary glaucoma is a common disease, constituting about one per cent of all cases of eye disease. Its accurate recognition is of the greatest importance for the general practitioner, the more so because here prompt and proper therapeutic interference can save everything, but a false diagnosis and improper treatment may destroy everything. Unfortunately, we still constantly get under observation many cases of glaucoma which have been incorrectly diagnosticated by the general practitioner, and which come to the ophthalmologist only when help is no longer possible. Cases of inflammatory glaucoma are often confounded with iritis or irido-cyclitis, and are accordingly treated with atropine, which has a particularly deleterious action in glaucoma. Cases of glaucoma simplex which present no external symptoms of inflammation are not infrequently regarded as commencing cataract, and the patients are put off in expectation of the cataract's becoming ripe, so that they delay until it is too late for iridectomy.

Glaucoma has been known from antiquity. Of course, it is only the inflammatory variety that has been thus known, since the non-inflammatory variety can be diagnosticated only by the ophthalmoscope. Hence this second variety and the other kinds of blindness, that are produced by diseases of the deeply situated membranes of the eye, and that have no external manifestations, were lumped together under the common name of amaurosis. Inflammatory glaucoma was usually regarded as having a connection with gout, and was hence called ophthalmia arthritica. The first to recognize

the increase in tension as the most important symptom of glaucoma were Mackenzie, and particularly Von Graefe. Heinrich Müller, a man deserving the greatest praise in all that relates to the pathological anatomy of the eye, was the first to demonstrate anatomically the pressure excavation of the optic nerve (1856); soon afterward it was accurately diagnosticated in the ophthalmoscopic picture by Weber and Förster. Mackenzie, starting from the fact of the increase of tension, had already attempted to effect the cure of glaucoma by repeated paracentesis of the cornea, without, however, obtaining any lasting result. Such a result was first obtained by Von Graefe, who, in the year 1856, employed iridectomy for the first time in glaucoma, after having found it efficacious in various other diseases of the eye. This was one of the most pregnant discoveries in ophthalmology, and one which will for all time redound to the glory of Von Graefe. We have only to remember that formerly every case of glaucoma inevitably led to blindness, and that now, thanks to iridectomy, the majority of glaucomatous patients can be cured. How many thousands there are who formerly would have been forced to sink year by year irretrievably into the night of blindness, but who now are saved for vision through Von Graefe's discovery!

439. Consequences.—The consequences of increase of tension, inevitably occurring if it lasts a long time, are excavation of the optic nerve, and reduction, with ultimate annihilation, of the sight.

Excavation of the optic nerve is dependent upon the recession of the lamina cribrosa. By the latter we understand that part of the sclera which lies at the point of entrance of the optic nerve into the eye, and which is perforated by numerous foramina designed for the passage of the bundles of fibers of the nerve (Figs. 264 and 267). The lamina cribrosa is that part of the fibrous tunic of the eye (corneo-sclera) which has the least tenacity, and hence gives way first to increased ocular pressure, which it does by bulging backward. But at the same time the optic-nerve fibers, set in the foramina of the lamina cribrosa, also recede, so that the surface of the optic nerve itself sinks back (*e*, Fig. 213, B). Upon ophthalmoscopic examination the papilla appears depressed below the level of the adjacent fundus—slightly at first, afterward a good deal—so that the margins of the papilla dip down abruptly, or are even overhanging. This condition is recognized chiefly by the bending or actual interruption of the blood-vessels at the spot where they pass from the retina over the edge of the papilla and dip down into its depth (Fig. 213, A). The nerve fibers, too, like the blood-vessels, undergo flexion or interruption at the edge of the papilla. This interruption, with the high pressure to which the nerve fibers are exposed within the eye, makes them atrophy. Accordingly, in the later stages, we see the papilla not only depressed, but bleached and bluish-white, because the nerve fibers are destroyed and the clear white lamina cribrosa is exposed.

With reference to *excavation of the optic nerve*, three varieties are distinguished—the physiological, the atrophic, and the glaucomatous. The *physiological excavation* (Fig. 214, A) originates from the fact that the bundles of fibers of the optic nerve, when separating from each other so as to curve into the retina, do so, not in the plane of the

retina, but behind it; the lamina cribrosa, however, is in its normal situation. The physiological excavation is always partial—i. e., even if it is very large it never takes up the entire papilla, because a certain space close to the edge of the papilla must always be occupied by the nerve fibers which are passing over into the retina (page 99, Fig. 24, *G*, and Figs. 20, 21, and 200). The *atrophic excavation* (Fig. 214, B) is caused by the disappearance of the nerve fibers that form the papilla of the optic nerve and lie in front of the lamina cribrosa—the lamina cribrosa itself remaining in place. The atrophic

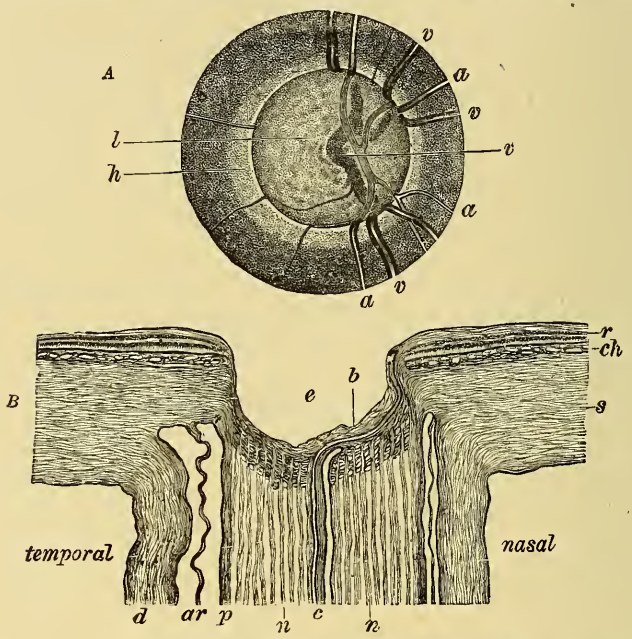


Fig. 213.—Glaucomatous Excavation of the Optic Nerve. Magnified 14 × 1. Cf. the normal optic nerve in Figs. 22, 23, 24.

A. Ophthalmoscopic Picture of the Papilla.—The papilla is bounded by a sharp, overhanging edge, at which the arteries, *a*, and the veins, *v*, of the retina appear to terminate in recurved ends. This is due to the fact that their continuation on the floor of the excavation is displaced somewhat laterally as compared with the portion situated in the retina, because those portions of the vessels that descend on the lateral wall of the excavation do not run in a precisely sagittal direction, but somewhat obliquely. The vessels within the excavation are seen but indistinctly. In the outer half of the excavation are seen the gray dots of the lamina cribrosa, *l*. The zone, *h*, of the fundus, adjoining the papilla, is decolorized (halo glaucomatosus).

B. Longitudinal Section Through the Head of the Optic Nerve.—This shows a deep excavation, *e*, on the floor of which only a few remains of the nerve fibers, *b*, are visible. The central vessels, *c*, ascend upon the retina, *r*, at the nasal margin of the excavation; the innermost layer (fiber layer) of the retina is considerably diminished in size through atrophy. *ch*, chorioid; *s*, sclera. The volume of the trunk of the optic nerve has been considerably reduced through the atrophy of the bundles of nerve fibers, *n*. As a result of this, the interspaces between the sheaths of the optic nerve (the pial sheath, *p*, the arachnoid sheath, *ar*, and the dural sheath, *d*) are dilated, especially on the temporal side.

excavation is total—i. e., extends over the entire papilla, but always remains shallow, for at most it can only attain a depth equal to that at which the lamina cribrosa lies behind the inner surface of the retina. In the atrophic excavation, the papilla is at the same time bleached white on account of the disappearance of the nerve fibers. The physiological and the atrophic excavations have this in common, that the lamina cribrosa remains undisplaced; as the lamina cribrosa forms the floor of the excavation the depth of the latter is confined within narrow limits. The *glaucomatous excavation* (Fig. 214, C) is distinguished from the preceding kinds, above all, by the fact that it

originates in a recession of the lamina cribrosa; it can hence attain a much more considerable depth than they. The glaucomatous excavation comprises the entire papilla, which, in the beginning, still shows the red coloration of health. Later on, the nerve bundles are destroyed by atrophy, so that the papilla becomes white, and displays exposed upon its floor the lamina cribrosa. With this is associated a still further increase in the excavation, the depth of which is increased by a space equal to the thickness of the nerve head which has been destroyed.

The ophthalmoscopic signs distinguishing the three kinds of excavation are, therefore, as follows: A partial excavation is physiological, a total one is pathological, and either atrophic or glaucomatous. The atrophic excavation is shallow, and the papilla, at the same time, very white. The glaucomatous excavation may be shallow or deep, according as it has existed for a longer or shorter time. In a shallow glaucomatous excavation the papilla is found to have still a good color—a feature which distinguishes it from the atrophic excavation. If the excavation is deep and total, it can only be a glaucomatous one, whatever color the papilla may have. In practice, the distinction between the individual forms of excavation is sometimes very difficult to make [particularly so, as the physiological excavation is sometimes very deep as well as large, and is often overhanging.—D.]

The *ophthalmoscopic picture* of a glaucomatous excavation of the optic nerve shows the papilla to be paler, and, in advanced cases, bluish or greenish white. A shadow is seen running along the margin, while the center of the excavation is the part lightest

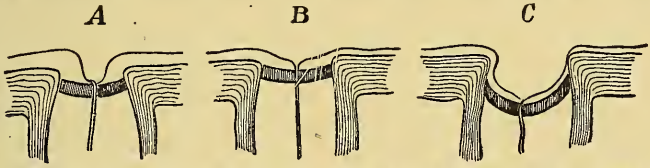


Fig. 214.—The Three Kinds of Excavation of the Optic Nerve. Schematic.
A, Physiological Excavation.—Funnel-shaped, partial, with normal lamina cribrosa.
B, Atrophic Excavation.—Bowl-shaped, total, with normal lamina cribrosa.
C, Glaucomatous Excavation.—Ampulliform, total, with the lamina cribrosa bulged out posteriorly.

in hue. On the floor of the excavation may be recognized the gray dot-marks of the lamina cribrosa (Fig. 213 A, *l*). The vessels do not emerge at the center, but, for the most part, close to the inner margin of the papilla. Where they pass over the edge of the papilla to go to the retina they show a bending, or, in deep excavations, an interruption, of their course. If the edge of the excavation is overhanging, the ascending portion of the vessel may be completely concealed behind it, so that the blood-vessels arising from the vascular entrance seem to disappear at the edge of the papilla to emerge again in the retina at some other spot. It is only in the inverted image that the course of the vessel can be seen distinctly in its whole extent at once; in the erect image, the vessels on the papilla and those in the retina are never seen clearly at the same time, since they lie at different depths and hence have a different refraction. That is, if the adjustment is made for the vessels in the retina (Fig. 213 A, *a* and *v*), the vessels on the floor of the excavation (v_1) look quite pale and hazy, and vice versa. The vessels in the excavation have, as compared with those in the retina, a myopic refraction, and hence, to be seen distinctly, require a correspondingly strong concave glass. From the difference in refraction between the margin and the floor of the excavation, the depth of the latter can be estimated (see page 112), and by repeated measurements of this sort we can determine whether, as time goes on, the depth of the excavation is increasing or diminishing. In the inverted image, the difference of level manifests itself by parallactic displacement (page 112). The caliber of the arteries is contracted, while the

veins are distended and tortuous—in fact, sometimes there is a whole convoluted mass of vascular loops lying on the floor of the excavation. These changes of vascular caliber are easily accounted for by the effect which the increase of pressure exerts upon the vessels of the vascular entrance, permitting less blood to enter the arteries of the retina and, on the other hand, obstructing the outflow of blood from the veins. The former therefore, are filled too little, the latter are filled too much. We very often observe a pulsation in the veins, and not infrequently, also, a pulsation in the arteries within the papilla. (For the explanation of this, see page 100.) When glaucomatous excavation has lasted for a long time, the papilla is usually found to be surrounded by a white or yellowish areola, which is the expression of an atrophy of the chorioid about the papilla (halo glaucomatosus, Fig. 213 A, *h*). The rest of the fundus in the later stages often shows the network of chorioidal vessels with great distinctness (Fig. 201) because of the diminished pigmentation of the pigment epithelium.

440. Impairment of Vision.—The atrophy of the optic-nerve fibers is also the most important cause of the *decrease of visual power* which accompanies the elevation of tension. Impairment of both direct and indirect vision occurs. The former finds expression in the gradual diminution of acuity of central vision, the latter in the limitation of the field, which begins, in the majority of cases, on the nasal side, as the temporal side of the retina becomes insensitive first. Finally, complete blindness supervenes.

The condition of the *sight* is not always in direct proportion to the depth of the excavation—in fact, it is not the recession of the lamina cribrosa as such that affects the vision, but the atrophy of the optic nerve, which, though produced by it, does not always by any means keep pace with the formation of the excavation. Thus we sometimes see cases with deep excavation and yet with normal visual acuity and a large field of vision. On the other hand, by a very considerable increase in pressure—as in glaucoma fulminans—the sight may be completely extinguished within a few hours by paralysis of the optic-nerve fibers without there being any excavation of the optic nerve, because the time is too short for it to be formed. Even in chronic glaucoma it is sometimes the case that the papilla atrophies and complete blindness actually ensues as the result of the increase of pressure before—sometimes even years before—an obvious glaucomatous excavation is formed. Accordingly, in forming a judgment as to the acuity of vision, we must be guided rather by the color of the papilla and the caliber of the retinal arteries than by the depth of the excavation, since atrophy of the nerve fibers makes itself known mainly through the pallor of the papilla and the narrowing of the retinal vessels.

The contraction of the *visual field* begins most frequently on the nasal side. A very frequent [according to some, constant] change is an up-and-down elongation of the blind spot, so that a sickle-shaped scotoma is produced [Bjerrum] arching above or below round the fixation point and even reaching the horizontal meridian on the other side of the latter (Rönne). In this way an annular scotoma may ultimately develop. In glaucoma simplex central scotomata may occur, so that with a large field of vision the central visual acuity is impaired early. The reverse also occurs, i.e.,a marked concentric contraction with good central vision. [For making out the enlargements of the blind spot and the central scotomata, Bjerrum's screen (Fig. 34) is very useful. I have gotten the best results by placing the patient 1.5 metres from the curtain, and using white test-objects varying from 2 to 7 mm. in diameter. The double-object test and, in the case particularly of central scotomata, the complementary-color test or homonymous-color test (page 124), are often very useful.—D.]

I. Primary Glaucoma

441. Varieties.—Primary glaucoma, also called simply glaucoma, sets in with varying symptoms. If the pressure rises suddenly to a considerable height, inflammatory symptoms are excited; on the contrary, these symptoms are wanting when the increase in tension develops gradually and keeps within narrow limits. Accordingly, an inflammatory and a non-inflammatory form of glaucoma (glaucoma inflammatorium and glaucoma simplex) are distinguished.

[Since the symptoms in the acuter form are those of congestion and œdema rather than of inflammation, it seems better to designate the two varieties by the names given by Elliot, namely, *congestive* and *non-congestive* glaucoma.—D.]

A. Glaucoma Inflammatorium [*Congestive Glaucoma*]

442. Inflammatory glaucoma runs a typical course, especially in the acute cases (glaucoma inflammatorium acutum), which, therefore, will be first described. In the course of inflammatory glaucoma the following stages are distinguished:

(1) **Stage of Prodromes.**—The prodromal stage, which in most cases precedes the inflammatory attack, is characterized first of all by attacks of obscuration of vision. The patient declares that during these attacks he does not see as well, having at the same time the feeling as if there was a cloud or smoke concealing objects from him. If there is a light in the room, he sees a ring about it having the colors of the rainbow. During the attack there is frequently a feeling of tension in the eye, or a dull frontal headache. If the physician examines the eye during such an attack, he finds the cornea a little dull and diffusely clouded, like glass that has been breathed upon. The cloudiness is greatest at the center, smallest at the periphery, and, on account of its uniformity of distribution, causes considerable disturbance of vision. It also produces the appearance of a colored ring about a luminous flame—an appearance similar to that which, for instance, we see when, on a misty night, we look at a gas flame in the street. The anterior chamber is somewhat shallower through advancement of the iris; the pupil is more dilated than usual and reacts sluggishly; the tension of the eye is distinctly increased. Frequently, too, slight ciliary injection is present.

Such an attack ordinarily lasts several hours, after which the eye returns completely to the normal condition once more, both as regards its appearance and as regards its function. The attacks at first make their appearance at pretty long intervals (of months or weeks); later on, they become constantly more frequent. Often specific causes for their existence can be demonstrated, such as hearty meals, late hours, emotional excitement (as at card-playing), etc. In many cases they return, even without cause, periodically, sometimes even every day, so that the patient, for example, declares that he always sees through a cloud in the morning, and com-

mencing with the afternoon begins to see clearly, or vice versa. If the attacks come on in the evening, they always cease when the patient falls asleep; even in the daytime an attack may be cut short by his going to sleep.

In the intervals between the prodromal attacks the sight of the eye is normal; but the patient complains that, to see near by, he has to employ stronger and stronger glasses—rapid increase of presbyopia through diminution of the power of accommodation (see § 796).

The prodromal stage sometimes lasts only a few weeks, sometimes is protracted over months or even years. In the latter case, the eye gradually undergoes permanent changes, so that it is no longer normal even in the intervals between the attacks. The eye acquires externally the glaucomatous aspect, and an excavation likewise forms in consequence of the oft-repeated increase of pressure. Consequently the sight itself is no longer perfect even during the time in which no attacks occur. In such a case we can no longer speak of the disease being in the prodromal stage, but must regard it as being now a chronic congestive glaucoma into which the prodromal symptoms have been imperceptibly transformed.

Although congestive glaucoma is so well characterized a disease, it is still very often diagnosticated too late. In the *prodromal stage* the diagnosis is obscured because the physician usually sees the patient in the interval between the attacks, when the eye looks normal both exteriorly and with the ophthalmoscope. [The tension also may be normal, but repeated tests should be taken to see if at some time it does not rise above the normal limit.—D.] The physician is, therefore, dependent for his diagnosis upon the description that the patient gives of his attacks. These are characterized by disturbance of sight (hazy vision, the appearance of colored rings about a light) and by headache (page 45). On the other hand, we cannot with certainty make the diagnosis of glaucoma even when the patient makes the direct statement that he has hazy vision and sees colored rings. For, these two sorts of disturbance of sight occur as a result of any diffuse cloudiness of the media and are hence not confined to glaucoma. For instance, they occur in chronic conjunctivitis when some mucus lies on the cornea. But in this case the phenomenon vanishes at once when the mucus is wiped off by rubbing the eye. Many persons, again, under appropriate conditions (e. g., if a flame is placed in front of a dark background in a darkened room) always see a colored areola. In this case we have to do with faint diffuse opacities, chiefly in the lens, which are commonly too minute to be demonstrated objectively. These cases also are easily distinguishable from glaucoma, for, on the one hand, the phenomenon is far less pronounced than it is in glaucoma, and, on the other hand, does not occur in periodical attacks, but can be elicited at any moment.

Accordingly, in cases in which no changes in the eye are objectively discoverable we cannot make the diagnosis of glaucoma with certainty simply from the description that the patient gives of his attacks—not with enough certainty, for instance, to make us willing to suggest an iridectomy on the strength of this description alone. We must require that we ourselves see the patient during a prodromal attack, in order to demonstrate the visible objective changes occurring in the course of it. We hence charge the patient to present himself without delay as soon as he is again attacked with disturbance of sight or with headache. If for extrinsic reasons this in not possible, we may use pilocarpine (or eserine) for making the diagnosis more certain, the patient being told to drop this in at once on the occasion of the next attack. If the morbid symptoms are completely relieved by this means in from ten to fifteen minutes, it is an argument

in favor of glaucoma, for miotics have no effect on non-glaucomatous forms of cloudiness of the media nor on headaches, that are of a different nature. [Miotics may occasionally relieve a headache, especially one of migrainous type, due to other causes than glaucoma.—D.]

[Another way of determining the presence of glaucoma in a suspected case is to instil a mydriatic (Jackson). One of comparatively transient action (euphthalmine or ½-per-cent homatropine) should be used, the tension being first taken with the tonometer. If the mydriatic determines an increase of tension, and still more if it determines a glaucomatous attack, the diagnosis is assured. In either case eserine should be instilled without delay, and the instillation repeated until the pupil is contracted—the patient being kept under the physician's observation continuously until this occurs. If this precaution is observed there is little risk in the procedure, which is the more justifiable since an eye that would develop a glaucomatous attack under these conditions would quite surely develop an attack spontaneously later, and perhaps, too, when the disease had made greater inroads on the sight and the field of vision.—D.]

443. (2) **Glaucoma Evolutum.**—This is ushered in by an *attack of acute glaucoma.* This sets in suddenly, after the prodromal stage has lasted a longer or shorter time. The cause of an acute attack—in case such a cause is discoverable at all—is like those which determine the prodromal attacks. Chief among them are to be mentioned states of congestion of the venous system, especially those due to enfeeblement of the heart's action; also mental emotions, particularly those of a depressing character; and, lastly, dilatation of the pupils. For the last-named reason a drop of atropine in an eye which is predisposed to glaucoma may excite an attack.

The acute attack manifests itself by violent pain radiating from the eye along the first and second branches of the trigeminus. The patient complains of pains in the head, the ears, and the teeth, which may reach an intolerable pitch. They deprive him of appetite and sleep; not infrequently vomiting and fever likewise set in. Simultaneously with the appearance of the pain the visual power falls rapidly away, so that only large objects—such as, for instance, the hand moved to and fro before the eye—can be recognized. The field of vision is considerably narrowed, and mostly on the nasal side. Objective examination shows the appearances of a violent external inflammation—œdema of the lids, and œdema or even chemosis of the conjunctiva, which is greatly congested. The injection, in accordance with its pre-eminently venous character, has a dusky-red color. The cornea is punctately dotted, has a pronounced smoky cloudiness, and is almost or quite insensitive to the touch. The anterior chamber is shallower, the iris is discolored and narrowed. Consequently, the pupil is dilated; very often, too, it is oval and eccentrically situated, the narrowing of the iris being then particularly great in special spots—most frequently above. The reaction of the iris is abolished. From the pupil we get a grayish-green reflex.[1] Ophthalmoscopic examination is impossible, on account of the marked cloudi-

[1] Hence the name "green cataract" (*grüner Staar*). In Greek, sea-green is called γλαῦκος, whence glaucoma. This reflex, however, is by no means distinctive of glaucoma. It is always found when the pupil is dilated, and at the same time the media are not completely tr. nsparent, e. g , in eyes with the ordinary senile reflex. Hence an old man's eye, when atropinized, looks on superficial examination very like a glaucomatous eye.

ness of the cornea. The tension of the eye is considerably elevated.

It may be seen that the symptoms of the acute attack are the same as those belonging to the prodromal attack, except that they are much more pronounced and are accompanied by inflammatory symptoms (injection, œdema of the lids and conjunctiva, and pain). The prodromal attacks may therefore be regarded in the light of abortive attacks of glaucoma, which retrocede before they have developed to their full height. But at length an onset of this sort takes place, that rises to the height of an acute attack, and after this a perfect return to the normal is no longer possible. The tension now remains permanently elevated, and the eye retains the glaucomatous aspect.

The *course* of the attack of congestive glaucoma is that after some days or some weeks—according to the severity of the attack—improvement, or even an apparent cure, sets in. After some days the pain diminishes in violence and afterward disappears altogether. The eye becomes free from discoloration, the cornea clears up, and the sight becomes better again. If the sight was still normal before the attack, it may improve to such an extent that the patient can still read and write; but the more the sight has been injured before the attack by a prodromal stage of long duration, the less is its improvement after the decline of the attack. We may say in general that after an attack has passed off, the vision never again reaches the same height that it had before the attack occurred. The attack, furthermore, leaves behind it objective changes in the eye, that give at the first glance proof of the sort of disease that is present. The over-distention of the anterior ciliary veins remains; the anterior chamber is shallower, and the iris is narrower, is turned to a slate-gray color, and reacts sluggishly or not at all; the tension is permanently increased. We then say that the eye exhibits the glaucomatous aspect (habitus glaucomatosus). Ophthalmoscopic examination, which becomes feasible again after the cornea has cleared up, shows at the optic-nerve entrance the signs of hyperæmia which really is only one of the evidences of the general hyperæmia that was present during the congestive attack. The excavation of the optic nerve is not present directly after the attack, because for its formation quite a long period of increased tension is requisite; it hence does not develop until later on. It is only in those cases in which there has been a long preceding stage of prodromes that the excavation is present during the attack.

After the subsidence of the attack the eye remains quiescent for quite a long time, and the patient entertains the hope of a permanent cure. Then a new attack sets in. This, as far as inflammatory symptoms and pain are concerned, is usually less intense than the first, but results in a still further reduction of the sight. Inasmuch as new attacks now constantly follow each other, at shorter or longer intervals, the sight at length becomes entirely extinct. The disease has then entered upon the third stage (§ 444).

The *acute congestive attack* is often misunderstood. In this the *pain* radiates from the eye into the whole of one side of the head, so that sometimes the patients are not for a moment aware that the pain is proceeding from the eye, but complain simply of violent "rheumatic" headache. If the attack has been accompanied by marked swelling of the lids, a history of erysipelas may be given. One should not allow himself to be led astray by such declarations as these, but should form his decision in accordance with the results of the objective examination. The principal thing that this shows us is the dull and uniformly cloudy look of the cornea. In former times this cloudiness was thought to be located not only in the cornea, but also in the aqueous humor and in the vitreous. As to the cloudiness of the vitreous, no proof whatever of it has been presented. An argument for the existence of cloudiness in the aqueous is the fact that not infrequently after it has escaped (in the performance of iridectomy), the pupil looks blacker than before. The main cloudiness, however, is situated in the cornea. Another important symptom of glaucoma is the dilatation and rigidity of the pupil, and by this we are guarded against any confusion with iritis or irido-cyclitis, since in these diseases the pupil is contracted.

A characteristic feature of glaucoma is the frequent, often periodical, *alternations in the course* of the disease. These are afforded by the transient obscurations of vision occurring in the prodromal stage, by the inflammatory attacks recurring at intervals in the inflammatory stage; even after the blindness is complete, there is still a constant alternation of bright and dark days, according to the patient's subjective luminous impressions.

The *character of the pupil* has a very great influence on the phenomena of glaucoma. Its contraction has a favorable effect, since it generally diminishes the tension in glaucoma, while dilatation of the pupil, on the contrary, increases the tension. Hence miotics have the power of cutting short the prodromal attacks, and of ameliorating the symptoms even in the inflammatory attack. The fact that the prodromal attacks can be cut short by the patient's going to sleep is probably also referable to the marked contraction which the pupil undergoes in sleep. Mydriatics, on the contrary, can excite an inflammatory attack in an eye that is predisposed to it, and that not only the powerful mydriatics, like atropine, but also homatropine [euphthalmine], cocaine [and, according to some, even dionine and holocaine]. We should always, therefore, take care that there is no suspicion of the existence of glaucoma, before making an instillation of a mydriatic in the case of an elderly man. If, however, we have had the misfortune to set up in this way an attack of inflammatory glaucoma, it may be possible, by the prompt and energetic instillation of eserine, to cause the attack to disappear, and that perhaps permanently.

[Exclusion of light by bandaging the eye or in other ways may also by inducing a dilatation of the pupil set up an attack of glaucoma (Elliot).—D.]

[Those who hold that the ciliary muscle promotes outflow from the eye (see pages 15 and 371), believe that a glaucomatous attack may be set up by inaction of this muscle and be prevented by its exercise (e. g., by reading, especially in a strong light) (Elliot).—D.]

It is a fact frequently observed that iridectomy, done in a glaucomatous eye, may give rise to a congestive attack in the second eye, if it is already predisposed to glaucoma. Nevertheless, it is not the operation as such, but the associated mental and physical depression which, as on other occasions, may here, too, excite a glaucomatous attack. The operation itself is not needed to produce this result. One day a lady came to me with a recent congestive attack in both eyes. She had a few days before had her first attack of glaucoma in the right eye, and had consulted Prof. Arlt for it. When he explained to her that an operation was necessary, she experienced such a violent fright that she got a congestive attack in the second eye while returning from the consultation in her carriage. Probably there are two factors that act together in the case of glaucoma produced by violent emotion—the disturbance of the circulation and the reflex dilata-

tion of the pupil. In an operation upon a glaucomatous eye, pilocarpine or eserine may be instilled into the second eye to prevent the outbreak of glaucoma in it; even this precautionary measure, however, does not afford complete security.

Febrile diseases of various sorts likewise determine an attack of glaucoma—a fact that has been observed quite often, especially in the great influenza epidemics.

[Injuries, such as blows on the head, abrasions and burns, or corrosive injuries near the limbus, etc., may set up an attack of glaucoma, usually transient (Kuemmell, Elliot).—D.]

444. (3) **Glaucoma Absolutum.**—In this, the third stage of glaucoma, the eye is completely blind, and presents the following picture: Contrasting with the porcelain-like, bluish-white sclera are the distended anterior ciliary veins, which unite round the cornea to form a bluish-red circle of dilated vessels. The cornea is shining and transparent, but insensitive; the anterior chamber is very shallow. The iris is reduced to a narrow gray marginal strip, which in places is almost entirely concealed by the limbus, and which at its pupillary margin is encircled by a broad black border. The dilated and rigid pupil is greenish, or of a dirty gray. The optic disk is deeply excavated, the eye as hard as stone.

Later on, degenerative changes make their appearance in the blinded eye, which are designated under the name of *glaucomatous degeneration*. The cornea becomes cloudy and covered with peculiar glassy-looking deposits. Upon the sclera dark ectatic prominences—most frequently in the region of the equator (equatorial staphylomata)—present themselves to view; the lens becomes cloudy (cataracta glaucomatosa). Although the eye has now been blind for a long time, the patient still constantly believes that he perceives light, especially under the form of a luminous haze, which on some days is more pronounced, on other days less so. These subjective luminous appearances for a long time uphold the patient in the belief that he will be able to regain his sight. Furthermore, attacks of pain keep making their appearance at intervals in the blinded eye.

The final outcome in glaucoma is usually atrophy of the eyeball. After the eye has been hard for years it at length becomes softer, smaller, and atrophic. In other cases, ulcus serpens develops with perforation and consecutive irido-cyclitis or even panophthalmitis, together with phthisis bulbi. Not until the glaucomatous eye has become shriveled does it allow its unfortunate possessor to have any lasting rest.

In the stage of glaucomatous degeneration various changes are observed in the *cornea*. (*a*) Most frequently together with marked cloudiness of the corneal parenchyma there are found gelatinous or hyaline deposits upon the surface of the cornea. (*b*) Vesicles develop upon the cornea, owing to the fact that the epithelium, either by itself or along with the newly formed deposits, is lifted from its bed by serous exudation—keratitis vesiculosa and bullosa (page 286 and Fig. 105). (*c*) Zonular opacity of the cornea (page 310), and (*d*) ulcers of the cornea, frequently leading to perforation (page 261), are observed. As a result of the latter (especially ulcus serpens) violent hæmorrhages may take place from the interior of the eye (page 472), or severe purulent inflammation succeeded by shrivelling of the eyeball. These affections of the cornea, so various in

character, are referable in part to the alteration in nutrition produced by the disturbance in the lymphatic circulation, partly to the paralysis of the corneal nerves, manifested in the complete insensitiveness of the surface of the cornea to touch. In consequence of these changes, the cornea becomes less able to resist external injuries.

In the course of glaucomatous degeneration the *lens* always becomes cloudy (*cataracta glaucomatosa*). From this lenticular cloudiness, produced by the glaucomatous process, is to be distinguished that form which occurs only as an accident in the glaucomatous eye, and which is designated as *cataracta in oculo glaucomatoso*. Thus, simple senile cataract, traumatic cataract, etc., may be present in a glaucomatous eye. The distinction between cataracta glaucomatosa and cataracta in oculo glaucomatoso is made by observing the appearance of the cataract and by testing the vision. Cataracta glaucomatosa is distinguished by its marked distention, its bluish-white color, and the vivid silky luster of its surface, while cataracta in oculo glaucomatoso has the appearance corresponding to its origin and its nature. In the former kind of cataract the eye, as a result of the glaucomatous process, is completely blind, and hence an operation for cataract is useless. In the second case, if the glaucoma is not too far advanced, such a degree of sight (perception of light) may still exist as promises a good result from the extraction of the cataract. The extraction, however, should in no case be performed immediately; on the contrary, the increase of tension should first be done away with by an iridectomy, and this may be succeeded, say some four weeks later, by the cataract operation. If we should extract a lens, without taking any such preliminary precaution, in an eye affected with increase of tension, we would run the risk of losing the eye through severe intra-ocular hæmorrhage (see page 256).

445. Varieties in Course.—The course of glaucoma with a violent attack of congestion, here pictured, corresponds to that form which is designated as *glaucoma inflammatorium acutum*. In the severest cases, which Von Graefe has described under the name of *glaucoma fulminans*, incurable blindness, in conjunction with the most violent symptoms of inflammation, may develop within an few hours. Of much more frequent occurrence than these cases, which luckily are rare, are those which run a less acute and typical course than belongs to acute glaucoma. These cases are designated as *glaucoma inflammatorium chronicum*. Here no pronounced congestive attack takes place; on the contrary, the prodromal stage passes imperceptibly into the stage of inflammation, the eye growing gradually red and sensitive, the cornea acquiring a smoky cloudiness, the iris becoming atrophied. The pain is neither so violent nor so continuous as in acute glaucoma. Often this sort of chronic course sets in after the first congestive attack, no complete subsidence of the symptoms of inflammation taking place. The final outcome is the same as in acute congestive glaucoma; and, anyhow, no sharp line of distinction exists between the two forms.

Glaucoma almost always attacks both eyes. Nevertheless, they are but rarely affected at the same time; it more frequently happens that the disease of the second eye follows months, or even years, after that of the first. The experienced physician, however, even in those cases in which one eye is still perfectly healthy, will frequently recognize in the latter a certain *disposition toward glaucoma*. This manifests itself in a shallow anterior

chamber, a somewhat dilated and sluggish pupil, and a comparatively high, even though not yet pathological, tension of the eye. With this there exist, usually, a hypermetropic state of the refraction and diminution of the range of accommodation.

[The eye often is small and the cornea therefore narrower than usual. In patients coming from a suspected stock, these features may be regarded as danger signs (Priestley Smith).—D.]

Congestive glaucoma is usually a disease of advanced life; it is found most frequently between the fiftieth and seventieth year. It never occurs in childhood and but very rarely indeed in youth. It attacks women [about twice as frequently as] men, especially women in whom the menopause occurs before the time. A *predisposition* to congestive glaucoma appears to belong particularly to hypermetropic eyes, highly myopic eyes having almost complete immunity against this disease. [Myopes enjoy only a relative immunity. Thus of 115 cases of congestive glaucoma examined by Gilbert, 77 per cent were hyperopes, the remaining 23 per cent being equally divided between myopes and emmetropes (Elliot). See also page 497.—D.] Furthermore, rigidity of the vessel walls (arteriosclerosis) and habitual constipation predispose to glaucoma. Again, among Jews, [Egyptians, and certain varieties of negroes] congestive glaucoma is much more frequent than among other races. Moreover, there are many families in which glaucoma is inherited.

[Familial glaucoma, according to Lawford, is continuous in descent, is transmitted by both sexes, exhibits the phenomena of anticipation, and occurs in all forms (Elliot). —D.]

B. Glaucoma Simplex [*Non-Congestive Glaucoma*]

446. In non-congestive glaucoma the increase of tension sets in very gradually, so that no inflammatory phenomena are produced. The eye either looks quite normal externally, or it gives evidence of the lesion affecting it in the greater prominence of the distended anterior ciliary veins, and also in a somewhat dilated and sluggish state of the pupil. The tension of the eye is shown to be elevated, but usually not to any considerable degree. Often on the first examination no increase of tension is found at all; it is not until we examine the eye repeatedly, and especially at different times of the day, that we succeed in demonstrating that the tension is elevated. At these times a slight, smoky cloudiness of the cornea is also sometimes present, such as appertains to the prodromal attacks of inflammatory glaucoma. Finally, there are cases of non-congestive glaucoma in which the tension is so low all the time that on palpation with the finger it is regarded as normal, while, nevertheless, examination with the tonometer shows a very slight but still certain elevation above the normal. In a few cases, it must be admitted, no tension surpassing the normal can be demonstrated even with the tonometer.

In consideration of the fact that in non-congestive glaucoma marked external symptoms, and sometimes, indeed, even any increase of tension, are wanting, we are thrown back upon the ophthalmoscopic examination for the establishment of the diagnosis. Such an examination shows the presence of a total excavation of the optic nerve, the depth of which corresponds to the duration of the process.

The subjective symptoms of non-congestive glaucoma, since the inflammatory attacks and the pain are wanting, consist almost exclusively of the disturbance of vision. This manifests itself by gradual diminution of the sight, and in many cases also by transient slight attacks of obscuration, like those belonging to the prodromal stage of inflammatory glaucoma. The decrease in vision expresses itself in a contraction of the field of vision, as well as in a diminution of the central visual acuity. The diminution in central vision often develops late, when the field of vision has already become very small, so that not infrequently the patients are still in a condition to read or carry on fine work, while they are scarcely able to go about any longer alone (see page 114). Before this point is attained, generally a long time (even many years) has elapsed, inasmuch as the reduction of vision takes place very slowly and gradually. For this reason the patient himself often does not become aware of the existence of his disease until late. The blinded eye may either remain always sound externally, or those inflammatory attacks which are characteristic of congestive glaucoma may occur—often, indeed, before the blindness has become complete. Non-congestive glaucoma, accordingly, not infrequently changes into congestive glaucoma.

Non-congestive glaucoma always attacks both eyes. In contradistinction to the congestive form, it sometimes occurs in young people, and attacks men as frequently as women. [Again it is found as often in myopic eyes as in those that are emmetropic or hyperopic (Gilbert and others cited by Elliot), whereas congestive glaucoma is found particularly often in hyperopia (see page 496).—D.]

The *relation of non-congestive to congestive glaucoma* has been the subject of manifold discussions. Since the former, on account of the absence of inflammatory symptoms, is entirely different externally from congestive glaucoma, it was not recognized as glaucoma at all until the discovery of the ophthalmoscope. Even Von Graefe did not originally place non-congestive glaucoma under the head of glaucoma, but called it amaurosis with excavation of the optic nerve. Jäger held to this view to the last, looking upon non-congestive glaucoma as an optic-nerve lesion sui generis—a "glaucomatous" optic-nerve lesion. [Schnabel holds a somewhat similar view, contending that the cupping in glaucoma is due to an active atrophy (cavernous degeneration) of the optic nerve. That the cupping, however, is due at the outset to increase of tension is shown by the fact that it may disappear after the normal tension is restored by operation or miotics. Nevertheless, there is some reason for thinking that the cavernous degeneration, although not the cause of the cupping, may still constitute an essential feature of some cases of glaucoma, since it and the atrophy that it produces may go on increasing

even after the tension is restored to normal (Elliot).—D.] But at present, the majority of ophthalmologists rank non-congestive glaucoma under glaucoma proper, since it has in common with its most essential symptom, the increase in tension. The interconnection of non-congestive and congestive glaucoma is also proved by the numerous intermediate varieties which form a continuous transition from one to the other, so that no sharp line of distinction can be drawn between the two. A non-congestive glaucoma, later in its course, often passes into the acute or chronic congestive form, and cases not infrequently occur in which congestive glaucoma is present in the eye first affected, non-congestive glaucoma in the one which was affected afterward.

Some cases of non-congestive glaucoma with very slight increase of tension are not always clearly distinguishable from simple optic-nerve atrophy with unusually deep atrophic excavation. In doubtful cases testing of the color perception may furnish a diagnostic guide. In optic-nerve atrophy, color blindness sets in early, while in glaucoma the ability to distinguish colors is retained for a comparatively long time.

447. Hydrophthalmus.—Hydrophthalmus is a disease of childhood. The eye is of unusual size (hence called buphthalmus, ox eye). The thin sclera is bluish, in consequence of the uveal pigment appearing through it; the cornea is larger and either clear and lustrous or, as in inflammatory glaucoma, dull and diffusely clouded. The anterior chamber is unusually deep, the iris is tremulous, and the papilla, after the process has lasted a pretty long time, is deeply excavated. The tension of the eye is considerably increased.

The disease may come to a stop spontaneously, or may continue until it produces blindness. In the former case, the increase of tension subsides after a time; the unnatural bigness of the eyeball persists, indeed, but does not increase, and the eye retains a moderate amount of sight, the degree of which is mainly dependent upon the condition of the optic nerve. In the second case, the enlargement of the eye keeps on—sometimes until quite extraordinary dimensions are attained—and in the mean time complete blindness ensues.

The disease is either congenital or develops in the first year of infancy, and generally in both eyes. Heredity plays an important part in its production. The nature of the disease has not yet been fully cleared up, but increase of tension is certainly the most important factor in it, leading, on the one hand, to the enlargement of the eye, and, on the other hand, to blindness through excavation of the optic nerve. For this reason hydrophthalmus is also called the glaucoma of childhood. Its difference in external appearance from the glaucoma of adults is accounted for in the main by the physiological properties of the eye in childhood. The extensibility of the sclera in childhood renders it possible for the heightened pressure to result in enlargement of the eye as a whole. In the eye of the adult, however, the rigidity of the sclera permits of its expansion through increase of pressure only at its weakest spot—namely, at the lamina cribrosa.

In hydrophthalmus the expansion of the coats of the eye is due to increased pressure acting on their inner surface. Hence we can comprehend that the *lens* does not share in

this enlargement, since it has to bear the pressure on its external surface. The lens, therefore, is the only part of the hydrophthalmic eye that retains its normal dimensions—in fact, it not infrequently falls behind them. Hence the lens is too small compared with the surrounding parts, and the space between lens margin and ciliary processes keeps growing larger. Thus the zonule of Zinn undergoes an elongation which leads to its partial atrophy. Hence in hydrophthalmus of pretty high degree we always find defective fixation of the lens manifested in tremulousness of lens and iris, and not infrequently inducing subsequent changes of position (luxation) of the lens, with their disastrous consequences.

Circumscribed, usually striate or band-like, opacities in the cornea of hydrophthalmic eyes depend upon spontaneous ruptures of Descemet's membrane due to stretching (cf. page 332).

Hydrophthalmus may also be a symptom of *neurofibromatosis multiplex* (Recklinghausen's disease), which is compounded of a number of changes, mostly congenital. These changes, which, it must be admitted, are never all found together in the same individual, are—in the skin, multiple neurofibromata, fibrous mollusca, and flat pigment moles; on the face, neuroma plexiforme and lymphangioma of the lids and orbit and unilateral hypertrophy of the face; lastly tumors of the optic and auditory nerves.

For the anatomical cause of increase of tension in hydrophthalmus, see page 509.

Theories of Glaucoma

448. Increase of Tension the Basis of Glaucoma.—All the essential symptoms of glaucoma can be accounted for as being the results of the increase in pressure. The recognition of this fact by Mackenzie, and particularly by Von Graefe, was the most important step made in establishing the theory of glaucoma.

The elevated intra-ocular pressure has, as its first result, a disturbance of the circulation of blood in the eye, the essential character of which is that of a *venous stasis*. The heightened tension, that is, brings about a compression of the veins in the interior of the eye, and especially in the vasa vorticosa, which, on account of their oblique course through the sclera, are particularly exposed to the influence of the intra-ocular pressure. The blood flowing away from the uvea is hence compelled, in great part, to travel through the anterior ciliary veins; these are consequently dilated, and, in old cases of glaucoma, form a dense venous network encircling the cornea. In non-congestive glaucoma the symptoms of obstructed circulation are confined to the dilatation of the anterior ciliary veins and to the ophthalmoscopically visible distention of the retinal veins. But in congestive glaucoma, in which the increase in tension and with it the disturbance of circulation set in suddenly, these changes induce the phenomena of *inflammatory œdema* in the same way as the incarceration of a hernia, for example, results in inflammatory œdema of the incarcerated coils of intestine. Inflammatory œdema is characterized by hyperæmia of the tissues and marked swelling, due to their serous infiltration, while—in contradistinction to plastic inflammation—exudates and the adhesions produced by them are wanting. Accordingly, the symptoms of acute congestive glaucoma, so far as

they affect the uvea, for example, differ greatly from the clinical picture of an irido-cyclitis; posterior synechiæ are only exceptionally observed in it, and exudations of greater degree, such as hypopyon, pupillary membranes, etc., are never observed. This absence of exudation, in spite of the violent external symptoms of inflammation, is, in fact, just the characteristic feature of inflammatory œdema.

449. Objective Signs of Increased Tension.—The œdema due to increased tension finds different expression in the different portions of the eye:

1. The *corneal cloudiness* of glaucoma is an œdema of the cornea, as has been proved by anatomical investigation. From this can be understood the

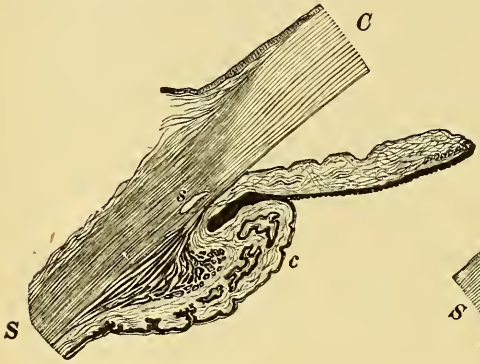


Fig. 215.

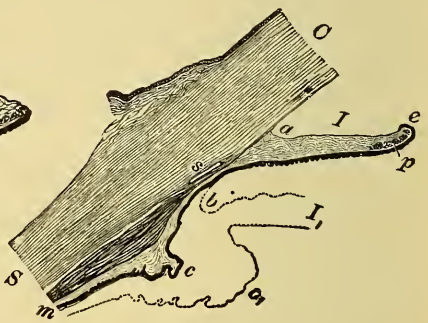


Fig. 216.

Fig. 215.—Iris and Ciliary Body in Recent Inflammatory Glaucoma. Magnified 9 × 1. The ciliary process, *c*, is so greatly swollen that it pushes the root of the iris forward and presses it against the sclera, *S*, and the cornea, *C*. The sinus of the anterior chamber, which should lie somewhat behind Schlemm's canal, *s*, is thus closed. The ciliary muscle shows the pronounced development of the circular muscular fibers (Müller's portion), characteristic of the hypermetropic eye.

Fig. 216.—Iris and Ciliary Body in Old Inflammatory Glaucoma. Magnified 9 × 1. The dotted line gives the outline of the iris, I_1, and the ciliary body, c_1, in the normal condition. The root of the iris is adherent to the sclera, *S*, and the cornea, *C*, wherever it has been pressed against them by the ciliary body. The attachment of the iris is hence displaced forward and lies in front of Schlemm's canal, *s*. So, too, the sinus of the anterior chamber is displaced from *b* to *a*. Wherever the iris has become adherent, it has been thinned through atrophy, so that in places—*b*, for example—it consists of scarcely anything more than the pigment layer. Even the free portion of the iris, *I*, appears in consequence of its atrophy narrower than the normal iris, I_1. Over the pupillary border, *e*, the retinal layer of pigment turns forward farther than usual, and the sphincter pupillæ, *p*, also shares to some extent in this eversion. The ciliary body owing to its having become atrophic, has again separated from the iris, and in fact more so than in the normal condition, so that it is now removed from the iris by a broad interval. The atrophy affects both the ciliary muscle, *m*, and the ciliary process, *c*.

suddenness of development of the corneal cloudiness, and also the promptness of its recession upon reduction of the pressure— e. g., after puncture of the cornea, or iridectomy. If we were dealing with an inflammatory infiltration of the cornea—i. e., with a keratitis—it could not possibly disappear again within a few hours.

2. *Inflammatory œdema of the iris* manifests itself mainly by discoloration and by the obliteration of the details of its structure. The anterior chamber becomes shallower, because the lens is pressed forward together with the iris, in consequence of the elevation of pressure in the vitreous chamber. Added to this there is a displacement forward of the attachment of the iris—that is, the greatly swollen ciliary processes press the root of the

iris forward, so that it is applied to the most anterior portion of the sclera and to the margin of the cornea (Fig. 215). Consequently, the iris appears to take its origin farther forward (Figs. 215 and 216). The ciliary nerves are compressed and paralyzed by the high intra-ocular pressure; hence the insensitiveness of the cornea and the paralysis of the iris (iridoplegia) with which are associated dilatation and loss of reaction of the pupil. The dilatation afterward increases still more in consequence of the atrophy of the iris, that develops as a result of the pressure exerted upon the latter.

3. The redness and clouded appearance of the papilla of the *optic nerve* during the inflammatory attack are caused by hyperæmia combined with some œdema; the excavation which subsequently takes place is the direct result of the elevation of pressure.

[That the excavation is due to the pressure is proved by the fact that it may disappear if the pressure is relieved (see page 497).—D.]

450. Anatomical Changes Produced by Increased Tension.—The anatomical changes corresponding to the objective signs above described are as follows:

In the *cornea* the cause of the cloudiness is found to be œdema of the epithelium. The fluid producing the œdema is found under the form of minute drops, between Bowman's membrane and the epithelium, and also between and in the epithelial cells themselves (Fig. 217). Even though this liquid itself may be quite clear it must cause a cloudiness of the whole epithelial layer, if it has an index of refraction differing from that of the epithelial cells. The dullness of the corneal surface is due to the fact that the epithelial cells are forced apart by the liquid and are pushed up in places so that the surface of the cornea is covered with minute inequalities (Fig. 79). When this

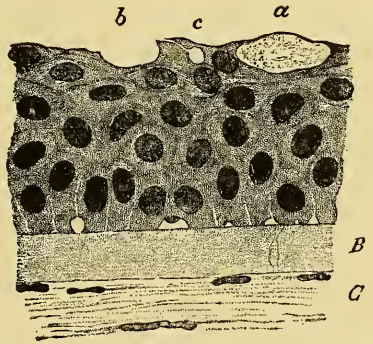


Fig. 217.—Corneal Epithelium in a Case of Increase of Tension. Magnified 500 × 1.

The specimen was from a case in which increase of tension set in after irido-cyclitis. *C*, parenchyma of the cornea with the flat nuclei of the corneal corpuscles. *B*, Bowman's membrane, through which in two places minute nerve filaments are seen passing. At the anterior ends of these nerve filaments and also at other spots there are seen between the bases of the lowermost cylindrical cells (foot cells) light-colored rounded spaces, which represent very minute droplets of liquid. The lines of division between the foot cells are in general represented by lighter colored lines, which indicate that the cells are to a certain extent pressed apart by fluid and their interconnections broken up. On the other hand, the cells of the middle layer are bordered by broad, dark lines representing the interlocking teeth of these, the pectinate cells. In the uppermost layers there are numerous cells altered by imbibition of fluid. In the cell *a*, this fluid occupies the greater part of the cell body, so that the cell itself is enlarged. At *b*, the anterior wall of a cell that was filled with fluid has fallen off. At *c*, the liquefied contents of a cell have been discharged through a slender opening. Owing to these changes in the uppermost cells, the surface of the epithelium as a whole is uneven.

elevation of the epithelium takes place over a pretty large area, small vesicles are formed upon the surface of the cornea.

In the *sclera* an increase in density has been demonstrated, and also a fatty degeneration of the fibers, which look as if sprinkled with minute drops of oil.

The *aqueous* is more albuminous, and coagulates readily in the air and in hardening fluids.

The most important changes affect the *uvea*. In recent inflammatory cases it presents the appearances of inflammatory œdema—i. e., infiltration with an abundant, readily coagulable liquid, while white blood corpuscles that have emigrated from the vessels are present in but scanty numbers; but what strikes one most of all is the marked distention of all the venous vessels, in consequence of which extravasations of blood are produced in many spots. The ciliary processes in particular, which of all the structures of the eye possess the most veins, are greatly swollen through the turgidity and distention of the vessels, and press the root of the iris against the sclera and the cornea. These structures become agglutinated, so that the periphery of the iris remains permanently connected with the sclera and cornea (peripheral anterior synechia), and so continues even when later on the ciliary processes retract again away from the iris (Fig. 216). This retraction takes place in consequence of the atrophy which, after the subsidence of the early inflammatory symptoms, sets in in all parts of the uvea. In the iris, the atrophy finds expression in its becoming narrower and thinner. Rigid connective tissue, from which the blood-vessels have for the most part disappeared, takes the place of the delicate network of anastomosing cells. In the vessels which are still present the walls are thickened, and the lumen is thus contracted or even entirely obliterated (Ulrich). The muscular bundles of the sphincter pupillæ, too, become atrophied. The part which is best preserved is the retinal pigment layer, which, by the great shrinking of the anterior layers of the iris, is drawn continually farther and farther forward over the edge of the pupil (ectropion of the pigment layer; Fig. 216, *e*). Hence, when we look at the eye from in front we find the margin of the pupil encircled by an unusually broad black rim, which sometimes covers half the breadth, or even more, of the surface of the iris. The atrophy attains its greatest height at the root of the iris—i. e., at that part which is adherent to the sclera and cornea (Fig. 216, *b*). In old cases nothing of the iris is left in this spot but the retinal pigment layer and one or two of the larger vascular trunks. These remains of the iris are intimately adherent to the wall of the eyeball; the ligamentum pectinatum is condensed into a tough, fibrous tissue; and, finally, even Schlemm's canal disappears.

The ciliary body diminishes in size through atrophy, so that it draws away again from its contact with the iris, and afterward becomes flatter and flatter, until at length it scarcely forms any projection at all (Fig. 216, *c*). The atrophy affects the ciliary muscle as well as the ciliary processes. In the chorioid the atrophy finds expression in the obliteration of the blood-vessels and the rarefaction of the pigment, so that the chorioid is finally in some spots reduced to a thin, transparent pellicle. This sort of atrophy of the chorioid takes place above all in the neighborhood of the papilla, and by it there is formed the halo glaucomatosus visible with the ophthalmoscope (*h*, Fig. 213, A). The atrophy also reaches a high degree in the spots where the vasa vorticosa pass from the chorioid into the sclera. The chorioid here grows fast to the sclera, which becomes thinned, and, together with the chorioid, bulges out to form an equatorial staphyloma. In the vasa vorticosa themselves is found proliferation of the vascular endothelium, leading to contraction, or even obliteration, of the lumen of the veins (Czermak and Birnbacher).

At the *optic-nerve entrance* the particularly striking feature is the displacement of the lamina cribrosa. This is condensed by the compression of its layers, and is dis-

placed backward so that not infrequently it even gets to lie behind the outer surface of the sclera (Fig. 213 B, *c*). The excavation of the papilla thus produced contains upon its floor atrophic nerve fibers, neuroglia, and some connective tissue (Fig. 213 B, *b*). Large excavations get to have overhanging edges (become ampulliform), because the short canal in the sclera, which is designed for the optic nerve, and which is laid bare by the excavation, is wider behind than in front (see Fig. 23).

In the head of the optic nerve and also behind the lamina cribrosa gaps (lacunæ) of greater or less size are frequently formed (Schnabel). [See page 497.]

In consequence of the destruction of the head of the optic nerve, the retina and the trunk of the optic nerve also atrophy; the latter becomes thinner, as a whole, and shows that its connective-tissue trabeculæ have been enlarged at the expense of its nerve fibers (Fig. 213 B, *n*).

Precise as is our information in regard to the anatomical changes above described, and many others, too, in glaucoma, we must be correspondingly cautious in the interpretation of their significance, if we are bent upon finding out the anatomical cause of glaucoma. Most of these changes, if not all, are simply the result of the increase of pressure—as is, without any doubt, the case with regard to the atrophy of the tissue and the excavation of the optic nerve. To find those changes which precede the increase of tension and cause it, we should have to examine the eye in the earliest stages of glaucoma. The opportunity for making such an examination has hitherto been but very rarely offered us. Indeed, most of the glaucomatous eyes that have been examined are those which have been enucleated in the stage of glaucoma absolutum because they were painful.

451. Subjective Symptoms Due to Increased Tension.—The violent *pain* in inflammatory glaucoma is excited by the compression of the extremely numerous sensory nerves of the ciliary body and the iris.

The *disturbance of vision*, too, is the result of the heightened intra-ocular pressure, which causes it in different ways, according as we are dealing with congestive or non-congestive glaucoma. In *congestive glaucoma* the disturbance of vision is brought about by—

(*a*) The glaucomatous opacity of the cornea, which affects chiefly central vision, and that, too, to a marked degree, on account of the uniformity of its diffusion.

(*b*) The ischæmia of the retina, due to the compression of the retinal arteries, by which is caused the contraction of the field of vision. This compression makes itself felt first in those sections of the arterial channel in which the blood pressure is lowest. But, since the blood pressure in the vessels is less the farther we get away from the heart, the terminal expansions of the arteries lying in the periphery of the retina are first affected by the compression. Hence the periphery of the retina first becomes insensitive to light, a thing which manifests itself by contraction of the field of vision. Moreover, the ischæmia does not become apparent in all parts of the retina at once or in equally high degree. Since the papilla of the optic nerve, and with it the point of entrance of the retinal vessels, lie to the nasal side of the posterior pole of the eye, these vessels have a greater distance to travel in going to the temporal margin of the retina than to the nasal margin. The

former is therefore first affected by the ischæmia, so that the limitation of the field of vision begins generally at its nasal margin.

Both the opacity of the cornea and the acute ischæmia of the retina belong solely to congestive glaucoma, and it is they which are the principal cause of the very great reduction of sight during the acute attack of glaucoma. They are of transitory nature, disappearing again as the elevated tension abates, and at the same time with their disappearance an improvement of the acuity of central vision and an enlargement of the field of vision set in.

(*c*) In the third place, the acuity of vision is diminished by direct pressure upon the optic-nerve fibers and their consequent atrophy—atrophy which takes place during the formation of the glaucomatous excavation. The disorder of vision thus produced is permanent, since the atrophy of the optic-nerve fibers is irreparable.

In *non-congestive glaucoma* the two first-named factors are wanting. The disorder of vision is here caused solely by the excavation and simultaneous atrophy of the papilla of the optic nerve.

452. Explanation of the Increase of Tension.—Easy as it is to deduce the symptoms of glaucoma from the elevation of tension, it is quite as difficult to account for the origin of the latter, and thus explain the essential nature of glaucoma. Of the many theories which have hitherto been propounded upon this subject, no single one is satisfactory in every respect. Only the most important of them will be adduced here, and that mainly with the object of showing upon what circumstances increase of tension in general depends.

The intra-ocular pressure is determined by the relation between the internal capacity and the elasticity of the envelopes of the eyeball, on the one hand, and the amount of its contents, on the other. If the latter factor increases or the former factors diminish, the pressure is elevated. An alteration of the internal capacity of the envelopes of the eyeball cannot be brought in to account for the increase of pressure, since the volume of the envelopes of the eyeball, as a whole, is unalterable. In advanced life, to be sure, the sclera is not only more rigid, but also a little contracted; but the diminution in volume so caused is extremely slight. We must, therefore, look for the cause of the elevation of tension in an increase in the amount of the contents of the eyeball, the envelopes of the eyeball being at the same time not sufficiently elastic to adapt themselves to their increased contents without marked heightening of the pressure. Now the amount of matter contained in the eyeball depends, on the one hand, upon the amount of ocular fluids which are constantly being secreted by the blood-vessels, and, on the other hand, upon the quantity of fluid which leaves the eye again through the lymph passages; it corresponds, that is, to the relation between inflow and outflow, between secretion and excretion.

In the normal eye this relation always remains about the same, since with any increase of the inflow the liquid, which is now subjected to an increased pressure, also leaves the eye more quickly, so that the normal pressure is soon restored. For any permanent increase of pressure to occur, a disturbance of this self-regulating action must be present. Such a disturbance can be looked for only in the excretory apparatus, for so long as this works normally, every increase in the amount of fluid would soon be compensated for by increase in the outflow. But if the outflow is interfered with, a normal or even a diminished inflow of liquid must lead ultimately to over-distention of the eyeball. Consequently the explanation that is accepted by most authorities at the present time to account for the increase of tension, namely the theory of Knies and Weber, presupposes a disturbance of the outflow. The most important path for the outflow lies in the sinus of the anterior chamber and passes through the ligamentum pectinatum into Schlemm's canal. It is at this spot that the obstacle must be looked for. Such an obstacle is produced in the following way:

A genuine glaucoma develops only in an eye which has a *predisposition* to it. This predisposition depends on insufficient spatial relations. These affect the eye as a whole and particularly affect the region of the anterior and posterior chambers. They are afforded by: 1. Smallness of the eye as a whole; 2. Shallowness of the anterior chamber;[2] 3. Undue protrusion of the ciliary processes; 4. Disproportionate size of the lens (Fig. 218). Such relations appertain to the hypermetropic eye when it has become old. The hypermetropic eye as a whole is smaller than the normal eye, its anterior chamber is shallower, and its ciliary processes protrude more than usual toward the lens because the ciliary muscle is hypertrophied on account of the constantly maintained accommodation. Yet the lens in such eyes is no smaller than it is in the emmetropic eye. And, as regards the lens, there is the additional fact that it increases in size progressively with advancing age, and yet the eye itself grows no larger, so that ultimately the lens becomes too big for the eye. Hence, the hypermetrope does not get glaucoma as long as he is young, but gets it only when with advancing age his lens has become too large, for then the space between the lens and ciliary processes becomes too narrow. [These predisposing factors may occur in many who are not hyperopes (see pages 496 and 497).—D.] It is through this space—the circumlental space—that the lymph flows from the vitreous to the aqueous cavity. When this space is contracted the lymph becomes dammed up in the vitreous. The latter consequently increases in volume, pushes the lens

[2] [As Priestley Smith says, the shallowness of the anterior chamber in persons affected with glaucoma has a double significance. In part it is a pre-existing condition, constituting one of the causes of glaucoma; in part it is a condition produced by the glaucomatous process itself and particularly by the glaucomatous attack (see page 500). The pre-existing shallowness is due to the continuous growth of the lens making it too big and too broad and perhaps also to peculiarities in shape of the suspensory ligament. The additional shallowness produced by the attack is probably due to turgidity of the ciliary processes resulting from sudden congestion. The swollen processes either imprison the vitreous behind the lens and so push the latter forward, or as they swell advance and draw the lens with them (Elliot).—D.]

and iris forward, and thus makes the anterior chamber still shallower. An actual increase of tension, however, is not yet present in such an eye because the sinus of the chamber is still present, so that an increased amount of aqueous, such as would correspond to the increased volume of the vitreous, can be still discharged exteriorly. An actual attack of glaucoma does not occur in the predisposed eye until this passage of outflow is blocked. This

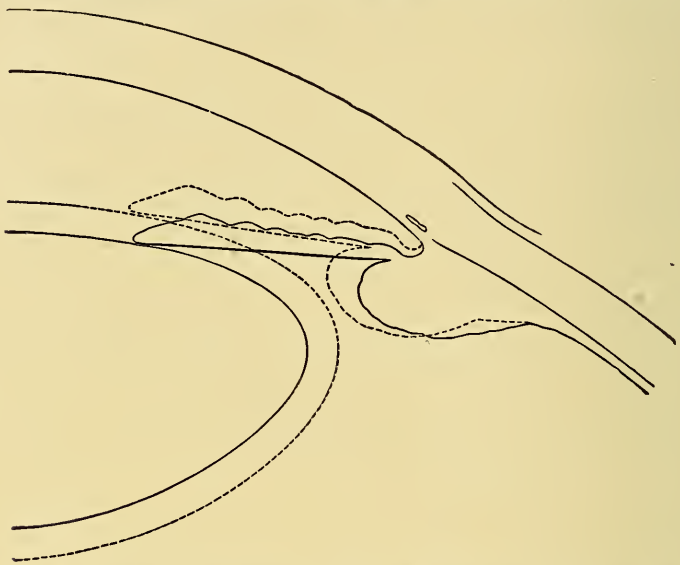


FIG. 218.—SCHEMATIC REPRESENTATION OF THE PREDISPOSITION TO GLAUCOMA.

The unbroken line corresponds to the spatial relations in the young emmetropic eye, the dotted line to the relations in the senile hypermetropic eye. In the latter the ciliary body juts further forward. The lens is enlarged. Owing to the increase of its equatorial circumference, its margin extends further toward the periphery, and, owing to the enlargement of the sagittal diameter, the iris is pushed forward so that the anterior chamber is shallower. The figure has some resemblance to Fig. 371, which represents the process of accommodation. The important difference between the two consists in the fact that in the latter figure the margin of the lens recedes by the same amount that the ciliary body advances, so that the circumlental space remains of constant width. In Fig. 218, on the contrary, the ciliary body and the margin of the lens advance toward each other and thus contract the circumlental space.

takes place from closure of the sinus of the chamber, as is produced by some one of those causes which, we know, can excite an attack of glaucoma. The *exciting causes* of this sort, that we are acquainted with, are disturbances in the circulation of the blood and dilatation of the pupil.

Let us, to begin with, consider the first case, a *stasis of blood* in the veins of the greater circulation. In the eye such a stasis makes itself apparent principally in the ciliary processes which are extremely rich in veins. The ciliary processes consequently swell up and, as the circumlental space is so narrow, soon extend to the margin of the lens. In this way the communication between the vitreous cavity and the anterior chamber is still more interfered with, and the vitreous, owing to the retention of lymph, swells up to a still greater extent than before, and pushes forward still more strongly upon the swollen ciliary processes. Since the latter are already abutting

against the margin of the lens, they can give way toward the front only. They swell forward into the posterior chamber until they reach the root of the iris, and jam the latter forward against the corneo-sclera (Fig. 215). But in doing so the iris shuts up the passage of outflow for the aqueous, and, hence, the intra-ocular pressure at once necessarily rises. In this way a glaucomatous attack is brought about.

A glaucomatous attack in a predisposed eye may also be set up by *dilatation of the pupil* (as a result of psychical excitation, or by mydriatics, etc.). When the iris grows narrow in the process of dilatation of the pupil, it increases correspondingly in thickness. If this takes place in an eye which has markedly projecting ciliary processes, on the one hand, and a shallow anterior chamber, on the other, the thick iris will block the narrow sinus of the chamber (Fig. 219) and thus excite increase of tension.

If the swelling of the ciliary processes soon recedes, or the pupil soon contracts again, the iris returns to its former position, the sinus of the chamber becomes free, the tension falls, and the attack, being in this case simply a prodromal one, passes off. If, on the other hand, a return to the normal condition does not take place soon, the root of the iris becomes agglutinated to the corneo-sclera, and out of this agglutination an adhesion afterward develops (Fig. 216). Then a return to the normal conditions has become impossible, and the glaucoma is permanent.

453. Summary of Glaucoma Theories.—The older theories of glaucoma endeavored to account for the elevation of tension by attributing it to an increase in the volume of the eyeball due to an increase of the inflow (see also page 509):

Von Graefe assumed the existence of an increased excretion of fluid by the vessels of the chorioid as a result of inflammation of the latter. Since the ophthalmoscopic symptoms of chorioiditis are generally wanting in glaucoma, Von Graefe, to get over this difficulty, assumed the existence of what he called a serous chorioiditis, the nature of which was supposed to consist in a serous transudation without any coarser anatomical changes.

Donders ascribed the increased secretion on the part of the chorioid to the influence of the ciliary nerves. He looked upon glaucoma simplex as the typical form of glaucoma because it was not complicated with inflammation. Hence he could not consistently look for the cause of the increased excretion of fluid in an inflammation of the chorioid. Rather, he supposed it to originate in an irritation of the chorioidal nerves, being thus a sort of neurosis of secretion, just as, for example, increased secretion takes place in glands upon irritation of certain nerves.

Stellwag referred the elevation of tension not to increased excretion of fluid, but directly to the increase of blood-pressure in the vessels of the interior of the eye. The pressure which the blood exerts on the vessel wall is borne only in part by the latter; so far as the vessels expand under the influence of the blood-pressure they transmit a portion of this pressure to their surroundings. The blood-pressure in the intra-ocular vessels accordingly forms an important item in the sum total of intra-ocular pressure, so that its elevation has as its direct result an increase in the ocular tension as a whole. The interior vessels that are to be considered in this connection belong principally to the uvea, that being the most vascular portion of the eye. The elevation of the blood-pressure in the vessels of this part is, therefore, according to Stellwag, the direct cause of glaucoma. It makes its appearance in consequence of obstructions to the circulation

which affect mainly the region of the vasa vorticosa, and are brought about by diminished elasticity and shrinkage of the sclera; for those sections of the vasa vorticosa which pass with a very oblique course through the sclera are liable to compression whenever the latter shrinks. According to Heerfordt, a compression of this sort takes place at the spot where the venæ vorticosæ enter the sclera from the chorioid. Here, at the back part of the channel of entry, the sclera forms a tapering prominence, which juts out into the veins and, it is said, closes them like a valve.

Against these theories the objection must be made, primarily, that an increase in the inflow or an over-distention of the vessels cannot by themselves account for the elevation of tension, since, if the conditions are otherwise normal, an increase in the contents of the eyeball is immediately compensated for by the increased outflow. If a few drops of liquid are injected into a healthy living eye, and the intra-ocular pressure is thus heightened, it returns to the normal again after a short time, since a correspondingly greater amount of liquid flows off through the lymph channels. If we ligate the venæ vorticosæ at their points of exit from the eye, we get an increase of tension in the sense of Stellwag's theory, but it is not permanent.

[Other theories that have been propounded to account for the development of glaucoma are: (*a*) œdematous and vascular conditions at the posterior pole of the eye (Fergus and others); (*b*) cavernous atrophy of the optic nerve (Schnabel—see page 497); (*c*) fibrosis of the pectinate ligament (Henderson); (*d*) increase in the amount of hydrophilous colloids in the eye, due to acidosis and causing œdema of the eyeball (Fischer and others). None of these seems tenable as explanations for glaucoma in general (Elliot).—D.]

The foundation for the views that now prevail was laid by Knies, who was the first to show that the peripheral adhesion of the iris, which had been already known to exist, occurred quite regularly in glaucomatous eyes, so that he brought it into causal connection with the glaucoma. He explained the adhesion itself as being due to an adhesive inflammation in the vicinity of the sinus of the chamber. But almost simultaneously Weber by examining a recent case of glaucoma proved that the cause of the obliteration of the sinus was not inflammation but the pushing forward of the iris by the swollen ciliary processes. Priestley Smith then demonstrated that glaucomatous eyes are on the average smaller than normal eyes, and have comparatively large lenses; he also was the first to recognize that the lens continues to grow even late in life. He also showed how the thickening of the iris, that occurs simultaneously with the dilatation of the pupil, acts. It is not the root of the iris that in this case is pushed against the cornea, for the root of the iris is very thin. But directly to the inner side of the root the iris attains its full thickness, so that here its anterior surface turns up and passes abruptly forward; and it is this point (*a* in Fig. 219) that first comes into contact with the posterior surface of the cornea when the iris is thickened. In this way the sinus is closed off so as to form a ring-shaped space which no longer communicates with the anterior chamber. Then in both anterior and posterior chambers, the pressure rises and

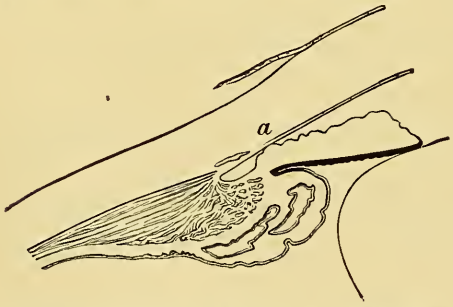


Fig. 219.—Iris and Ciliary Body in a Very Hypermetropic Eye in Dilatation of the Pupil. Magnified 9 × 1.

The iris is contracted and thickened, so that at *a* it comes into contact with the posterior surface of the cornea and cuts off the sinus from the rest of the anterior chamber. The ciliary body is unusually large; the circumlental space narrow.

forces the most peripheral, or root portion of the iris against the sclera. The return to normal conditions, such as occurs in the case of the prodromal attacks, Czermak accounts for upon the supposition that owing to the increase of tension a state of irritation sets in, which by reflex action causes contraction of the pupil, so that the iris is again drawn away from the cornea. But, in order for this to take place, it is necessary that the sphincter pupillæ should be strong enough, and, moreover, no adhesion must have formed between the iris and the cornea. [In the contrary case, a return to the normal is impossible, and permanent glaucoma ensues.—D.]

Even the present theories are not free from objections. So far, no explanation of glaucoma has yet been propounded which is satisfactory in every respect. The reason for this is perhaps to be found in the statement that all cases of primary glaucoma probably do not develop in the same way, so that *one* explanation could not possibly fit all cases. And, in particular, it might be possible that glaucoma simplex and inflammatory glaucoma would have to be referred to different causes. In non-congestive glaucoma the sinus of the anterior chamber is often found to be open, so that closure of the latter cannot be the cause of the increase of tension. In hydrophthalmus the anterior chamber is particularly deep and apposition of the root of the iris to the cornea is not to be thought of. But probably the sinus is partially filled by a fibrous tissue, representing a residue of the fetal ligamentum pectinatum, which in the normal eye disappears in part even before birth, but in hydrophthalmus remains permanently and forms a rather dense tissue. Commonly, too, Schlemm's canal is wanting, and in these congenital anomalies we have to look for the cause of the hindrance to filtration outwards.

[Elliot gives the following classification of the factors, any one or several of which may be effective in causing the development of glaucoma, especially in old age:

A. Anatomical Conditions:
   (*a*) Enlargement of the lens.
   (*b*) Slackening of the zonula, so as to allow advancement of the lens.
   (*c*) Thickening of the fibers of the pectinate ligament (Henderson).

B. Degenerative Changes causing:
   1. Increase in the volume of liquid secreted. From—
      (*a*) Nervous irritation of the secretory fibers due to inflammation of the chorioid (Von Graefe), inflammation of the ciliary body (Bjerrum), or irritation of the secretory nerves (Donders). This view, which had been given up (pages 507, 508), has recently been revived and has some arguments to support it.
      (*b*) Degeneration of glandular elements, especially in the ciliary body. The increased secretion here would be analogous to that which takes place in the kidney in chronic nephritis.
      (*c*) Alterations in vaso-motor pressure (see E, below).
   2. Alteration in character of secretions poured into the eye. This is accepted as a probable cause in glaucoma secondary to irido-cyclitis, and it is likely that it is also a cause in primary glaucoma. (According to Priestley Smith, the aqueous is secreted by the ciliary processes, the vitreous by the pars plana of the ciliary body, and it is likely, he says, that senile degeneration may affect these two zones unequally, so as to cause over-production and retention of fluid in the vitreous with normal production of the aqueous. The lens would then be shoved forward and a condition of chronic glaucoma would be set up. There are pathological findings that agree with this assumption).

C. Hydrophilism of the eye tissues.

This is the theory of Fischer and others (see page 508). It must be regarded as improbable.

D. Autogenous intoxications.

These set up low forms of inflammation which may act either by obstructing the passages of outflow or by hampering the ciliary muscle[3] (Risley, La Grange). Cases thus caused are closely akin to cases of secondary glaucoma.

E. Changes in vascular system.

(*a*) Increase in general vascular tension. The well-founded general belief is that this has no relation to the development of glaucoma.[4]

(*b*) Increase in vascular pressure limited to the eye. A possible cause, but at present not susceptible of proof.

(*c*) Loss of vaso-motor control in the body or in the eye. One of the principal factors in producing congestive glaucoma and probably important for simple glaucoma too. The glaucoma set up by irritation of the fifth nerve and the sympathetic are to be ascribed to this factor, the effect of which is also seen in the various exciting causes of glaucoma in general and especially of the acute attack, viz., hunger, cold, sleeplessness, nervous and bodily exhaustion, anxiety, emotional stress, constipation, cardiac obstruction, etc. The effects of such vascular disturbance are particularly marked in age, when resistance to shocks of every kind is lessened.—D.]

Treatment of Primary Glaucoma

(A) OPERATIVE TREATMENT

454. Glaucoma passed for an incurable disease until Von Graefe discovered the curative action of iridectomy. Afterward still other methods of operating in glaucoma were devised, none of which, however, has been able to displace iridectomy.

1. **Iridectomy.**—Iridectomy (see §§ 853, 857) must satisfy certain conditions, if it is to be efficient in combating glaucoma. The wound should lie in the sclera, not in the cornea; and the excision of the iris should be carried to the ciliary margin, and be made as broad as possible. [If the incision is made well back of the limbus and the root of the iris is torn away up to its insertion, it does not seem necessary to make a broad iridectomy. —D.] Incarceration of the iris in the wound after the operation should be avoided by careful reposition. If possible, the iridectomy is made upward, so that the coloboma may be covered by the upper lid, and not cause trouble through the dazzling due to irregular refraction.

Iridectomy is often *difficult to perform* in congestive glaucoma, on account of the cloudiness of the cornea, the shallowness of the anterior chamber, the rottenness of the iris, and the great painfulness of the parts. Hence the excision of the iris does not always turn out to have been done correctly. Luckily it is precisely in congestive glaucoma that even a less successfully performed iridectomy is usually followed by the effect

[3] [If Thomson's views of the action of the ciliary muscle in pumping liquid out of the eye are correct (see page 15), the development of glaucoma can be effected by anatomical and degenerative as well as toxic changes affecting this muscle.—D.]

[4] [It seems likely that when, as often happens, high vascular tension and glaucoma are associated, one condition is not dependent on the other, but both arise independently from the same exciting causes (viz., those given under *c* below), constantly repeated.—D.]

desired. Above all, we must avoid injuring the capsule of the *lens*, an event which might easily happen from the narrowness of the iris and the shallowness of the anterior chamber. Such an eye is greatly jeopardized, since the injured lens swells up, and thus gives rise anew to increase of tension.

Traumatic cataract, however, may follow an iridectomy for glaucoma without any fault of the operator. When the intra-ocular pressure is high or when there is strong pressure exerted by the patient, the edge of the lens is pushed against the incision, and in consequence the capsule of the lens may be ruptured at this portion of the lens margin. The result is a subsequent opacification of the lens. Because of the wound in the capsule the lens may make a spontaneous exit through the incision, either immediately after the operation is completed or some days later.

Iridectomy in congestive glaucoma is usually *followed by hæmorrhage* into the anterior chamber and into the retina. The sudden diminution of the pressure, the fact that we are operating in a very hyperæmic eye, and the degeneration of the vessel walls are all accountable for this. The blood in the anterior chamber is sometimes unusually slow in being absorbed, because the normal channels of outflow are stopped up. The retinal hæmorrhages cause no special harm, except when one of them happens to involve the region of the yellow spot.

On account of the great tension of the glaucomatous eye, the *edges of the wound* after iridectomy do not close up as well as in the case of other iridectomies—e. g., those made for optical purposes. Hence, more frequently than is otherwise the case, we get, instead of direct union of the edges of the wound, healing with the interposition of an interstitial tissue, as a consequence of which ectasis of the cicatrix or cystoid cicatrization is readily produced.

[Iridectomy and also sclerectomy both by Elliot's trephining and still more by La Grange's method are often followed by *detachment of the chorioid* (see page 472).—D.]

In *glaucoma simplex* it sometimes happens that iridectomy [and occasionally also sclerectomy] have for their immediate result a marked reduction in the sight. This is to be apprehended when the field of vision was so very much contracted before the operation that its limits at one spot reached nearly up to the point of fixation. Then a slight intussusception of the confines of the visual field carries them beyond the point of fixation, so that central vision is lost. Hence the rule is to do iridectomy as early as possible, while the field of vision is still large.

In *hydrophthalmus*, iridectomy is associated with greater danger than in the glaucoma of adults, and that mainly because of the defective condition of the zonula. By reason of this, when, after the escape of the extremely abundant aqueous, the lens is driven forward, rupture of the zonula, and hence escape of vitreous through the wound may take place. Another source of danger consists in the fact that we are dealing with children, of whom quiet behavior after the operation is not to be expected. Less dangerous and yet successful has proved in my hands an iridectomy in which I make the incision very short, but combine it with the exsection of a scleral flap by La Grange's method (see § 861), so that the operation has the effect of a sclerectomy. Another operation that in hydrophthalmus is less dangerous than iridectomy is trephining by Elliot's method (§ 862).

455. Other Operations.—Why does iridectomy diminish the intra-ocular pressure? In an eye the tension of which is normal, the latter is not diminished by an iridectomy; for instance, if an iridectomy is made on account of an opacity of the cornea, the eye does not therefore become permanently softer. It is only a pathologically heightened tension that iridectomy reduces. Of the many reasons that have been given for this action in reducing tension, one only will be here adduced, because it has given rise to

a new operative procedure. This is based on the idea that by the incision in the sclera made by the iridectomy a scar is produced which allows fluid to filter through it, as the normal sclera does not do. This filtration cicatrix, it is thought, affords a substitute for the obliterated ligamentum pectinatum. From the view that iridectomy owes its efficacy to the section in the sclera, sclerotomy and sclerectomy, and other operations have taken their origin.

2. *Sclerotomy* consists in making in the sclera an incision, which is placed as far as possible in the periphery of the anterior chamber, and in which no iris is excised (for the technique, see § 860). There is no doubt that sclerotomy, too, which for a time was very much practised, has permanently cured many cases. For the most part, however, the cure has not been final, so that an iridectomy has had to be resorted to subsequently. At the present time, therefore, most operators perform sclerotomy only in those cases in which for technical reasons, iridectomy cannot be performed or in which in spite of an iridectomy the increase of tension has returned.

3. Of more lasting effect is *sclerectomy*, which is done either in the way proposed by La Grange or by trephining (§§861, 862). The removal of a bit of the entire thickness of the sclera produces a fistulous cicatrix with more certainty than does a simple incision, whose margins in the course of time may become quite solidly re-adherent. The effect of sclerectomy in forming a fistula is shown in the œdema of the conjunctiva over the cicatrix (cystoid cicatrization—see page 396). It must be admitted, however, that such a cicatrix is much more liable to the danger of late infection than is a solid cicatrix.

[Sclerectomy, more particularly in the form of Elliot's trephining operation, has largely superseded iridectomy in cases of simple and also often in cases of chronic congestive glaucoma. Recently there has been a tendency to return to iridectomy even in these conditions—this partly because of the danger of late infection, referred to above, which, according to some, occurs in some 7 per cent of the cases. In acute congestive glaucoma the pre-eminent value of iridectomy has not so far been successfully contested.—D.]

4. Many *other operations* have been devised for glaucoma, especially De Vincentiis's operation (§ 863), cyclodialysis (§ 863), [and iridotasis (§ 859)].

5. *Enucleation* is indicated when an eye which has been rendered perfectly blind by glaucoma is continually painful, and an iridectomy is either impossible of performance on techincal grounds or has been already performed without success. In this case enucleation is done simply with the object of relieving the pain, and may, in suitable cases, be replaced by opti-co-ciliary neurotomy (see § 895).

456. Time for Performing Operation.—It is best to operate as early as possible. In congestive glaucoma the operation should be done in the prodromal stage, in case the patient can make up his mind to it. If he wait until the inflammatory attack, we cannot tell how severe it is going to be,

and, in any case, we operate then under less favorable conditions. Under all circumstances the operation is demanded in the prodromal stage when the other eye is already rendered blind by glaucoma; in this case, too, the patient will more readily comprehend the necessity of the operation. When an eye has already been rendered blind by glaucoma, the restoration of sight is no longer possible by an operation; but yet one is often performed to relieve the painfulness of the eye, or to prevent the development of glaucomatous degeneration.

In glaucoma simplex it is not a question of interfering within a few days or weeks, as is often the case in congestive glaucoma, but even here the operation should not be long deferred; the earlier we operate, the better results we obtain.

457. Prognosis of Operation.—The success of an operation in respect to vision can be estimated approximately beforehand, if account is taken of what morbid changes can and what cannot be removed by the operation. Iridectomy and the other operations reduce the intra-ocular pressure to the normal amount. They remove the glaucomatous cloudiness of the cornea and the disturbance of vision produced by it, as well as the disturbance of vision caused by the compression of the retinal vessels. But the excavation and the atrophy of the optic-nerve fibers associated with it either do not abate at all or do so in but very slight degree, so that the disturbance of sight, as far as it is dependent upon them, persists. From these facts is deduced the effect of operation in the separate forms of glaucoma:

(*a*) In *congestive glaucoma* the result of operation in recent acute cases is extremely favorable. The pain accompanying the glaucomatous attack ceases a few hours after the operation, the cornea in the next few hours or days becomes clear and sensitive once more, and the other inflammatory symptoms likewise speedily disappear. The sight, which during the attack was very much reduced through the cloudiness of the cornea and the compression of the retinal vessels, increases very considerably after the disappearance of these factors. If it was still normal before the attack, it becomes almost normal again after the operation. But if a long prodromal stage with the formation of an excavation has preceded the attack, both the acuity of direct vision and the visual field have already ceased to be normal some time before the attack, and will then be more imperfect still after the operation. We may accordingly put it down as a rule in acute congestive glaucoma that, provided we make an iridectomy *soon after the outbreak of the inflammatory attack*, a degree of sight is secured, *which is somewhat, but not much, smaller than it was before the attack*. The later the operation is done after the inflammatory attack, the less successful it is. In a few cases, to be sure, the result of the operation is to this extent not so favorable, that, in spite of the operation's being correctly performed, the increase of tension either keeps up or sets in anew. In these cases it is generally possible to

attain our end by a second operation (iridectomy or sclerectomy). Finally, there are cases in which, in spite of all attempts at operative interference, complete blindness supervenes. These unfavorable cases, however, are rare in acute glaucoma; so that, as a rule, one may count upon a good result from the operation, and one, too, which is lasting.

In chronic congestive glaucoma in determining the prognosis of operation we must estimate how much of the disturbance of vision present is to be charged to the cloudiness of the media, and how much is due to the excavation and atrophy of the papilla of the optic nerve. The former portion is removed by operation, the latter not.

(*b*) In *glaucoma simplex* the result of operation is less pronounced and also less permanent than in congestive glaucoma. The visual disturbance in the variety of glaucoma simplex, in which the media are clear, is caused solely by the changes in the papilla of the optic nerve. Since operation cannot remove these changes, it is impossible to restore by means of it the normal acuity of vision. Operation can only do away with the increase in tension, and thus put a stop to the advance of the process. If we are dealing with cases in which the increase of tension is minimal only, iridectomy is almost absolutely useless; and in the other cases, on the contrary, it gives greater promise of a permanent good result the more pronounced the increase in tension is. The rule is that the sight is *maintained in statu quo* by operation, or, at most, is slightly improved. In many cases a repetition of the operation is necessary in order to obtain even this result, or the failure of sight goes on unchecked in spite of the operation. This may even take place when, in consequence of the iridectomy, the intra-ocular pressure has become permanently normal. We then assume that an atrophy of the optic-nerve fibers, when once initiated, keeps on progressing in spite of the reduction of the intra-ocular pressure [cf. remarks on cavernous atrophy, page 497]. Particularly unfavorable are those cases in which iridectomy actually exerts a bad effect upon the sight, the latter falling away very rapidly after the operation, so that blindness sets in earlier than it would have done without the iridectomy [see page 511]. Sometimes inflammatory symptoms and pain make their appearance just after the operation, when they were not present before. The eye feels hard directly after the operation, the anterior chamber fails to re-form, and the eye becomes blind rapidly and with symptoms of violent pain. These cases, which, to be sure, are rare, are known as *glaucoma malignum*.

The *prognosis* of iridectomy in glaucoma, then, is as follows: In congestive glaucoma the operation acts favorably upon the inflammation and upon the eyesight, and its good results are permanent; it is hence unconditionally indicated. In glaucoma simplex only the maintenance of the status quo is to be counted upon. In a certain number of cases the operation is unsuccessful or actually does harm. Nevertheless, as without an operation

the eye will certainly grow blind, iridectomy is indicated in glaucoma simplex too—as soon as an increase of tension can be distinctly made out. We endeavor to perform the iridectomy as early as possible, for the more advanced the disease is, the more uncertain is the result of the operation.

Favorable as is the action of iridectomy in congestive glaucoma, we ought not to conceal the fact that in many cases which have remained for years apparently cured *blindness nevertheless ultimately develops*, either because a very slight increase of tension insidiously sets in or without increase of tension and simply as a result of a gradually advancing atrophy of the optic nerve, just as is the case in glaucoma simplex (see page 514). But as this outcome does not usually set in until after years have elapsed, and as furthermore glaucoma is a disease of advanced life, most of the patients do not survive to experience this melancholy sequel, so that in general the prognosis of iridectomy in congestive glaucoma may be regarded as favorable.

Views differ in regard to the efficacy of *iridectomy in glaucoma simplex*. Von Graefe estimated the number of definite cures produced by the operation at rather more than half the cases; in one quarter of the cases relapses occurred, which were cured only by a second iridectomy, while in the rest of the cases blindness gradually set in in spite of the operation. Only in 2 per cent of all the cases did the operation have actually a bad effect. Since then reports in regard to the curative effects of iridectomy in glaucoma simplex have been published by different authors, as by Hirschberg, Sulzer, Nettleship, Charles Stedman Bull, Gruening, etc. Most of these statistics prove, in harmony with the statements of Von Graefe, that in about half the cases the operation has put a stop to the progress of the disease. [The reports of various operators, collected by I. C. Wecker, showed results ranging from nearly universal failure to over 70 per cent of successes. These differences are perhaps to be explained by supposing that the unsuccessful operators either habitually performed iridectomy too late or failed to make a sufficiently peripheral incision or failed to detach the root of the iris.—D.] Dr. Laska has collected my own observations upon this point, and from them the following results have been obtained: Out of thirty-nine cases, iridectomy had a favorable result in nineteen—that is, in about one half—the sight either being kept stationary or actually improving; but in twenty cases the eyesight failed in spite of the operation, either from the subsequent reappearance of the elevation of tension, or even without this taking place. The value of these statistics, small as they are, lies in the fact that only cases that had been under observation a pretty long time were accepted in making them up. The mean period of observation amounted to five years in the nineteen cases that were cured; several of these had been followed up for more than ten years.

[The above remarks on the prognosis of iridectomy and the time for operating apply in general to the other operations. Because of the uncertainty of action of iridectomy in simple glaucoma, many have resorted to sclerectomy (especially trephining) and cyclodialysis, believing that thus more permanent results would be secured. Whether this is so or not is still undecided and, indeed, cannot be determined until we have had longer time, in which to test the newer methods. Quite recently, in this country at least, the trend has been rather away from the latter and toward iridectomy again.—D.]

(b) MEDICINAL TREATMENT

458. The miotics, eserine and pilocarpine, are powerful agents in combating increase in tension. They act only when the iris is capable of contracting satisfactorily; hence, in old cases of glaucoma with a completely atrophic iris they are useless. Their action is accounted for upon the sup-

position that by the contraction of the pupil the iris is stretched in a radial direction, and so is drawn away from the wall of the eyeball to which it has been applied, so that the sinus of the chamber again becomes free. Unfortunately, the effect of miotics upon the ocular tension is not lasting, inasmuch as it vanishes with the disappearance of the miosis. The miotics cannot, therefore, cure glaucoma permanently, and thus enable us to dispense with iridectomy; they are, however, valuable auxiliaries in the treatment of glaucoma.

In the prodromal stage of glaucoma miotics are employed to cut short the prodromal attacks. If the patient instils a miotic at the commencement of the attack, the latter comes to an end in about half an hour. Thus we can for a long time prevent the prodromal attack from rising into an acute inflammatory attack. Nevertheless, we should not protract the prodromal stage in this way until excavation of the optic nerve with permanent impairment of sight sets in. As soon as this threatens, we must proceed to iridectomy. Treatment with miotics cannot escape the imputation that because of its momentary good effect it often causes the patient to put off operation too long.

During the acute inflammatory attack, also, miotics reduce somewhat the elevated tension, and thus ameliorate the pain and contribute to the disappearance of the glaucomatous cloudiness of the cornea. In this way it becomes more feasible to put off the operation for a few days, if circumstances require it. Moreover, the operation is rendered easier of performance, since the iris, very narrow before, becomes broader through the contraction of the pupil. [In acute glaucoma a 1- or 2-per-cent solution of pilocarpine or better a ½-per-cent solution of eserine should be used, and the instillations repeated until the desired contraction of the pupil is secured. Preliminary to operation, purging and the administration of bromides and morphine are also useful (Koller).—D.]

In glaucoma simplex the action of miotics is dubious, and the more so the less pronounced the increase of tension is. [There is accumulated evidence to show that by the continuous use of miotics the progress of chronic glaucoma and glaucoma simplex can be arrested—apparently for a number of years. Pilocarpine should be used in ⅛ - ½ -per-cent solution, two, three, or four times a day, the number of instillations and the strength of the solution being gauged by the effect on the tension and the pupil—the aim being to keep the latter continuously contracted. Owing to its unpleasant effect eserine is not much so used, although it is usefully combined in ⅛ - ¼ -per-cent solution with the pilocarpine, especially at night when its disagreeable action is less noticed. In addition, as Elliot points out, every means should be taken to prevent loss of vaso-motor control and to avoid the factors that make for the latter and the factors that may cause autogenous intoxication (overwork, worry, nervous and bodily exhaustion,

improper diet, all depressing conditions, including concealed sources of infection in the teeth, tonsils, nasal sinuses, intestinal tract, etc.).—D.]

The mydriatics are as prejudicial in glaucoma as the miotics are useful.

[Ziegler in acute, especially fulminating glaucoma, has had good results from the hypodermic injection of *hyoscine* (0.6 mg.) followed by an injection of *pilocarpine* (8 to 10 mg.). The former, he thinks, arrests a perverted lymph secretion, the latter stimulates a normal flow of lymph. After this combined treatment, eserine will sometimes act, when previously ineffective.

The adherents of the hydrophilous theory (see page 508) advocate the use of *sodium citrate* in glaucoma, but this treatment has not answered expectations. *Massage* of the eyeball reduces its tension, although less readily so in eyes predisposed to glaucoma (Elliot); and this has been advocated as a means of treating glaucoma (Pyle).—D.]

II. Secondary Glaucoma

459. By secondary glaucoma we understand an increase of tension which appears in the course of other diseases of the eye, and as a consequence of them. Accordingly, the increase of tension here forms the complication of an already existing affection, although, all the same, it entails the results peculiar to itself, just as in the case of primary glaucoma. If it is associated with inflammatory symptoms, it produces in the cornea, the iris, etc., the changes belonging to inflammatory glaucoma. In other cases it manifests itself merely through the increase in tension perceptible to the touch, and also through the pressure excavation of the optic nerve with the disturbance of sight that is caused by it—namely, the contraction of the visual field and the diminution in central vision. Moreover, its termination in blindness and in degeneration of the eyeball is the same as in primary glaucoma.

460. Etiology.—The clinical picture of secondary glaucoma varies according to the disease which it accompanies. The affections of the eye leading to increase of tension are as follows:

1. *Ectasiæ of the cornea and the sclera.* Of the former, it is above all those connected with incarceration of the iris—i. e., the staphylomata—that almost universally lead to secondary glaucoma. It is only the exception that ectasiæ without incarceration of the iris, such as keratectasia ex ulcere or keratectasia after pannus or after keratitis parenchymatosa, give rise to increase of tension. Of scleral staphylomata, those which occur after rupture of the sclera may entail increase of tension, and so also may the ectasiæ that develop after scleritis. Most ectasiæ of the sclera, however, are the result, not the cause, of the increase in tension.

2. *Incarceration of the iris* in a cicatrix of the cornea or sclera, and also the partial apposition of the iris against the posterior surface of the cornea may lead to increase of tension even without any ectasia being present.

3. *Irido-cyclitis*, especially in those cases in which, except the deposits

on the cornea, no exudates worth mentioning are found. The increase of tension in these cases is often only transitory.

4. *Seclusio pupillæ*, whether originating in adhesion of the entire pupillary margin to the capsule of the lens or in the inclusion of the former in a cicatrix of the cornea. Seclusio pupillæ leads to the accumulation of aqueous in the posterior chamber and to consequent protrusion of the iris, which is accompanied by increase of tension.

5. The *lens* becomes a cause of secondary glaucoma in two ways—by being luxated and by being swollen. All forms of luxation are of significance in this regard; but the most dangerous cases are those in which the lens is wedged into the pupil or lies wholly in the anterior chamber. Sudden swelling of the lens after injury or operation may likewise cause increase of tension, especially in the case of elderly people, whose scleræ are rigid. Increase of tension also occurs at times after cataract extraction and discission of secondary cataract.

6. *Intra-ocular tumors*, such as sarcomata and gliomata, in a certain stage of their development excite the symptoms of secondary glaucoma.

7. *Hæmorrhages into the retina* are the expression of changes in the vessels or disturbances of circulation, which sometimes lead to elevation of tension. This is most frequently the case in old people with arteriosclerosis; the elevation here usually makes its appearance under the form of inflammatory glaucoma—glaucoma hæmorrhagicum.

8. *Chorioidits* and *myopia* of a high degree often give rise to an increase of tension under the form of glaucoma simplex.

[Glaucoma due to *autogenous toxæmia*, which probably sets up a low-grade inflammation blocking up the channels of outflow (page 510), if not to be classed under secondary glaucoma, is at least akin to it (Elliot).—D.]

461. Factors Causing the Increased Tension.—These vary greatly according to the varying nature of the cases themselves, and very often we do not at all know how to account for the development of the increase of tension. [In some cases secondary glaucoma is due to *perverted secretion*—i. e., to alterations in the character and quality of the liquid poured into the eye. This is particularly the case in the form accompanying irido-cyclitis (Elliot).—D.] Other varieties of secondary glaucoma are due to *anatomical changes*. These are:

1. Obliteration of the sinus of the chamber due to the fact that the iris has been drawn forward and brought into contact with the cornea. This is probably the usual cause of increase of tension in the case of anterior synechiæ, by which the iris is drawn forward and brought close to the cornea, and the more so the more peripheral the site of the incarceration of the iris. The narrowing or the blocking of the sinus of the chamber in these cases does not extend all the way around as in genuine glaucoma, but is confined to the sector which corresponds to the anterior synechia (Fig. 119). Whether in this case increase of tension sets in or not depends upon whether the portion of the sinus that is still free is adequate for the excretion for the liquids of the eye. After

an iridectomy the stump of iris which remains at the site of the coloboma may push itself into the wound, become united there, and to a corresponding extent block the sinus. After an extraction of the lens made with the iridectomy the incarceration of a tag of capsule may have the same result, the capsule jamming the iris stump against the corneo-sclera.

2. Pushing of the iris against the cornea by a greatly swollen or dislocated lens or by a tumor of the ciliary body.

3. Agglutination of the iris to the posterior surface of the cornea after the two have been for a long time kept in contact because of a corneal fistula.

4. Attachment of the periphery of the iris to the corneo-scleral junction because of inflammatory adhesion, as occurs in many cases, and especially in the chronic cases, of irido-cyclitis.

5. Blocking of the sinus of the chamber due to infiltration of the ligamentum pectinatum, to cysts of the iris, and to tumors of the iris.

6. Blocking of the sinus of the chamber by tissue which has remained there since fetal life. This is the case in aniridia, in which the iris is never completely absent, but is simply reduced to a short stump, in the angle between which and the corneo-sclera the tissue above mentioned is situated. In hydrophthalmus the increase of tension probably depends on a condensation of the tissue of the ligamentum pectinatum, which originates in fetal life and is frequently associated with absence of the canal of Schlemm.

7. Impermeability of the ligamentum pectinatum due to blocking of its open spaces by blood, leucocytes, or precipitates from the aqueous. It is changes of this sort that probably cause the increase of tension in recent cyclitis, or when many lens fragments are present in the aqueous. It is also possible that in cases of this sort the filtration of the aqueous may be hindered by the greater proportion of albumin that the latter contains, even without there being any obstruction in the spaces of the ligamentum pectinatum.

8. Impermeability of the ligamentum pectinatum due to the formation of an epithelial lining (anterior-chamber cysts—page 445), e. g., after cataract operation.

9. Abolition of the communication between the posterior and anterior chambers in the case of an adhesion of the entire margin of the pupil to the lens or to the cornea (seclusio pupillæ). In luxation of the lens into the anterior chamber, a cut-off action of this sort may result because the iris is jammed against the posterior surface of the lens by the aqueous which has been secreted by the ciliary processes, so that the lens acts like a ball valve (Fig. 240).

10. Elevation of the pressure in the vitreous cavity produced by venous stasis in thrombosis of the retinal veins (glaucoma hæmorrhagicum) and in blocking of the venæ vorticosæ (by tumors, by endophlebitis, and also in experimental ligation of the venæ vorticosæ).

11. Elevation of the pressure in the vitreous cavity due to increase in the volume of the vitreous; occurring suddenly in hæmorrhage or exudation (panophthalmitis), and gradually in tumors.

12. Elevation of the pressure in the vitreous cavity due to protrusion of the retina or chorioid into it as the result of hæmorrhage, exudation, or the formation of new growths in these membranes.

462. Treatment.—The treatment of secondary glaucoma must above all endeavor to remove the cause underlying the elevation of tension. For instance, in seclusio pupillæ the communication between the two chambers should be restored by means of an iridectomy, a dislocated or swollen lens should be removed, if possible, and so on. For the symptomatic treatment

of the increase of tension itself, miotics, dionin, free diaphoresis, or quinine internally may be considered according to the exigencies of the case. Of operations those at our command are paracentesis of the cornea and iridectomy. The former diminishes the pressure by letting out the aqueous, but does so only temporarily, so that it is suited simply to those cases in which the increase in tension is foreseen to be of short duration—e. g., in swelling of the lens and in irido-cyclitis. The paracentesis may be repeated several times, according to the demands of the case. A lasting elevation of tension can be combated only by iridectomy or sclerectomy. Glaucoma hæmorrhagicum gives the most unfavorable prognosis. Here we cannot count with certainty upon the effect of iridectomy, since sometimes it is immediately succeeded by blindness coming on rapidly and with great pain. Eyes which contain a new growth, or which are both blind and painful, require enucleation.

Hypotony

463. Diminished Intra-Ocular Pressure (Hypotony).—This is found in very diverse affections of the eyeball. It is always a sign that the contents of the eyeball have diminished in volume.

Hence a high degree of diminution of tension is observed when, after the perforation of the eyeball, either the aqueous has flowed away or the lens or vitreous has escaped. This may be the result of an injury or of the spontaneous perforation of an ulcer. If the perforation in healing leaves a fistula or a cystoid cicatrix, through which aqueous continually escapes, the softness of the eye may persist for a long time (even many years). After the use of a bandage which has been too tightly applied, we find the eye softer for a short time, because, under the increased pressure due to the bandage, an increase in the outflow of fluids from the eye has taken place. So, too, the eye becomes very soft when the volume of the vitreous is diminished by shrinking of exudates—and hence, in those cases in which an atrophy of the eyeball is developing after irido-cyclitis. Hence progressive softening of the eyeball in the course of an irido-cyclitis is an ominous symptom. Slight degrees of decrease of tension accompany many cases of inflammation of the cornea, both suppurative and non-suppurative, and also occur not infrequently after slight injuries (erosions) of the cornea, especially if these were associated with a contusion. Of the affections of the deep parts, detachment of the retina is particularly associated with diminution of tension. Finally, slight degrees of the latter are found in paralysis of the sympathetic, and also after the instillation of cocaine, pilocarpine, and eserine (page 19).

Cases occur which are known as *ophthalmomalacia*, or essential phthisis, in which diminution of tension appears spontaneously, without known cause. The eye suddenly becomes very soft, smaller, and injected, and not infrequently marked photophobia and neuralgic pain are associated with the condition. This state of things may last for hours or days, when it gives place to the normal condition. In many instances such attacks recur at intervals (intermitting ophthalmomalacia). The cause of this rare disease in many cases remains unknown; in other cases there has been an injury preceding it. The prognosis is good, as the ophthalmomalacia usually leaves no lasting ill results behind.

CHAPTER VIII

DISEASES OF THE LENS

Anatomy

464. The lens (crystalline body, lens crystallina) lies between the iris and vitreous, and, together with the zonula, divides the eye into a smaller anterior and a larger posterior section—the cavity of the aqueous and the cavity of the vitreous. It is a transparent and colorless structure of lenticular shape, the anterior surface of which is less, the posterior surface more curved. In the lens we distinguish an anterior and a posterior pole, and the rounded edge, or equator, where the anterior and posterior surfaces of the lens meet. The sagittal diameter (thickness of the lens) amounts in the adult man to 5 mm., the equatorial diameter to 9 mm.

The lens lies within the circle formed by the ciliary processes, but in such a way that its equator is distant about 0.5 mm. from the apices of the processes. The interspace between the ciliary body and the equator of the lens is called the circumlental space. The posterior surface of the lens is embedded in the fossa patellaris of the vitreous. The lens is kept in position by the suspensory ligament, or zonula ciliaris.[1]

If after rupturing the zonula we take the lens out of the eye, we find it, in the first place, inclosed in a transparent capsule—the capsule of the lens. If after removing the capsule we try to crush the lens of an elderly man between the fingers, the softer peripheral masses separate, while the harder central portion remains uncrushed between the fingers. The former form the cortex, the latter the nucleus of the lens (see Fig. 172, *r* and *k*). These are distinguished not only by their consistence but also by their color. The cortex is colorless, while the nucleus has a yellowish or brownish hue. The nuclear layers owe their greater consistency and also their coloration to a process which is known as sclerosis, and which consists mainly in a loss of water. The sclerosis begins even in childhood, but advances so slowly that it is not until the age of twenty-five that a distinct, although still small, nucleus is present. Since sclerosis of the lens fibers is a change due to advancing age, it affects first the oldest fibers—i. e., those that lie in the center of the lens. By a continuous progress of the sclerosis from the center to the periphery of the lens, the nucleus steadily increases in size as the years go on, and the cortex diminishes in like proportion, so that at length, at a very advanced age, almost the entire lens is converted into nucleus, or is sclerosed. There are many individual differences in this regard, so that persons of the same age have lenticular nuclei of different size. The size of the nucleus is of practical importance in the operation for cataract.

[1] Synonyms: Zonula Zinnii, ligamentum suspensorium lentis.

The sclerosed portion of the lens is hard and rigid, incapable of changing its shape. Hence, the further advanced the sclerosis of the lens is, the less able is the latter to make that alternating change in its shape which is requisite for the act of accommodation. For this reason the accommodative power diminishes with advancing age (presbyopia; see § 762).

The nucleus reflects more light than the non-sclerosed part of the lens. Hence, the pupil in elderly people, whose lens has a large nucleus, is no longer of such a pure black as in youth. It gives a gray or grayish-green reflex (the senile reflex), which by the inexperienced is easily confounded with beginning cataract

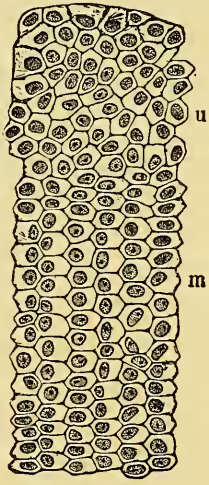


Fig. 220.

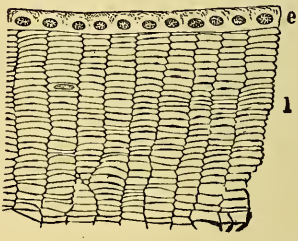


Fig. 221.

Fig. 220.—Capsular Epithelium of the Cortex. (After Rabl.) The epithelium which was left attached to the capsule when the latter was stripped off is seen in surface view. The area represented lies directly in front of the nuclear zone of the lens. In front of the latter the epithelial cells, which further forward are disposed irregularly (*u*), become arranged in regular meridional rows, *m*.

Fig. 221.—Equatorial Section Through a Human Lens. (After Rabl.) *e*, epithelial cells of the anterior capsule; *l*, cross section of the lens fibers which are arranged so as to form radiating lamellæ. In one of these fibers an oblong nucleus is visible.

465. Histology of the Lens.—The external envelope of the lens is formed of the lens capsule. This is a homogeneous membrane (Fig. 222, *l*), which is thicker upon the anterior than it is upon the posterior surface of the lens. The anterior capsule of the lens is further distinguished by having a single layer of cubical epithelial cells, the epithelium of the lens (*e*, Fig. 222). This plays an important part in the growth of the lens, as the fibers of the latter originate from the cells of the capsular epithelium. If we follow the epithelium of the anterior capsule toward the equator, we see that at this point the epithelial cells, which were irregularly disposed before now get to be arranged in meridional rows (Fig. 220, *m*). Then these epithelial cells become taller and taller, until finally they are converted into long fibers, the fibers of the lens (Fig. 222, *f*). Since the latter have originated from merid-

ional rows of epithelial cells, they are arranged in radiating lamellæ [Fig. 224], which explains why opacities of the lens so often occur under the form of radial striæ. As the cells become elongated their nuclei recede from the capsule into the interior of the lens, so that a zone is found along the equator, in which there are numerous nuclei lying in the lens substance itself.

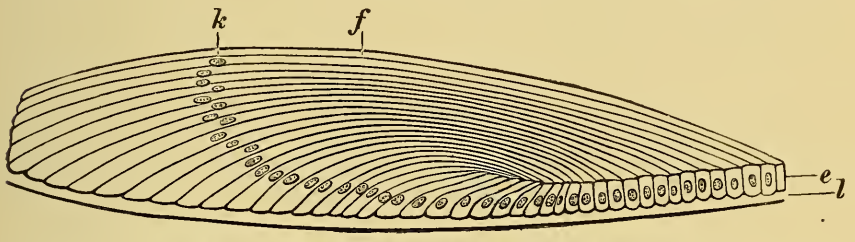


FIG. 222.—NUCLEAR ZONE OF THE LENS. (After Babuchin.)

l, lens capsule. The epithelial cells, *e*, by a process of gradual elongation, grow out into the lens fibers, *f*, with the nuclei, *k*.

This nuclear zone, as it is called (*k*, Fig. 222; cf. also Fig. 142, *k*), represents that district of the lens in which the growth of the latter takes place. This growth occurs by a process of apposition, new epithelial cells constantly growing out into lens fibers, which are placed outside of and next to the older lens fibers. In this way the lens acquires a concentrically laminated as well as a radial structure. The fibers lying in the center of the lens are thus the oldest, and the most exterior fibers are the youngest. The reason for nuclei not being present outside of the nuclear zone in the interior of the lens is that the nuclei disappear from the older lens fibers.

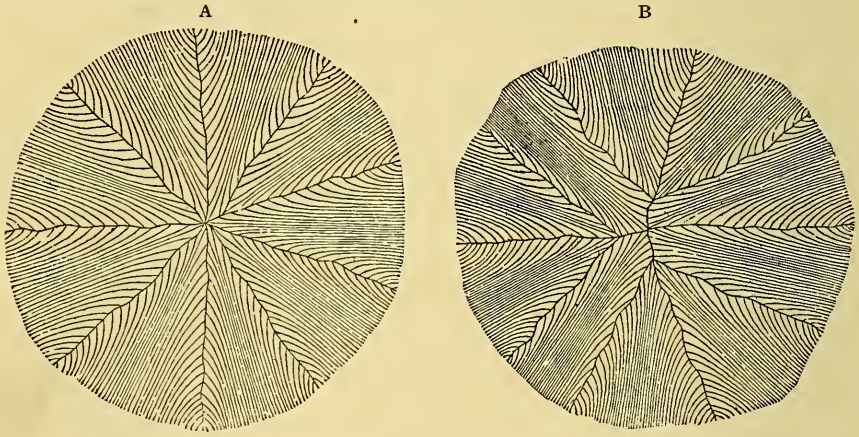
FIG. 223.—STELLATE FIGURE OF THE POSTERIOR SURFACE OF THE LENS. DRAWN FROM A LENS HARDENED IN MÜLLER'S FLUID. Magnified 2 × 1.

From the posterior pole of the lens start three primary rays, one of which is directed straight downward, the other two inward and upward and outward and upward. These divide into their branches so near their origin, in this case, that the Y-shaped figure is here not at once obvious.

The lens consist of fibers having the form of long, prismatic, six-sided cords. They are closely applied to each other, and are held together by a cement substance. The fibers begin and end upon the anterior and posterior surfaces of the lens, along lines which radiate from the anterior and posterior poles (Figs. 223 and 224). Here they form a Y-shaped figure—the stellate figure of the lens—which can be recognized even in the living eye in adults by means of lateral illumination. The three rays of the stellate figure branch, and thus divide the lens into a number of sectors [see Fig. 224] whose apices meet in the region of the anterior and posterior poles of the lens. In pathological cases—i. e., in opacities of the lens—the sectors often stand out very distinctly. The fibers of the nucleus are distinguished from those of the cortex by being slenderer and having edges that, owing to the shrinking

of the fibers, are finely crenated; there is, however, no sharp line of distinction between the nucleus and cortex.

466. Development.—The structure of the lens is easy to understand, when we know its development. The lens springs from the ectoderm, which becomes invaginated so as to form a vesicle (*L*, Fig. 161). Since the coating of cells upon the posterior wall of the vesicle grows out and is used up in the formation of lens fibers (Fig. 162), no such coating is found in this situation later on; hence, the posterior capsule of the lens has no epithelium. By this outgrowth of cells and their transformation into long fibers the vesicle is filled up so as to form a solid sphere. In this sphere each of the newly formed fibers extends from the anterior to the posterior lens capsule (Fig. 162). Similarly in the adult lens each individual fiber stretches from a ray



[Fig. 224.—Lens-Star. (After Babuchin.) From Norris and Oliver.

A, central portion of anterior lens-star; B, central portion of posterior lens-star. In both figures are seen the lens fibers terminating in the lines of junction of the sectors.—D.]

of the posterior to a ray of the anterior lens-star. The subsequent growth of the lens by apposition continues, as in the case of other epithelial structures, during the entire life. But while in the other epithelial structures (e. g., the epidermis, hair, and nails) the exfoliation of the oldest cells serves to maintain a state of equilibrium, no such exfoliation is possible in the lens, which is completely shut in; and in this case compensation takes place by a diminution in volume of the oldest fibers through a process of shrinking (formation of the lens nucleus). This diminution in volume, however, does not fully offset the appositional growth, so that the lens keeps on enlarging even in advanced life. In the sixty-fifth year of life it has about one-third more volume than in the twenty-fifth (Priestley Smith).

467. Zonula Ciliaris.—The zonula ciliaris consists of delicate, homogeneous fibers, which take their origin from the inner surface of the ciliary body, beginning at the ora serrata. The fibers at first keep in contact with

the surface of the ciliary body (*z*, Fig. 142), but leave it at the apices of the ciliary processes, and, becoming free, pass over to the edge of the lens (free portion of the zonula; z_1, Fig. 142). As they do this, they diverge so that some go toward the very equator of the lens, others in front of the equator and behind it, to reach the lens capsule, with which they become fused. The space, triangular on cross section, included between the fibers of the zonula and the equator of the lens, is called the canal of Petit (*i i*, Fig. 142). It is connected with the posterior chamber by means of slit-like gaps between the separate fibers of the zonula.

468. Function of Lens.—The optical function of the lens consists in its bringing the rays that have been already made convergent by the cornea still closer together, so that they unite upon the retina. For this purpose the refractive power of the lens has to be less or greater, according as the rays are parallel or divergent when they fall upon the eye. This alteration in the refractive power (accommodation) is produced by a change of shape of the lens (see § 758).

In regard to the metabolism of the lens, see page 11.

I. Opacities of the Lens

A. General Considerations

469. Nature and Site of Cataract.—Opacities of the lens, called cataract,[2] may be situated in the lens itself or in the capsule. Accordingly, we distinguish between lenticular and capsular cararacts; by the combination of the two is produced capsulo-lenticular cataract.

Historical.—Cataract was already well known to the ancient Greek and Roman physicians. On account of the gray appearance of the pupil, they denoted it by the name of glaucoma, which word in the course of time has changed its meaning. The ancients also knew the operation for cataract, which they performed by depressing the opaque lens into the vitreous by means of a needle (depressio cataractæ). Nevertheless, they had an erroneous conception of the nature of the disease, in that they located the opacity not in but in front of the lens. This error originated from the views that they held with regard to the function of the lens. This body, bright as crystal, the most obvious thing when the eyeball is opened, was considered by the ancients to be the true seat of vision, the percipient organ, such as now we know the retina to be. According to this view, the loss of the lens would necessarily entail complete blindness; but since the ancients knew that in the operation for cataract the opacity is removed from the pupil, and nevertheless the sight is not lost, but, on the contrary, is restored, they could not consistently regard the opacity as located in the lens. They thought the opacity which they depressed into the vitreous was situated in front of the lens. They believed that it originated from the pouring out of an opaque liquid between the iris and lens, and hence they called cataract hypochyma (ὑπό, beneath, and χέω, I pour) or suffusio, suffusion. Since it was imagined that the opaque liquid fell down from above in front of the lens, the name cataracta (cataract), which still is usually employed, came into use in the middle ages. The German word "Staar" is likewise very old. The expression staraplint (Staarblind) occurs as early as the eighth century.

[2] Waterfall, from καταῤῥήγνυμι, I pour down.

It means really eyes which are starr (rigid)—i. e., fail to follow objects because they do not see them. Cataract is known as "grauer Staar," on account of the gray color of the pupil, to distinguish it from "schwarzer Staar"—i. e., those varieties of blindness in which the pupil remains black (blindness due to disease of the fundus of the eye). "Grüner Staar" is glaucoma.

Our knowledge as to the true nature of cataract dates from the beginning of the last century. Even before this, one or two savants, like Mariotte and Boerhaave, recognized the real situation of the opacity, without their doctrine's, however, obtaining general acceptance. In the year 1705, Brisseau, a French surgeon, had the opportunity of performing an autopsy upon the body of a soldier who had a mature cataract in his eye. Brisseau performed depression of the cataract upon the cadaver and then opened the eye, when he found that the opacity which he had depressed into the vitreous was the lens. He laid his observations, together with the conclusions drawn from them, before the French Academy, but obtained no credence. The Academy confuted him by holding up the doctrines of Galen in regard to cataract. It was not till three years later, when new proofs had been brought, that the Academy recognized the new doctrine, which soon found general acceptance.

470. Objective Signs.—The objective symptoms of lenticular opacity vary according to its extent and its intensity—partial opacities often requiring for their recognition lateral illumination or the use of the ophthalmoscope, and, if the opacities lie far in the periphery, artificial dilatation of the pupil as well. By reflected light (with focal illumination) the lenticular opacities present themselves under the form of gray or white spots or striæ. These often exhibit shapes which have a connection with the structure of the lens—e. g., the shape of sectors or radii. By lateral illumination it can be determined at what depth the opacities are situated in the lens. Opacities of the anterior capsule are distinguished by their brilliant white hue, sharp outline, and very superficial situation; sometimes they form a distinct prominence upon the anterior surface of the lens. When seen with the ophthalmoscope—that is, by transmitted light—the lenticular opacities do not appear white, but dark, like black dots or striæ, which stand out in contrast with the red hue of the pupil (see page 92). Commencing, slight opacities of the lens can be recognized only by means of the ophthalmoscope. Far advanced opacity of the lens can be recognized at a glance with the naked eye by the change of color of the pupil, which is white or a gray of varying degrees of brightness.

In former times when focal illumination and the ophthalmoscope were unknown, one, in making a diagnosis of commencing cataract, had to depend mainly upon the subjective symptoms, especially myodesopsia (the seeing of muscæ volitantes), which was therefore much more exactly studied and worked up than at present. At that time it was quite possible for pupillary membranes, which made the pupil appear gray or white, to be regarded as opacity of the lens, and they were therefore called cataracta spuria. We shall not fall into this mistake if we observe the connection which a pupillary membrane almost always has with the margin of the pupil, and which is particularly marked when we call in the aid of atropine. But, even with all our present auxiliaries, it is often impossible to say whether the lens behind a dense pupillary membrane is transparent or opaque.

471. Subjective Symptoms.—The subjective symptoms of opacity of the lens consist in a disturbance of vision, the degree of which depends upon the situation and the nature of the opacity. Patches of cloudiness that are small, sharply circumscribed, and at the same time as opaque as possible—as, for example, anterior polar cataract—cause little or no impairment of the sight. Larger opacities disturb the sight to a considerable degree, and, moreover, alarm the patient by the production of peculiar phenomena, such as muscæ volitantes and polyopia. The seeing of *muscæ volitantes* (mouches volantes) consists in the patient's noticing black specks in the field of vision, which, however, if caused by opacities of the lens, change their place only with the movements of the eye, and hence (in contradistinction to opacities of the vitreous) always occupy the same spot in the field of vision. They become objects of cognition by casting a shadow upon the retina, which is perceived by the latter. Multiple vision (*polyopia monocularis*) causes the patient to see the same object double and multiple. It may sometimes have a very disturbing effect, as a case related by Becker shows. A lamplighter in the castle of a prince, when he lighted the candelabra and chandeliers in the salons the evening before a soirée saw thousands of lights, which confused and frightened him to such a degree that he got the idea that he was dealing with a ghost. The reason for the polyopia is found in the optical irregularities which develop in the lens as it grows opaque (irregular lenticular astigmatism), so that the lens throws upon the retina not one but several images of the same object. These phenomena often bring the patient to a physician at a time when as yet no considerable diminution of the sight exists.

The diminution in *visual acuity* depends, with regard to its degree, upon various circumstances. It is greater when the opacity is diffuse, less when it is sharply circumscribed, so that quite clear interspaces are found between very opaque spots. This case is the same as with a window-pane, through which nothing can be distinguished when it is uniformly covered with watery vapor; although, if a wire screen is placed in front of the otherwise clear pane, we can still see pretty well through it. The interference with vision is also greater when the opacity is situated in the central portions of the lens than when it occupies the periphery. In the latter case, in fact, the sight may be perfectly normal; this being particularly the case as long as the opacities continue to lie completely behind the iris. Upon the situation of the opacities furthermore depends the sort of illumination that will be required in order for the patient to see the best. With a central opacity the sight is better when the pupil is dilated, because the still transparent, peripheral portions of the lens are then used for seeing. Persons thus affected, therefore, see better when the illumination is reduced, as in the evening twilight; they have *nyctalopia*. In bright daylight they complain of being dazzled, and shade their eyes with the hand. In such a case the vision can also be

improved by the artificial dilatation of the pupil by means of atropine. The reverse occurs when the opacities occupy the periphery of the lens. Then vision is better when the pupil is contracted, so that the opacities are covered by the iris. Such patients try to get a bright light, and see better by day than by night—*hemeralopia.*

Later on, as the opacity increases, the sight becomes more and more reduced, the muscæ volitantes and the polyopia disappear, and the patient grows blind. But even when he has lost the ability to distinguish objects (qualitative vision), he has still always left him the perception of light—the distinction between light and darkness, or quantitative vision. The examination of the perception of light (see § 754) is of great importance with regard to the prognosis of a complete opacity of the lens. If the perception of light is deficient or entirely wanting, this proves the existence of a complication on the part of the retina or the optic nerve, in which case an operation for cataract would have little or no result.

Myopia often develops in the beginning of senile cataract. In this case we are dealing with elderly people, who formerly saw well at a distance and used convex glasses for reading; and who then began to notice that they could read fine print again without glasses, and are perhaps very much pleased at this so-called "second sight." That, as an off-set to this, they do not see as well at a distance as formerly, often escapes their notice. Examination of the eye with glasses shows, that it has become myopic, so that the near point has got back again to the reading distance. This myopia is ascribable to an increase in the density of the lens that takes place while the cataract is beginning to form, an increase by which the refractive power of the lens is heightened.

An opacity of the same character causes more disturbance of vision when it is situated at the posterior than at the anterior pole of the lens; for the nodal point of the eye—i. e., the point through which all rays must pass that enter the eye without undergoing refraction (principal rays)—lies close to the posterior pole of the lens (cf. § 752, Fig. 362).

472. Morbid Anatomy.—The anatomical changes forming the basis of lenticular opacity have been chiefly studied in senile cataract. Becker is the one who by his profound researches has done the most to advance our knowledge of the development of cataract. The changes affect the capsular epithelium and the lens fibers.

In the *capsular epithelium* we find destruction of the epithelium cells here and there, and side by side with this, what may be looked upon as a regenerative process, namely, a growth of epithelial cells. This latter process, however, takes place irregularly, so that close by the gaps in the epithelial layer spots are present in which several rows of cells lie on top of each other (Fig. 225, e_1). Moreover, the epithelium often grows backward past the equator, so that the posterior capsule, too, gets to have an irregular epithelial coating on its anterior surface.

The opacity in the *lens* itself begins sometimes directly beneath the capsule, sometimes deeper in, at the border line between the nucleus and cortex. Here by separation of the lens fibers there are formed clefts filled with liquid. [These clefts have been attributed to irregular shrinking. As the lens hardens, the soft cortex normally adapts itself to the changing size of the hard nucleus. In a lens that is becoming cataractous it is

supposed that shrinking occurs too fast or too irregularly for this adaptation to take place, in which case the lens fibers separate and clefts are formed.—D.] The lens fibers themselves, which bound the clefts, are at first still normal and hence transparent. The fluid which collects between them may at first be transparent, too, and nevertheless the spots look cloudy, because the fluid in the clefts has a different refractivity from that of the lens substance itself. So, too, a white opaque foam is produced when we mix transparent air with equally transparent water by agitation. Afterward the lens fibers themselves become cloudy. They look at first as if sprinkled with fine dust, owing to the accumulation in their interior of a fatty substance in extremely minute drops. At the same time that the lens fibers get cloudy, their caliber becomes uneven, because they swell up in spots (*q*, Fig. 225). In this way bodies that are large, vesicular, and frequently nucleated (vesicular cells; *b*, Fig. 225) are produced. Finally, the lens fibers break down completely, so that from the lens tissue is formed a pultaceous mass consisting of drops of fat, the so-called spheres of Morgagni (a coagulation product, Fig. 225, *M*)

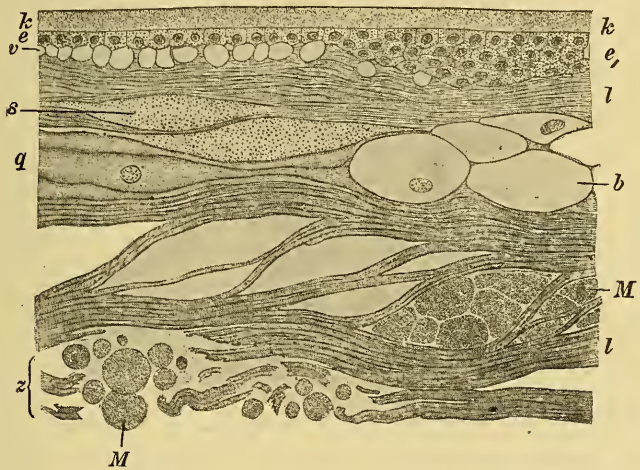


Fig. 225.—Cataracta Capsulo-lenticularis. Enlarged 170 × 1.

k, anterior capsule of the lens; *e*, epithelium, occurring at e_1 in several layers because of proliferation; *l*, normal lens fibers; *v*, light-colored vacuoles (drops of liquor Morgagni) between *l* and the epithelium. The fissures originating through the separation of the lens fibers are filled with a granular mass (coagulated fluid), *s*, which in places forms the spheres of Morgagni, *M*. The lens fibers themselves are swollen up (*q*), or transformed into vesicular cells (*b*), or entirely disintegrated (*z*).

remains of lens fibers, and an albuminous liquor (Fig. 225, *z*). As the lens fibers break down, the connection between them and the capsule, which is a very intimate one in the normal lens, is loosened, and a liquid (the liquor Morgagni) collects in open spaces between the lens and capsule (at *v* in Fig. 225, appearing under the form of separate vacuoles, but at *r*, Fig. 172, accumulated in greater amount and separating the capsule from the cortex). By this process the removal of the lens from the capsule, as has to be done in a cataract operation, is facilitated.

The nucleus of the lens is transformed by the sclerosis of the lens fibers into such a resistant mass that generally it remains unchanged in the midst of the disintegrating cortex (*k*, Fig. 172). Hence the nucleus of a cataractous lens is usually not essentially different from the nucleus of a healthy lens of the same period of life (Becker). But, if there is no hard nucleus present yet, the disintegration of the lens is complete (Fig. 173).

The subsequent changes in the opaque and disintegrated lens consist in the first place in a gradual resorption of the pultaceous lens masses. In this way lenticular

opacities may clear up again; not, to be sure, in the proper sense of the word, by the opaque lens fibers' becoming once more transparent, but by the disappearance of the opaque parts. The sclerosed nucleus resists resorption as it does disintegration. Cholesterin is not infrequently excreted in the cortex in tabular crystals, which are sometimes large enough to be visible to the naked eye as glistening points. Lime salts may be deposited in the pultaceous lens masses.

Capsular opacity is not situated in the capsule itself, which never becomes opaque, but is deposited upon the capsule. Opacities of the anterior capsule are caused by an opaque tissue which is found on the inner surface of the capsule, between it and the lens (Fig. 226). This tissue takes its origin from a proliferation of the capsular epithelium. The cells of this latter increase in number so that a multiple layer of them is formed (Fig. 225, e_1). From this layer there is formed by the growth of the epithelial cells into elongated fibers a sort of fibrous tissue which looks like connective tissue, but still is not true connective tissue, since it has originated from epithelium. By the interposition of this tissue between the capsule and the lens a distinct elevation is formed upon the anterior surface of the lens.

The opacities of the posterior capsule lie, as a rule, upon its posterior surface.

Inflammation of the lens—*phakitis*[3]—does not exist. Inflammatory elements, such as round cells, occurring in the lens, do not originate in it, but enter the lens from the outside through an opening in the capsule.

B. Clinical Forms of Cataract

473. Every opacity begins at first at some special spot in the lens (partial cataract). It may remain permanently limited to this spot (partial stationary cataract), or it may gradually spread over the whole lens and lead to total cataract (progressive cataract).

(a) PARTIAL STATIONARY CATARACT

474. (1) *Cataracta Polaris Anterior.*—A small white dot is seen at the anterior pole of the lens. Anatomical examination has proved that it represents an opaque tissue lying beneath the anterior capsule and between it and the lens—that is, that we are dealing with a capsular cataract (Fig. 226). Hence this form of opacity is also called anterior central capsular cataract.

Anterior polar cataract may be either congenital or acquired. Congenital anterior polar cataract is bilateral, and consists of a minute faint dot upon the anterior pole of the lens. It is caused by some interference with the development of the lens, the precise nature of which has not been determined. The acquired form originates from a central ulcer of the cornea. When such an ulcer perforates and the aqueous escapes, the lens pushes forward so that its umbo comes into direct contact with the suppurating cornea. From the latter toxic substances diffuse through the capsule of the lens and destroy its epithelium. Afterward a reactive proliferation of the adjoining epithelium, which has been preserved, takes place together with the formation of a new tissue beneath the capsule, this tissue appearing under the form of a white spot.

The apposition of the lens to a perforation in the cornea leads to ante-

[3] From φακός, lentil.

rior polar cataract only in the case of small children, never in adults. The most frequent cause of perforation of the cornea in early childhood is ophthalmia neonatorum, and hence this latter is to be looked upon as the ordinary cause of anterior polar cataract.

Acquired anterior polar cataract is bigger and more densely white than is the congenital form. Sometimes the layer of opaque tissue that lies beneath the capsule and corresponds to the cataract is so extensive as to produce a distinct visible conical protrusion of the anterior pole of the lens. This is called a pyramidal cataract (Fig. 121, *p*).

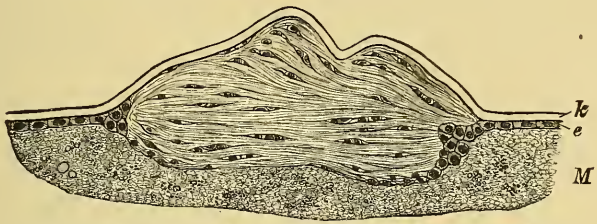


FIG. 226.—ANTERIOR CAPSULAR CATARACT. Magnified 40 × 1.

The capsular cataract forms a projection upon the anterior surface of the lens, covered by the capsule, *k*, which is unchanged and simply thrown into folds. The capsular epithelium *e*, loses its regularity at the border of the cataract, its cells being increased in number and separated by the cataract from the capsule, so as to form for a short distance the posterior boundary of the cataract. The cataract consists of a fibrous tissue, with cells lying in the spindle-shaped gaps between the fibers. Succeeding the cataract posteriorly, is liquor Morgagni, *M*, which is coagulated into a pulverulent mass, separating the capsule from the cataractous layers of the lenticular cortex (which are not represented in the drawing).

Anterior polar cataracts of small area may exist without giving rise to any essential disturbance of vision, this generally being caused more by the opacity of the cornea than by that of the capsule. Treatment, therefore, is generally not required except in those rare cases in which the cataract is so large as to occupy almost the whole of the pupil when the latter is contracted. Then an iridectomy would be indicated.

An anterior polar cataract is sometimes joined by a filament of connective tissue with a central cicatrix of the cornea. This takes its origin from the time when the lens was applied to the cornea after the perforation of the ulcer. The lens and cornea were then glued together by a mass of exudation, which may afterward become organized and be drawn out into a long filament when the anterior chamber is reconstituted. Generally the filament ends by rupturing, but exceptionally it may persist all through life and connect the corneal cicatrix with the anterior pole of the lens.

There are cases of anterior polar cataract in which the corneal cicatrix that is left by the ulcer does not lie in the pupillary area, but in the periphery of the cornea. Hence, it follows that the perforation must have occurred at some point not directly in the center of the cornea. Indeed it appears that in rare cases deeply penetrating ulcers may cause an anterior polar cataract even when there is no perforation. Of course, then the toxins formed in the cornea cannot act so intensely on the lens as when the lens comes in direct contact with the suppurating cornea, and hence a capsular opacity develops but rarely in such cases. That a capsular opacity always occupies the anterior pole is accounted for by the fact that in the area of the pupil—which is very narrow as long as the inflammation lasts—the lens capsule is directly exposed to the deleterious action, while in the rest of its extent it is protected by the iris.

Corneal opacities acquired in very early childhood often clear up to an extraordinary degree, so that in anterior polar cataract it is not always a dense cicatrix, but often only a slight cloudiness of the cornea, that is found. If this latter is overlooked, the method of development of the cataract might become a matter of doubt.

Anterior polar cataract, through subsequent contraction of the newly formed tissue, sometimes causes a wrinkling of the adjacent portions of the anterior capsule, visible upon examination with a magnifying glass. Beneath the capsular opacity disintegration of the adjacent lens fibers sometimes takes place. In such a case opacity of the entire lens (total cataract) may afterward ensue.

475. (2) *Cataracta Polaris Posterior.*—This consists of a small white dot at the posterior pole of the lens (Fig. 231), which, on account of its deep location, is generally to be discovered only with the ophthalmoscope. It belongs to the posterior capsule, and is hence also called posterior central capsular cataract. Posterior polar cataract is congenital, and dates from the time when the hyaloid artery passed through the vitreous to the posterior pole of the lens (see page 392 and Fig. 162). When this disappears incompletely, some of its tissue remains upon the posterior capsule. Hence, we sometimes find posterior polar cataract simultaneously with persistence of the hyaloid artery. The interference with vision is inconsiderable when the cataract is small. Treatment, none.

The anterior and posterior polar cataracts are capsular, while the partial stationary cataracts about to be mentioned are all lenticular.

476. (3) *Circumscribed Opacities of Various Kinds in the Lens itself.*—In this category belongs *central cataract*, a small spherical opacity directly in the center of the lens. The *cataracta fusiformis*, or spindle-shaped cataract, consists of an opaque line which runs in the axis of the lens from the anterior to the posterior pole, and presents a spindle-shaped swelling at a point corresponding to the center of the lens. In *cataracta punctata*, extremely minute white dots are found, either distributed uniformly through the whole lens or united in a group in the anterior cortical layer. Besides the forms just mentioned, numerous other forms of circumscribed stationary lenticular opacities are known, all of which, however, occur so rarely that they do not need to be minutely described here. All these opacities are sharply circumscribed, and are sometimes of very regular and graceful shape; they are congenital, and are mostly found in both eyes. They are often inherited, although the same forms of cataract are not always met with in the different members of the same family. Eyes affected with cataracts of this sort not infrequently present other congenital malformations too, or are found in individuals whose whole development, mental or physical, is imperfect. Most of these opacities in themselves cause little impairment of the sight, which, however, is often defective for other reasons.

477. (4) *Perinuclear (or Zonular, or Lamellar) Cataract.*—This is the most frequent form of cararact in children. After dilating the pupil, we see a gray, discoid opacity in the lens, surrounded by a perfectly transparent

marginal portion (Fig. 227). The diameter of the opaque disk is sometimes larger, sometimes smaller, and so, too, consequently, is the width of the peripheral, transparent zone (*P*). Upon examination with the ophthalmoscope, the cataract appears as a dark disk, surrounded by the transparent and therefore red and illumined periphery of the pupil. The darkness of the disk is greater near the edge than in the center, by which circumstances a zonular cataract is distinguished from a solid opacity, which, as it affects the nucleus too, would necessarily be densest in the center. Along the outline of the opacity, which usually is sharply defined, small opaque dentations are not infrequently found, called riders, projecting from the margin of the cataract into the transparent periphery, like the spokes on the steering-wheel of a steamship (in Fig. 227 represented only in the upper half of the cataract).

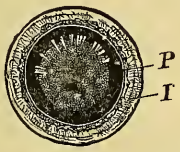


Fig. 227.

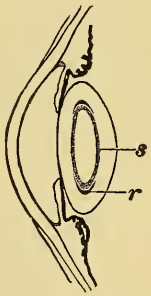


Fig. 228.

Fig. 227.—Lamellar Cataract Seen by Reflected Light. Magnified 3 × 2. The iris, *I*, has retracted under atropine. The opacity forming the lamellar cataract is denser at the margin than at the center. The riders are depicted in the upper half, but are left out below to show how a lamellar cataract looks without them. Between the margin of the opacity and the margin of the pupil, *P*, is a black interspace corresponding to the transparent periphery of the lens.

Fig. 228.—Lamellar Cataract in Cross Section. Schematic. Magnified 2 × 1. The layers *s*, placed between nucleus and cortex, are opaque, but the adjacent layer is so only in the equatorial region, *r*, so that riders are formed.

Lamellar cataract is caused by an opacity of the layers lying between the nucleus and cortex (Fig. 228, *s*), while these two themselves are transparent (Jäger). The riders on the periphery of a lamellar cataract signify that opacities are present in a second layer outside of the first, and that, moreover, they are present at first only at isolated spots along the equator of this first layer (*r*, Fig. 228). These partial opacities embrace the equator of the inner opacity in front and behind; they ride upon it, as it were, whence the name riders.

Lamellar cataract almost always affects both eyes. It is either congenital or, if not, originates in earliest childhood. It is found, above all, in children who have suffered from convulsions (Arlt). At the same time there are found residua of rickets in the bones and changes in the teeth (Horner). In very recent times lamellar cataract has been connected with the tetany of childhood (Peter), and to this also the convulsions have been attributed. Inheritance of lamellar cataract is not infrequent.

Perinuclear cataract is stationary as a rule, although there are cases in which it gradually develops into a total opacity.

The degree of interference with vision, due to lamellar cataract, does not depend upon the diameter of the opacity, for lamellar cataracts of even small diameter are always large enough to occupy the whole pupillary area of the lens, so that the transparent peripheral zone is always entirely concealed behind the iris when the pupil is not dilated. Hence, as far as vision is concerned, the denseness of the opacity is the only thing that has to be considered. As this latter varies greatly, all gradations are found between almost normal sight and considerable impairment of vision.

Treatment is required for lamellar cataract only when the interference with vision is considerable. In these cases there are two ways to choose from for improving the sight by operative means. The transparent periphery may be exposed and rendered available for vision by means of an iridectomy, or the lens may be removed altogether. The latter is accomplished in young people by discission; in older ones, in whom a hard nucleus is already present in the lens, by extraction. Each of these procedures has its definite indications, its advantages, and its disadvantages.

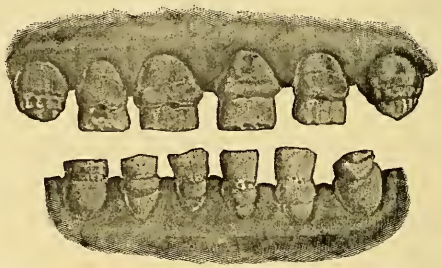


FIG. 229.—TEETH WITH HYPOPLASIA OF THE ENAMEL FROM A MAN WITH LAMELLAR CATARACT.

The teeth are yellow with rough surface. In most of them two furrows, separated by a protuberance, run around the crown. The protuberance represents an enamel of normal thickness, while in the course of the furrow the enamel is very thin. So too, the little pits which are to be seen most distinctly on the right upper canine tooth, indicate spots where the enamel is thinned.

Iridectomy is proper only when the peripheral, transparent zone of the lens is pretty broad. It retains for the patients the possibility of seeing at a distance and near by without glasses, but the visual acuity is never particularly good, since in fact the central opacity remains, and the iridectomy causes disfigurement by depriving the pupil of its round shape, and also gives rise to dazzling. On the other hand, removal of the lens produces a radical cure, and, in favorable cases, may raise the visual acuity to normal; the pupil too remains round and mobile. This operation, however, renders the patient exceedingly hypermetropic and deprives him of the ability to accommodate, so that he is compelled always to use glasses and is dependent on them all his life. Hence, we prefer iridectomy when the transparent periphery of the lens is broad enough to make distinct vision possible. To

establish the availability of the periphery of the lens for vision, the visual acuity is determined, first, with the pupil contracted, and then after its artificial dilatation with atropine. If the visual acuity is considerably increased in the latter case, iridectomy is indicated. [Where to place the iridectomy can be found by noting how a stop with pear-shaped aperture has to be placed before the dilated pupil for the patient to see best (see § 856).—D.] In all other cases removal of the lens is to be performed and this is the usual operation for lamellar cataract, since but few cases are suitable for iridectomy.

Horner propounded the view that lamellar cataract was the result of *rickets*—the latter being a disorder of nutrition which, besides affecting the bones, affects epithelial structures too, especially the teeth and the lens. The teeth, especially the incisor teeth, in the slight cases display horizontal rows of small depressions or horizontal furrows in the enamel (Fig. 229). When these defects are still more strongly marked, the tooth gets to have a sort of terraced shape, and is tapered down toward the incisor surface. Sometimes the teeth are so abortive in development that they are represented by small, cubical or irregular stumps. In the more serious cases the enamel coating is absent on the incisor surface or is even absent altogether; the dentin lies bare, and upon its rough surface a thick layer of yellow tartar is deposited. Owing to this deficiency in the enamel coating, the teeth become rapidly carious and break off, so that it is not unusual to find, especially in peasants, nothing of the incisor teeth except the broken-off stumps. The lens, which in its development has much in common with the teeth, is also supposed to undergo some disorder during a period of its development, while later, after the disappearance of the disorder, normal transparent layers of lens substance are again deposited.

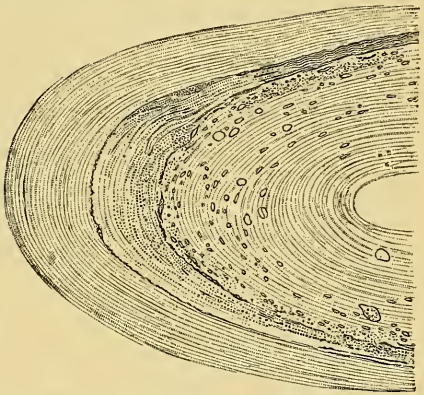


Fig. 230.–Lamellar Cataract. Magnified 12 × 1.

The specimen was from a man, fifty-seven years of age, who had suffered since childhood from lamellar cataract of both eyes. The diameter of the opacity, measured in the extracted lens, amounted to 6 mm. In the specimen the capsule and also the most superficial layers of the cortex are wanting, as they were left behind in the eye when the extraction was made. The layers lying between the nucleus and the cortex show numerous small cavities (droplets), which, in conformity with the fibrillary structure of the lens, are elongated, and are arranged concentrically. In some places they have coalesced so as to form larger cavities. Such droplets are found in the nucleus also, but in smaller number while the cortex is free from them. Besides the zone of vacuoles, which corresponds to the peri, nuclear opacity, two slender clefts are visible, forming arcuate lines, one of which is still within the zone of vacuoles, while the other runs along the peripheral border of the latter.

The discovery of the way in which lamellar cataract develops is rendered difficult from the fact that we do not at all know at what period of life it develops. The diameter of the lamellar opacity is so small as to form an argument for the intra-uterine origin of the cataract; on the other hand, in some cases it has been actually observed that a lamellar cataract originated after birth. But in every other case what we see is a lamellar cataract that is already fully formed, and we do not know when it developed. It is easy to undestand why this should be so. For the persons thus affected with it are not blind, but are simply weak-sighted; hence the affection from which they suffer does not generally make itself apparent until the time when greater demands are made upon the eyes—that is, in the first years of instruction at school.

Detuschmann and others have made *anatomical examinations* of lamellar cataracts. These examinations showed that within the opaque layer numerous small gaps, or vacuoles, filled with liquid are present between the lens fibers; while within the nucleus itself only one or two such vacuoles are to be found. In addition, larger fissures occur, surrounding the nucleus like a shell (Fig. 230).

Lamellar cataract does not always present a uniformly gray disk, but frequently exhibits a *complicated structure.* Densely opaque dots or graceful figures are often noticed in the anterior or posterior opaque layers, or markedly opaque sectors are contrasted with the less opaque matter in their vicinity. Again, while the riders correspond to partial opacities of a neighboring layer, the latter may also be opaque through its entire extent so as to surround the inner opaque layer like a cloak, while at the same time separated from it by a thin transparent stratum. Thus there originate double or even triple lamellar cataracts.

People suffering from lamellar cataract are often *myopic;* for on account of the indistinctness of the retinal images, they are compelled to bring objects closer to the eye in order to make up in the size of the retinal images what they lack in clearness. From this apparent myopia a real one usually develops subsequently, because through the habit of constantly looking at objects near by, the posterior wall of the eyeball becomes stretched and the axis of the eyeball elongated.

478. (5) *Cataracta Corticalis Anterior et Posterior.*—In this there is found in the anterior or posterior cortical layer of the lens a stellate or rosette-shaped figure, the center of which corresponds to the pole of the lens, while its rays are directed radially toward the periphery (Fig. 232). Anterior cortical cataract is much rarer than the posterior variety; sometimes both are

Fig. 231.—Posterior Polar Cataract. Magnified 2 × 1.

Fig. 232.—Posterior Cortical Cataract. Magnified 2 × 1.

found together. The two forms of cataract occur generally in those eyes which suffer from affections of the deep parts, like chorioiditis, retinitis pigmentosa, fluidity of the vitreous, etc.; the disturbance in the nutrition of the lens, thus produced, induces the formation of an opacity in it. The interference with vision is usually considerable, since it is caused not only by the opacity of the lens, but also by the involvement of the fundus. Anterior and posterior cortical cataracts remain stationary for many years and then at length pass into total opacity of the lens. They accordingly constitute a transition between the stationary and the progressive forms of cataract. When they have induced total cataract, they afford a bad prognosis for the operation on account of their being complicated with a lesion of the fundus.

Posterior cortical and *posterior polar* cataracts are frequently confounded. Accordingly, I present a drawing of the two kinds of cataract side by side to show the difference. Polar cataract is a round dot (Fig. 231), and represents a tissue that is deposited upon the posterior surface of the posterior capsule of the lens. Anatomically, therefore,

this form of cataract does not belong to the cataracts at all, since the opaque tissue lies outside of the lens system. It hence shows no indication whatever of a radial structure, which is the very feature that is characteristic of a posterior cortical cataract. The latter, in fact, is not only much larger than a posterior polar cataract, but also has always, in conformity with the radiating arrangement of the lens fibers at its posterior pole, the shape of a star or a rosette, with coarse or fine radial striation (Fig. 232).

Cataracta corticalis anterior and posterior are sometimes observed after *injury of the lens*, and that both when the lens capsule is opened and also in simple contusion of the lens without opening of the capsule. The stellate opacity in the cortex develops in the days immediately succeeding the injury, and may either rapidly pass over into complete opacity of the lens, or may remain stationary, or may even disappear again. The rapid development of these opacities, as well as the fact that they can disappear, indicates that they consist in some change of the lens fibers, which can abate again—perhaps being a swelling of the fibers without disintegration.

(b) PROGRESSIVE CATARACT

479. Course.—Progressive cataracts begin as partial opacities, which steadily extend until at length they occupy the entire lens. This is true, with the qualification that the portion of the lens already sclerosed—that is, the nucleus—ordinarily remains exempt from opacity. Hence, opacity of the lens in all its parts occurs only in young persons whose lens as yet has no hard nucleus; in older people the nucleus generally remains transparent. The time required for a lenticular opacity to involve all the parts that are capable of becoming opaque at all, varies very greatly. There are cases in which a transparent lens becomes completely opaque within a few hours, while other cataracts require many years before they can become total.

We distinguish in the course of a progressive cataract four stages, which are best marked in the most frequent form, senile cataract. To this form, therefore the following description mainly applies:

480. (1) *Cataracta Incipiens.*—Opacities occur in the lens, between which are found spots that are still transparent. The shape of the opacity is most frequently that of sectors (so-called spokes), the base of which look toward the margin, and their apex toward the poles of the lens.

One of the precursors of cataract is the *unequal refractivity* that ophthalmoscopic examination frequently shows in the separate portions of the lens. In that case, when the pupil is illuminated with the ophthalmoscope, spots are seen here and there which first shine more vividly red than the rest, then upon a slight turn of the mirror appear as dark shadows in the red of the pupil. These are comparable to the striæ that are found in defective glass [or in a block of ice].

Often the nucleus of the lens is distinguished by its specially great reflective power, so that, without being really opaque, it becomes visible upon ophthalmoscopic examination as a dark-red globular body, in the center of the brightly illuminated pupil. In conjunction with the marked difference in refractivity between the cortex and nucleus that exist in these cases, a fairly high degree of shortsightedness (*lenticular my-*

opia) develops.[4] Such eyes show even to the unaided view a specially strong senile reflex; the pupil looks so gray that one might think himself justified in assuming the existence of a commencing cataract. The diagnosis of cataract, however, should be made only when the ophthalmoscope actually shows circumscribed opacities in the lens.

Such opacities appear most frequently under the following forms:

1. Opaque *sectors* (radii) which look grayish-white by reflected light, black by transmitted light, and whose apices converge toward the pole of the lens (Fig. 233). They correspond to the natural grouping of the lens fibers into sectors. Sometimes they are broad and triangular, sometimes narrow, and are occasionally represented by nothing but slender radiating lines. That form of cataract in which the lens is traversed by numerous, very slender radiating lines is found chiefly in myopic eyes. The clouding of the sectors begins in their periphery, where they are visible with the ophthalmoscope long before their apices project into the region of the pupil and impair vision. As a rule, they appear first in the lower portions of the lens.

2. A *diffuse, smoke-like cloudiness* occupies the central portion of the lens. It belongs to those layers which directly inclose the nucleus. This sort of opacity disturbs the sight much earlier and to a much greater extent than do the opaque radii, because, in the first place, it is found in the pupillary area from the start; and, secondly, because it is diffuse and does not leave any places that are quite transparent.

Fig. 233.

Fig. 234.

Fig. 233.—Incipient Cataract under the form of opaque sectors, which look black when seen by transmitted light with the ophthalmoscope.

Fig. 234.—Incipient Cataract under the form of an irregular disk, which is more markedly opaque at its edges, and which is situated in the posterior layers of the cortex.

3. A disk-shaped *opacity* which is situated *in the posterior layers of the cortex*, but which, in contradistinction to the typical posterior cortical cataract (Fig. 232), presents an irregular and ill-defined contour and a cobweb-like structure (Fig. 234). This sort of opacity, too, causes from the outset very great disturbance of the sight.

4. We find, as an extremely frequent occurrence, in the eyes of old people, a ring-like opacity which lies near the equator of the lens, and which, on account of its resemblance to the arcus senilis of the cornea, was named by Ammon the *arcus senilis* (sive *gerontoxon*) *lentis*. This opacity is composed of two parallel opaque rings, one of which lies in front of the equator of the lens, and the other behind it. It does not impair vision, since it lies wholly behind the iris, and, moreover, it shows little tendency to spread.

Frequently, in commencing senile cataract, several or even all of the above-mentioned forms of opacity are met with.

5. While ordinarily the *opacity of the anterior capsule* is not associated with a cataract until the latter becomes hypermature, it exceptionally happens that such an opacity precedes the lenticular opacity proper and so represents the beginning of the senile cataract. A densely white dot forms in the center of the pupil. This gradually

[4] [To the alteration in the nucleus, producing this increased refractivity of the lens as a whole, the name nuclear sclerosis is applied by some, although the term properly denotes any progressive hardening of the nucleus. The full amount of lenticular myopia produced by nuclear sclerosis (in this restricted sense) amounts to some 3 or 4 D.—D.]

enlarges by outgrowth taking place at its edge. Then the lens fibers directly adjacent become opaque, and finally the entire lens opacifies. The whole process runs an extremely slow course and takes a number of years before it produces complete opacity of the lens.

6. In young people, cataract often begins in the form of *irregular*, *macular* or *cloud-like* opacities.

481. (2) *Cataracta Intumescens.*—In proportion as the lens becomes more opaque, it contains more and more water, and hence swells up. This increase in volume of the lens is recognized by the increasing shallowness of the anterior chamber. As long as the opacity has not reached to the anterior capsule, the iris throws a shadow upon the lens. To see this, a light is held near the eye and to one side of it. Then a black shadow is seen at that side of the pupillary margin that is turned toward the light (Fig. 235). This arises from the fact that the opaque layer of the lens upon which the iris casts its shadow lies some distance behind the iris. This opaque layer acts

Fig. 235.

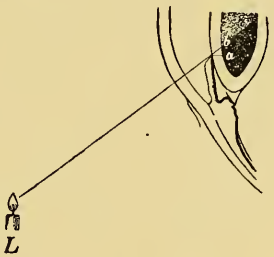


Fig. 236.

Fig. 235.—Shadow of the Iris Seen from in Front. The crescentic shadow appears at that side of the pupillary margin that is turned toward the source of light, *L*.

Fig. 236.—Shadow of the Iris upon the Lens in Schematic Cross Section. The inner layers of the lens are supposed to be opaque, the peripheral ones transparent. The source of light, *L*, throws upon the surface of the opacity a shadow from the iris, whose central border is at *b*. An observer looking from a point straight in front of the eye, sees a portion of this shadow, of the width *a b*, running alongside of the pupillary margin of the iris.

like a screen which receives the shadow of the iris. An observer, looking at the eye from in front, sees then that portion of the shadow which does not lie behind the iris itself (*a b*, Fig. 236). This portion of the shadow becomes narrower the nearer the opacity approaches the iris, and finally disappears altogether when the opacity reaches the anterior capsule. The distended lens has a bluish-white color and a marked silky luster of the surface, and shows very clearly the stellate markings of the lens.

During the stage of intumescence the opacity of the lens becomes total. As soon as this has taken place the lens begins gradually to lose water, so that it returns once more to its former normal volume. The lens then enters upon the stage of maturity (3) or is *ripe*.

The intumescence of the maturing cataract is caused by the swelling of the cortex. It is, therefore, more distinctly pronounced the softer the cataract is, since then there is much cortex; on the other hand, it is altogether wanting in the dark, hard cataracts which consist of scarcely anything but nucleus. For the same reason, too, the

ordinary phenomena of hypermaturity do not occur in the latter; instead of undergoing further disintegration, the horny lens remains unchanged, or, at most, thickening of the capsule is added.

How long a time does it take for a cataract to become ripe? The *progress* of a cataract is sometimes rapid, sometimes slow, the latter especially in senile cataract which not infrequently remains in an almost unchanged condition for years. Hence, if we find in an elderly patient the first stages of a cataract which as yet produces no interference with vision worth mentioning, the indication is, in the interest of the patient, not to frighten him by communicating his condition to him, as he perhaps may enjoy sufficiently good vision for several years to come. For our own security we may communicate the discovery to some near relative of the patient's. Sometimes, again, the lenticular opacity progresses by fits and starts—a cataract which has remained unchanged for quite a long time becoming almost completely mature within a few months, or even weeks.

For all these reasons it is for the most part impossible to answer with precision the patient's question as to when the cataract will become ripe. The following diagnostic points may serve for an approximate determination: The lenticular opacity develops the more rapidly the younger the person is. Light-colored cataracts become matured more rapidly than dark ones, and those with broad radii more rapidly than those with slender radii. A cataracta nigra can never become matured in the ordinary sense of the word, since it is not a cataract proper but a sclerosis of the entire lens which may be said to have been converted in toto into a nucleus, and hence always preserves a certain degree of transparency. For the laity a criterion of the ripeness of a cataract —i. e., of its readiness for operation—is the fact that the eye is no longer in a condition to count fingers. This does not hold good for dark cataracts (see page 545) which generally do not become so opaque that the patients cannot perceive the larger-sized objects. Nevertheless, these can be operated upon with good results, since the lens has been transformed into a hard, horny, translucent mass which can readily be shelled out cleanly from its capsule.

The rapidity of ripening is also influenced by the etiology of the cataract. Certain cataracts, such as diabetic, traumatic, and glaucomatous cataracts, furthermore complicated cataracts, particularly those resulting from detachment of the retina, are distinguished by their rapid rate of progress. The time required for ripening can be most readily determined, at least in the case of senile cataracts, when the other eye already contains a ripe cataract and the time that this has taken to develop is known, since presumably the rate of advance of the cataract is the same in both eyes.

[Patients with progressive cataract ought to be re-examined from time to time to determine their vision and *refraction*. For often in them the sight deteriorates not so much from progressive opacification itself as from the changes in lenticular density and corresponding changes in refraction that the opacification produces (cf. pages 528 and 537). In such a case often a change of glasses will not only enable the patient to see better, but will give him the comfort of knowing that his cataract is not advancing as fast as he supposed.—D.]

Opacities of the lens, especially when examined with a magnifying glass and focal illumination conjointly, often present quite clearly the appearance of being formed of minute drops. Sometimes these are of bluish color (cataracta cærulea). Such opacities are most frequently found in cataracts of young persons and in complicated cataracts.

482. (3) *Cataracta Matura.* (Stage of Maturity).—The anterior chamber is once more of normal depth, and the iris no longer casts a shadow—a proof that the opacity of the lens has become total. The lens has lost its

bluish-white, iridescent look, and has a dull-gray or brownish color; the radial markings of the stellate figure of the lens are still for the most part recognizable. A mature cataract has the property of separating readily from its connection with the capsule. This is partly because the disintegration of the lens fibers has proceeded right up to the capsule, partly because the lens, formerly enlarged, has diminished again in volume, and thus the connection between the surface of the lens and the capsule is loosened. The lens then lies in its capsule like a ripe fruit in its rind (Arlt); and thus it has become ripe for operation, since it is of great importance that the cataract should be capable of removal from its capsule without many portions of the lens remaining behind. These would form a new opacity in the pupil—a secondary cataract—and thus render the result of the operation doubtful.

483. (4) *Cataracta Hypermatura.*—The further metamorphosis of a mature cataract consists in the complete disintegration of the opaque lenticular mass. This becomes converted into a pultaceous substance, which no longer shows any trace of the original structure of the lens, its formation out of sectors, etc. Hence, in a hypermature cataract we either see no marking at all, or nothing but irregular spots—no radii nor sectors. The consistence possessed by a hypermature cataract depends upon whether the gradual loss of water, which began after the intumescence of the cataract and reduced the latter to its normal volume, keeps on or not. If the loss of water continues, the pultaceous mass which is produced by the disintegration of the lens fibers becomes more and more inspissated. It dries up along with the nucleus of the lens into a flat, cake-like mass; the anterior chamber consequently becomes deeper and deeper. This is the ordinary form of hypermature senile cataract.

Fig. 237.—Morgagnian Cataract. Natural size. The pupil is dilated with atropine; the dark nucleus in this case is particularly big.

If the loss of water ceases after the lens becomes entirely opaque, the lenticular mass grows more fluid in proportion as it keeps on breaking up into smaller and smaller parts. If this process goes on in a young person, in whom there is no hard nucleus in the lens, the latter becomes liquefied through and through, so that the lens consists of a milky fluid (*cataracta fluida* sive *lactea*). If this metamorphosis affects an old lens, the nucleus, which has failed to become opaque, also escapes disintegration, and sinks under the form of a heavy compact mass to the bottom of the liquefied cortex. The cataract has then a homogeneous white appearance, corresponding to the milky cortex, and in its lower portion presents a brownish shading, which is bounded above by a semicircular line, and which represents the upper half of the dark nucleus. As the latter alters its position with the movements of the head, the brownish shadow can also be seen to change its place. This form of cataract is known as Morgagnian cataract (*cataracta Morgagni;* Fig. 237).

A liquefied lens, however, does not remain permanently unaltered, but inspissation of the fluid occurs later on through the gradual loss of water, the disintegrated lens masses being at the same time in part resorbed. In this way the lens constantly diminishes in volume until, in cases in which no nucleus has been present, it is transformed into a thin, transparent membrane (*cataracta membranacea*). In children, in whom resorption is carried especially far, the opaque lenticular masses disappear altogether in places. The two layers of the lens capsule, which has remained transparent, come into apposition, and thus there are formed perfectly transparent spots in the opaque lens, recognizable by reflected light as black gaps in the white pupil. The child begins to see again, a sort of spontaneous cure of the cataract having occurred.

When a hypermature cataract has lasted a long time, changes set in which lead to complications: (*a*) Cholesterin, or lime salts, are deposited in the lens mass. The former is recognizable with the naked eye under the form of glistening points in the opaque lens. Calcification of the lens (*cataracta calcarea* sive *gypsea*) takes place chiefly in complicated cataracts. It is characterized by a peculiar coloration, varying from chalk-white to yellow. (*b*) The anterior capsule becomes thickened by proliferation of the capsule cells, so that out of a simple lenticular cataract there is formed a *cataracta capsulo-lenticularis*. The capsular opacity presents itself under the form of a densely white, irregular spot upon the gray or brownish surface of the lens, usually occupying the central part of the anterior capsule, over an area about corresponding to the pupil (Fig. 238). (*c*) The lens becomes tremulous. The shrinking of the hypermature cataract affects not only its thickness, but also its equatorial diameter. In proportion as the latter diminishes in size, the zonula of Zinn is stretched, and thereupon undergoes a corresponding atrophy of its fibers. Consequently, the attachment of the lens becomes imperfect, so that the lens shakes with the movements of the eye (*cataracta tremula*). Spontaneous luxation of the lens may even take place through partial or total rupture of the zonula. In consequence of these changes an operation for hypermature cataracts is often more difficult, and gives rather less favorable results than the operation in the stage of maturity.

FIG. 238.—CAPSULAR CATARACT IN A CASE OF HYPERMATURE CATARACT.

The center of the dilated pupil is occupied by an irregular, brilliantly white capsular thickening, upon which can be recognized the fine wrinklings of the capsule. In the vicinity of the large capsular opacity are found thickenings of the capsule, which have just developed, and which form white dots contrasting strongly with the subjacent brownish and radially striate, opaque lens.

By a combination of thickening of the capsule with various degrees of consistence of the lens special varieties of cataract are produced. A liquefied lens in a thickened sac-like capsule is called *cystic cataract* (*cataracta cystica*). By the term *cataracta arida siliquata* is understood a shriveled cataract within a thickened capsule; deriving its name (dry, silique-shaped or pod-like cataract) from its similarity to a dried pod (silique).

By the shriveling of the cataract in the stage of hypermaturity the anterior chamber becomes deeper, until finally the iris, instead of projecting forward in the shape of a cone, lies in a plane. If the diminution in the size of the cataract keeps on, the iris is not drawn backward so as to form a funnel, except when it is joined to the lens capsule by posterior synechiæ. Otherwise the iris remains stretched in a plane, and the shriveling lens becomes farther and farther removed from the iris, so that the latter, deprived of its support, becomes tremulous. A dark interspace is then seen between the iris and the lens, and the iris again throws a shadow upon the latter. This shadow must not, of course be confounded with that which is found in immature cataract. Nor should the black rim of pigment on the margin of the pupil, seen in every case of cataract, be regarded as the shadow cast upon the iris. It is easily distinguished from a shadow by its appearance, and also by the fact that it is visible not only at the side toward the light, but all round the iris.

In the stage of hypermaturity in which the opaque layers become thinner through resorption, the sight often increases a little, so that, for example, the fingers can be again distinguished. Really serviceable vision sometimes comes on in young people, when the resorption goes on so far that spots are formed which are perfectly transparent. In senile cataract, in which a hard nucleus is present, it is extremely rare for a spontaneous restoration of sight to occur, although it may take place in the following ways: (*a*) By resorption, in exceptional cases, not only of the cortex but also of the nucleus to such an extent that nothing but slight opacities remain. (*b*) By the formation of a Morgagnian cataract, and the subsequent transformation of the fluid portion of the cataract into a clear, transparent liquid. Then the upper part of the pupil is transparent and black, while the brown nucleus is seen lying in its lower part. These cases are not so very rare, only they were not formerly correctly diagnosticated. Afterward the transparent liquid, and even the nucleus itself, may be resorbed, so that only a thin membrane is left. (*c*) By spontaneous dislocation of the lens, so that the pupil again becomes partly or entirely black.

[The striæ and other opacities of an incipient cataract sometimes, though rarely, undergo spontaneous absorption.—D.]

Operations upon hypermature cataracts give rather less favorable results than those performed at the time of maturity. The chief danger attending operation in the stage of hypermaturity is prolapse of the vitreous during the operation on account of the defective condition of the zonula. Moreover, the thickened and opaque anterior capsule requires special management. We must try to remove it with the capsule forceps before delivering the lens. If the capsule does not tear when we grasp it with the forceps, it is generally possible to extract the lens in the unopened capsule, and we then get a particularly clear pupil. But if we have not been able to remove the opaque capsule altogether, an after-cataract forms, which does not disappear by subsequent resorption as cloudy lens masses do; and in this case a secondary operation (discission) is required.

484. Diagnosis of Stage.—As the diagnosis of the stage of a cataract is what determines the question of the performance of an operation, and hence is of great practical importance, the distinguishing signs of the separate stages will be summed up in the following words:

1. Cataracta incipiens. Anterior chamber of normal depth; transparent spots still to be found in the lens between isolated opacities.

2. Cataracta intumescens. Anterior chamber shallower; iris usually casting a shadow; lens bluish-white and having a silky luster; markings of the stellate figure of the lens very distinct.

3. Cataracta matura. Chamber of normal depth; no shadow cast by the iris; markings of the stellate figure of the lens still recognizable.

4. Cataracta hypermatura. Anterior chamber of abnormal depth; no shadow cast by the iris; surface of the lens appearing quite homogeneous (in the case of liquefaction), or showing irregular dots and spots in place of the radial markings of the lens-star.

485. Hard and Soft Cataracts.—According to their *consistence*, total cataracts are distinguished into hard and soft (*cataracta dura* et *mollis*). This has reference to the nucleus of the cataract. By soft cataract, therefore, we understand one having no distinct hard nucleus (Fig. 173), while those cataracts are known as hard which inclose a hard nucleus, although the cortex is soft (Fig. 172). The nucleus, in fact, does not usually become subject to cataractous changes, and therefore retains its natural consistence. The distinction between hard and soft cataract is made chiefly from a practical point of view. For the extraction of cataract from an eye a section must be made, the dimensions of which depend mainly upon the size of the nucleus. The wound must be large enough for the nucleus to pass easily through it, as otherwise the nucleus either cannot be removed from the eye at all, or, if it is squeezed forcibly through the wound, it crushes the lips of the latter. The soft cortex is stripped off from the nucleus, as the latter passes through the wound, and can subsequently be readily removed from the eye by stroking; it is unnecessary, therefore, to pay any regard to it in making the section. Hence, in soft cataract a small wound (simple linear extraction) suffices, while in hard cataracts the wound must be the longer the bigger the nucleus is.

For these reasons it is of importance before undertaking an operation to diagnosticate whether the cataract contains a hard nucleus, and if so, about how large it is. For this purpose we must take into account the age of the patient and the appearance of the cataract. The age is to be considered, inasmuch as the development of the nucleus is in direct proportion to it in healthy, and hence also in cataractous, lenses. Cataracts of children and young people have no nucleus; in older persons the nucleus is in the main the larger, the more advanced the age is. Nevertheless, it is not to be forgotten that very considerable individual variations occur with regard to the size of the nucleus. A careful inspection of the cataract, however, enables us to see the nucleus directly. It can be recognized upon lateral illumination as a dark reflex emanating from the depths of the lens. From this we can estimate its size, and from its color estimate its consistence also. The darker—reddish or brownish—the nucleus is, the harder (and usually, too, the larger) it is.

It may happen that the sclerosis of the lens has advanced so far that it has been entirely, or all except a small residue, transformed into a nucleus. It is then converted into a hard, dark-brown, transparent mass. The pupil

looks black, and it is only on careful inspection—especially with the aid of lateral illumination—that we recognize that it is of a peculiar dark brown. This condition is known under the name of *cataracta nigra*. Properly speaking, it is not a cataract in the true sense of the term, but a far-advanced senile alteration of the lens—a total sclerosis of it. Such lenses are always large and hard, and require a large section for their removal.

To what has been said in regard to the nucleus of the lens and its behavior in cases of cataract, exceptions occur. There are cases of cataract in children, in which the lens, instead of being soft, has quite a hard nucleus or even has a wax-like consistence throughout. On the other hand, cases of senile cataract have been observed without nucleus. In exceptional cases, the nucleus and not the cortex may be the first part to become opaque in the lenses of adults (*cataracta nuclearis*).

C. Etiology of Cataract

486. (1) *Cataracta Congenita.*—The cause of this is either a disturbance of development or an intra-uterine inflammation of the eye. Both the stationary partial cataracts (particularly anterior and posterior polar cataract) and the progressive forms of cataract may be congenital. Congenital cataracts are usually bilateral and often inherited. Heredity, however, makes its influence felt in non-congenital cataracts also, and, in fact, even in senile cataract; there are families many of whose members become blind from senile cataract, and that, too, for the most part at an uncommonly early age.

It is only in exceptional cases that *congenital cataracts* are discovered immediately after birth, the rule being that they are not made out until the child is some weeks or months old; for newborn children have very narrow pupils, and moreover, because they sleep so much, keep their eyes shut most of the time, so that no notice is taken of the fact that their pupils are not black. Then, too, as such young children do not fix their eyes steadily upon objects, the fact that they do not see is not obvious. Partial congenital cataracts, if they do not cause any notable impairment of sight, are often not noticed until the patient is of quite a mature age, or perhaps are never discovered at all. Many congenital cataracts are complicated, as can be seen from the changes found at the same time in the iris, expecially posterior synechiæ. They are hence the result of a fetal inflammation of the uvea. The formation of the cataract must in many cases be dated pretty far back in intra-uterine life, since children sometimes come into the world with cataracts that have already become shriveled. Here, therefore, the entire process of ripening and of shriveling has been evolved in utero.

487. (2) *Cataracta senilis* is by far the most frequent form of cataract. Indeed, it occurs very frequently in old people, but not so regularly as to be regarded as a physiological attribute of age—as the turning gray of the hair is, for example—but rather as a pathological process. It usually does not make its appearance until after the fiftieth year of life, but is exceptionally observed in the years between forty and fifty. The fact of a cataract occurring in an elderly person does not of itself justify the diagnosis of senile cataract. An old man may get a cataract in consequence of traumatism, etc. Hence, to establish this diagnosis, it must be demonstrated that neither in

the eye nor in the body in general are diseases which might account for the development of the cataract, so that the latter can be attributed only to the effect of age. Senile cataract always affects both eyes, but rarely at the same time, so that generally one eye is in advance of the other in respect to the development of its cataract.

Endeavors have been made, hitherto in vain, to discover some general disturbance of nutrition as the *cause* of senile cataract. Some ascribe it to purely local causes. In the process of transformation of the inner layers of the lens into nucleus (sclerosis) these layers diminish somewhat in volume. Under normal conditions this process of shrinking is conducted so slowly and gradually that the cortical layers are able to adapt themselves to the diminished volume of the nucleus. But, if the shrinking goes on with exceptional speed or irregularity, there may be produced undue traction and subsequent separation of those layers of the lens which lie between nucleus and cortex. In this situation fine fissures are formed in which fluid accumulates; afterward the adjacent lens fibers themselves become opaque, and thus afford the initial impulse which leads to the opacity of the entire lens (Becker).

Others suppose that senile cataract develops from altered composition of the aqueous, as a result of which the epithelium of the anterior capsule is injured. The alteration of the aqueous might be the result of local (senile) changes in the ciliary processes which secrete the aqueous (Peters) or the result of disturbances of the general metabolism, e. g., such as occur in chronic nephritis (Michel) or from accumulation of toxic decomposition-products (cytotoxins) in the body (Römer). Since senile cataract has such a different aspect in different cases, both at its outset and also when the opacity of the lens is complete, it is probable that the conditions which at present we call senile cataract are not all due to the same cause. Finally some have charged sunlight with being the cause of cataract, because they noticed that the first sectors of opacity develop in the lower portions of the lens which are most exposed to the sun's rays as they come from above. That a part of the rays that traverse the lens—and particularly the rays of short wave length—are absorbed by the lens is proved by the marked fluorescence that the latter exhibits. [The view that *light*, especially light rich in ultra-violet rays, is the cause of cataract receives some substantiation from the experiments of Burge, who found that lenses thus exposed became opaque, provided they were at the same time charged with sugar or certain salts of calcium, magnesium, or silicon. (See page 23.) It further appears that cataractous lenses do contain an unusual amount of magnesium and calcium salts and in the case of Indian cataracts an unusual amount of silicon. If these findings are substantiated for cataract in general, it would seem that the development of cataract would represent the combined effect of light acting in a prolonged or excessive manner on the eye and of some disorder of metabolism acting to charge the lens with sugar or mineral salts. This sort of combined action may explain *glass-blowers'* cataract. This is a very slowly developing posterior cortical opacity, occurring according to Legge in 30 per cent of glass workers over 40. It affects those especially who have for over twenty years been exposed to the light and occurs especially in the left eye, which is habitually turned toward the light. According to Verhoeff and Bell, heat, not light, is the chief factor in causing it.—D.]

488. (3) *Cataracts due to General Diseases.*—The most frequent of these is diabetic cataract. This mainly develops when the amount of sugar contained in the urine is high, and usually matures rapidly. It is always bilateral.

It was formerly believed that the cause of *diabetic cataract* was to be looked for in the abstraction of water; for if a fresh, transparent lens with uninjured capsule is laid in a solution of sugar (or even a solution of salt), the lens becomes clouded, owing to the fact that the solution absorbs water from the lens with avidity. If the opaque lens is then put back in plain water again, it again becomes clear. The same experiment can also be performed upon living animals. The blood in the vessels of a frog is replaced by a solution of sugar or salt, whereupon the lenses become opaque. Then, if the frog is put back again into the water, the lenses clear up once more. Upon the basis of these experiments it was assumed that in diabetes the fluids of the eye, and particularly the aqueous, on account of the amount of sugar they contain, act by withdrawing water from the lens, so that the latter becomes opaque. This view appeared to be confirmed by the fact that diabetic cataract occurs only when the amount of sugar in the urine is great. More recent analyses of the aqueous in diabetics have, however, shown that the amount of sugar contained in it is very small, much smaller than the amount required to produce opacity of the lens in the experiments cited. Hence, although it is not to be denied that the altered composition of the fluids of the eye is accountable for diabetic cataract, nevertheless, the action of this factor is not to be conceived of simply as an abstraction of water, but as being due to more complicated disturbances in the nutrition of the lens, the nature of which is not yet exactly known.

The retinal pigment of the iris in diabetes is often found to present a marked degree of œdematous swelling, even when the iris during life did not exhibit any symptoms of inflammation (Kamocki). Since this state of things has been observed only in diabetes, it must be attributed to this disease, and is perhaps explainable as due to the action of the altered aqueous upon the retinal pigment of the iris. It is probably in a similar way that the altered nutrient fluids which enter the lens lead to its opacification. [For the possibility that the cataract may be due to the effect of light acting upon a lens containing sugar, see page 546.—D.]

A form of cataract which really depends upon the abstraction of water is probably that which sometimes makes its appearance in the last stages of *cholera*.

The prognosis of diabetic cataract, as far as the operation upon it is concerned, is less favorable than in senile cataracts, because in diabetes wounds show less resistance to infection, and, moreover, diabetes predisposes to iritis. Hence, if we have to operate upon the diabetic cataract, we wait until by suitable treatment the amount of sugar in the urine has been reduced to the lowest possible point. It is said that in diabetic cataracts which have not advanced too far a partial disappearance of the opacities has sometimes been observed after a successful treatment of the diabetes (by the Carlsbad water cure). Such cataracts, therefore, would be the only ones—and they only in exceptional cases—that can be improved by medicinal treatment. [See, however, page 551, note.]

Not every cataract that is seen in a diabetic patient is a diabetic cataract. Diabetes being of common occurrence in advanced life, it often happens that opacities of the lens are found in patients affected with it. Such opacities must be regarded as senile cataract, if they exhibit the ordinary appearance of the latter and the slow development characteristic of it.

An interesting example of opacity of the lens, due to an altered composition of the nutrient fluids, is *naphthalinic cataract*. This is produced when naphthalin is administered to rabbits. Retinitis develops first with opacities of the vitreous, and subsequently a cataract forms (Bouchard). So also in man retinitis and cataract have been seen to occur after the application of naphthol ointments (when used for skin disease). Other forms of cataract following the ingestion of poisons are those that appear in *ergotism*, *rhaphania*, and *pellagra*. In these diseases convulsions are pro-

duced by the action of the poison. The same is true of tetany, which most observers are inclined to attribute to auto-intoxication, and in which, too, cataract may develop. [This may occur in the tetany that follows removal of the thyroid or parathyroid glands —D.] In adults the opacity due to tetany takes the form of a soft, total cataract, in children perhaps that of a lamellar cataract (see page 533). The formation of cataract also occurs sometimes in connection with epileptic, eclamptic and perhaps even hysterical convulsions.

489. (4) *Cataracta Traumatica.*—All injuries which make an opening in the lens capsule result in opacity of the lens. When the lens fibers come into contact with the aqueous, they become permeated by the latter, swell up, and break up into layers through a process of cleavage (page 12). When the traumatism affects the posterior capsule, the vitreous acts in the same way as does the aqueous.

The opening of the capsule usually occurs through direct injury inflicted by means of a punctured or incised wound, through the penetration of a foreign body, and also designedly through an operation (discission). Contusions of the eyeball also, which do not perforate its tunics, may cause opacity of the lens. In many of these cases it is likely that rupture of the capsule, probably in the region of the equator of the lens, is caused by the contusion. But it is also a fact that lenticular opacity is caused by simple concussion without opening of the capsule.

The development of cataract after injury of the capsule proceeds in the following way: As early as a few hours after the injury the lens is found to be clouded in the vicinity of the capsular wound. Soon swollen lens fibers protrude through the capsular wound, so as to project under the form of gray flocculi into the anterior chamber. Later on these break off and fall to the bottom of the chamber. Sometimes the entire chamber is found to be filled with the swelling and disintegrating fragments of the lens. While these prolapsed masses of lens substance are becoming gradually smaller through resorption and finally disappear, new flocculi keep protruding through the capsular wound. At the same time the opacity spreads farther and farther in the lens itself, so that usually within a few days the lens is opaque throughout. In favorable cases the lens may disappear completely by gradual absorption, so that the pupil becomes clear and black, and thus a spontaneous cure of the cataract takes place. In most cases, however, resorption comes to a stop earlier from reclosure of the capsular wound. Then opaque portions of the lens still remain in the shrunken capsular sac and form a shriveled cataract, which requires an operation for the restoration of sight.

The course of traumatic cataract is unfavorable when either inflammation or increase of tension is associated with the process. *Inflammation* is, for the most part, like the cataract itself, to be regarded as the direct consequence of the traumatism, by which the membranes of the eye (particularly the uvea) either suffer a severe mechanical injury or undergo infection.

Then the clouding of the lens and the inflammation (irido-cyclitis) go on at the same time. The inflammation leads to the adhesion of the opaque lens to the neighboring parts, especially the iris and ciliary body (*cataracta accreta*), and by this adhesion the operation for the cataract is rendered difficult. In the severest cases the inflammation is so violent that alone it is sufficient to cause destruction of the eye, either as a result of panophthalmitis or through plastic irido-cyclitis terminating in atrophy of the eyeball.

Slight inflammations of the iris may also, it is likely, occur secondarily as a result of the swelling of the traumatic cataract, owing to which the iris is subjected to pressure or to traction.

Increase of tension may also be caused by a swelling traumatic cataract. These cases are not very dangerous if they come under the observation of a physician, since the increase of tension can be done away with by timely interference (by paracentesis of the cornea, by removal of the lens, or by iridectomy). But if such a case is not given the proper treatment, the sight is usually destroyed through excavation of the optic nerve.

In traumatic cataract it is the rule that the opacity of the lens becomes total, spreading rapidly from the site of the wound in the capsule to the rest of the lens. Exceptionally, however, cases are observed in which the opacity of the lens remains partial or indeed actually disappears again. For this to occur, the capsule wound must be very small, so that it closes quickly and the aqueous no longer has access to the lens fibers. Most favorably situated in this regard are those capsular wounds that lie behind the iris, by the adhesion of which to the wound the latter is very soon closed up. In such cases it may happen that a circumscribed opacity remains confined to the site of injury, or, if a foreign body has penetrated the lens, is found only along the track of the wound. By resorption of the opaque portions the opacity itself may even in part clear up again. Sometimes, too, as a consequence of injury, stellate anterior or posterior cortical cataracts develop which may likewise remain stationary or may even retrogress (see page 537). Vossius has described another kind of transient traumatic opacity of the lens. It occurs after contusion of the eye and consists of a faint gray ring directly beneath the anterior capsule of the lens, which in respect to position and size corresponds to the border of the pupil. The opacity passes off after one or more weeks. The way in which it is probably produced is that the aqueous as it rebounds presses the border of the pupil against the capsule of the lens.

[A special form of traumatic cataract is that produced by *lightning* stroke or electric shock (page 25).]

490. (5) *Cataracta Complicata.*—By this term we mean cataracts occurring as the result of other diseases of the eyeball. What occurs in this case is either an alteration in the character of the nutritive material which the lens receives from the tissues surrounding it, or a diffusion through the lens capsule of deleterious substances produced by inflammation—the main result of such diffusion being that the capsular epithelium is injured. The affections of the eyeball most frequently leading to the formation of cataract are: (*a*) violent inflammations in the anterior sections of the eye, such as extensive suppuration of the cornea (particularly that produced by ulcus

serpens) and irido-cyclitis; (*b*) sluggish inflammations in the posterior sections of the eye, such as chorioiditis (particularly irido-chorioiditis chronica), myopia of high degree, retinitis pigmentosa, detachment of the retina; (*c*) glaucoma in the stage of glaucoma absolutum (*cataracta glaucomatosa*).

The diagnosis that a cataract is complicated may be made in those cases where there is a disease of the anterior section of the eye, simply by the external examination of the eye. Morbid changes can be made out in the cornea or iris, and also adhesions between these organs and the cataract. But if the pathological changes which have led to the production of opacity in the lens appertain to the deeper portions of the eye, they may not be visible from the outside. Even in such cases, however, the cataract often, by its peculiar appearance, shows that it is complicated. Thus in chorioiditis and retinitis pigmentosa, stellate anterior and posterior cortical cataracts are found (see page 536); and if the cataract is total, it is often distinguished by being liquefied or calcified, by the thickening of the capsule, by the presence of a yellow or green discoloration, by tremulousness of the lens, etc. If the cataract presents nothing exteriorly that points to its being complicated, the only way in which the diagnosis can be made is by examining the perception of light, a thing which should be done in every case. Such examination will often demonstrate the perception of light to be deficient or altogether wanting in complicated cataract.

It is of practical importance to recognize the fact that a cataract is complicated, because by this fact the prognosis and treatment are influenced. The prognosis is less favorable than in uncomplicated cataracts, both because the operation is more difficult to perform, and because the result, as far as sight is concerned, is less successful. Moreover, complicated cataracts frequently require that special methods of operation be used. In fact, many complicated cataracts cannot be operated upon at all.

If the two eyes are of a different color (e. g., one has a brown, the other a blue iris [*heterochromia iridis*]) it may happen that a cataract develops in one of them and in that case it always develops in the *lighter eye* of the two. In the absence of other causes, this must be regarded as something connected with the lack of pigmentation in the lighter eye; it being assumed that a disturbance of nutrition is at the bottom of both morbid conditions. It is true that nothing more definite than this is known regarding such a disturbance of nutrition; but that it is present is clear from the fact that in the lighter-colored eyes we [nearly] always find the evidence of a chronic cyclitis in the form of very minute deposits. [See page 368.] Accordingly, the cataract in these eyes is to be put under the category of complicated cataracts. [The difference in color in these cases may be very slight, one iris being simply a somewhat lighter blue or gray than the other, and yet the lighter eye showing pronounced degenerative changes while the other remains normal. The cyclitis in these cases is often extremely chronic, extending over a number of years, and may begin even several years before the difference in color of the irides is noticed.—D.]

D. Treatment of Cataract.

491. No kind of *medicinal treatment* is effectual against cataract[5]. An improvement of the sight may be obtained by means of atropine in those cases in which the opacity occupies principally the pupillary area of the lens. In that case, after dilatation of the pupil, the peripheral transparent portion of the lens can be employed for vision.

The popular remedies and quack remedies which are alleged to have been of assistance in cataracts are mostly such as contain belladonna, and act favorably upon the sight in the way just mentioned. The improvement thus obtained, however, is transient, disappearing as soon as the peripheral layers are implicated in the opacity by the progress of the cataract. A cure of cataract can be secured only by operative means. The indispensable prerequisite for this is that the light-perceiving parts (retina and optic nerve) should be healthy, a matter which is determined by careful testing of the light perception (see § 754).

The methods of *operation* at our command are mainly discission and extraction. Discission is chiefly adapted for the cataracts of young people which do not yet contain a solid nucleus [§ 864]. It can be performed in every stage of the growth of the cataract, and hence, too, in partial opacities of the lens. Moreover, discission is indicated in membranous cataracts, not to effect their absorption, which would be impossible, but to tear a hole in them (dilaceration) [or cut one (§ 867)]. The indications for extraction will be given at the same time with the description of the methods for performing it (§§ 869, 870 et seq.). Extraction gives its best results when the cataract is ripe. Hence we should put off the operation until this occurs; provided always that the other eye also retains sufficiently good sight in the meantime. But if the other eye also becomes so clouded that the patient is incapacitated from work, the cataract may be extracted even before it is fully ripe. Healing then takes place with a good result as in ripe cataract, except that layers of transparent cortex are more apt to remain adherent to the capsule during the operation. These afterwards become opaque, so that a secondary cataract is formed which requires a secondary operation (discission). Different operative methods have been proposed to accelerate the process of ripening, among which Förster's (iridectomy with massage of the lens, § 874) is the most employed.

Cataracts which are *congenital* or which develop in childhood should be operated upon as early as possible. Children can be subjected to the operation of discission with good results at the age of a few weeks. If the cataract is not operated upon, the development of the retina is arrested and amblyopia ex anopsia is produced (§ 568). Consequently, the good result

[5] [It seems possible that absorption of incipient cataract, which sometimes occurs spontaneously may be effected in some patients, especially those of a plethoric habit and of rheumatic tendencies by thorough-going reduction of diet and measures for the prevention of intestinal auto-intoxication (L. Connor and others).—D.]

of a cataract operation that is performed at a later date is comparatively small so far as vision is concerned.

In *traumatic* cataract our first thought should be to combat the inflammation which usually follows the injury. Iced compresses are of the most service against this impending inflammation, and also against the great swelling of the wounded lens. Removal of the opaque lens should not be performed at once except when, owing to its own great swelling, it is itself the cause of inflammation or of increase of tension. Otherwise, it is better to put off the operation until later, lest the inflammatory symptoms be aggravated or brought on anew by it. If we wait a long time, often a great part of the cataract is absorbed spontaneously, so that instead of extraction a simpler operation (discission) can be performed. So, too, in complicated cataract associated with inflammatory symptoms we had better await the decline of the inflammation before operating, unless imperative indications compel us to an earlier performance of the operation.

Sometimes we operate for cataract even in an eye which has no longer perception of light and hence cannot regain any sight—doing this merely for cosmetic reasons in order to give the pupil its natural black hue again.

492. The Aphakic Eye.—An eye which has been operated upon for cataract is, in consequence of the loss of the lens (aphakia), hypermetropic to a marked degree, and has, moreover, lost its power of accommodation, so that distinct vision can be obtained only by suitable convex glasses. [See § 876.]

Shall we operate upon an eye affected with a mature cataract if the other still sees well? In the case when a beginning development of a cataract is already present in the second eye this question is evidently to be answered in the affirmative. To know whether we shall also operate when the second eye is perfectly healthy and gives us no reason to anticipate the formation of a cataract, we must ask what gain the patient would derive from a unilateral cataract operation. How is vision performed with two eyes, one of which has its lens and the other has not? In that case a very great difference exists between the refraction of the two eyes—that is, a marked degree of anisometropia. Binocular single vision is still possible, but the images are never sharp and distinct in both eyes at the same time. The plan that naturally suggests itself of correcting the aphakic eye by a corresponding convex glass, and thus making both eyes alike, proves to be impracticable[6] (see § 794). But, though the patient is not able to use the operated eye for distinct vision at the same time with the other, he yet derives from it the advantage of an enlargement of the field of vision. In one-eyed people the field of vision for the single eye is limited toward one side by the nose, while the man who sees with two eyes has a binocular field of vision which stretches very far toward both sides. An eye which has been operated upon for cataract furthermore, even though it never wears a proper convex glass, nevertheless retains all its functional capacity, so that it can at once take the place of the other eye in case that should become unserviceable. It is, therefore, a reserve for the future. If we had left the cataract with the idea of not operating upon it until something had happened to

[6] [Not always. There have been cases in which the full correction has been successfully applied to the two eyes after cataract operation, and cases in which this can be done are probably more frequent than is supposed (see Note to § 794).—D.]

the other eye, we might perhaps be obliged to operate upon a very hypermature cataract under unfavorable conditions.

For *erythropsia* following cataract extraction, see page 22.

II. Changes of Position of the Lens

493. Anatomical Cause.—Changes of position of the lens always have their anatomical cause in changes of the zonula of Zinn. This in the normal eye is tightly stretched, and holds the lens so firmly that the latter remains perfectly immovable even with the most violent motions of the head. Hence, any tremor of the lens, and still more any displacement of it from its natural position, presuppose a relaxation of the firmness of this attachment. Such a relaxation can take place either from a simple elongation and loosening of the fibers of the zonula, or else from their rupture or complete destruction. Changes of this sort may affect either single portions or the entire circumference of the zonula.

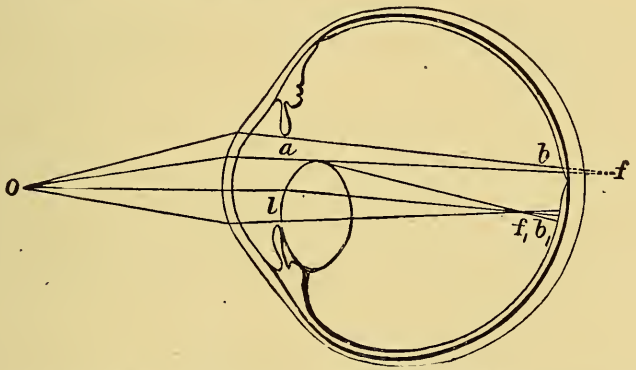


Fig. 239.—Subluxation of the Lens. Schematic.

The lens has sunk so far downward that its upper edge is visible in the pupil. In consequence of the relaxation of the zonula, the lens bulges strongly, and is in contact by its lower border with the ciliary processes; moreover, the lower half of the iris is pressed forward by it. Above, on the contrary, the anterior chamber, owing to recession of the iris, is abnormally deep. Of the beam of rays emitted by the luminous point *O*, a portion goes through the aphakic part, *a*, of the pupil; these rays, on account of the absence of the lens, are insufficiently refracted, so that they come to a focus behind the retina at *f*, and form upon the retina a diffusion circle, *b*. That portion of the beam passing through the section, *l*, of the pupil, which contains the lens, undergoes excessive refraction on account of the increased convexity of the lens, so that the rays intersect in front of the retina at f_1, and form upon the retina a diffusion circle, b_1. This latter gets to lie below the fovea centralis (and below the diffusion circle, *b*), because all rays passing through the lens undergo a deviation downward on account of the prismatic action of the latter. Thus two images of the point *O* are produced upon the retina. The monocular diplopia thus caused is not, therefore, the result of the two sorts of refraction present in the area of the pupil, but is the result of the prismatic action of the margin of the lens.

494. Objective Signs. Subluxation.—The objective symptoms of a change of position differ according as the lens is only displaced a little (subluxation), or has left its place in the fossa patellaris altogether (luxation).

(*a*) *Subluxation* may consist in the lens' being a little tilted, so that one edge of it looks somewhat forward, the opposite one somewhat backward. This is recognized from the unequal depth of the anterior chamber. Another sort of subluxation is produced by lateral displacement of the lens, so that it no longer lies in the center of the fossa patellaris. In this case, too, the anterior chamber is unequally deep. If, for example, the lens is

somewhat depressed, the anterior chamber would be found to be deeper in its upper half, shallower below (Fig. 239). Furthermore, when the pupil is dilated (and, if the displacement is marked, without this) we can see the edge of the lens. This, in the example above selected of depression of the lens, would run transversely across the pupil, forming an arch which is convex upward. That part of the pupil which is situated above it, and which has no lens (Fig. 239, *a*) would be a deep black, while the lower part (*l*) which contains the lens would be faintly gray. This arises from the fact that even the most transparent lens reflects some light. Really, therefore, the normal pupil is not perfectly black, but of a very dark gray—a fact of which one can convince himself in those cases in which, through displacement of the lens, one part of the pupil is aphakic, and therefore is of a pure black.

In both cases—that is, when the lens is tilted and when it is laterally displaced, conditions which are often combined—there occurs in movements of the eye tremor of the inadequately attached lens and with the lens of the iris also (iridodonesis).

A transparent, luxated lens looks differently, according as we regard it by reflected or transmitted light. By reflected light the lens appears faintly gray, and its edge has a golden luster, almost as if it were a self-luminous body. This is so because the rays of light that come from in front and enter the marginal portions of the lens undergo total reflection at the posterior surface of the latter; for at this spot they pass from a denser medium (the lens) into a rarer medium (the vitreous), and hence are refracted away from the normal of incidence; but as, in consequence of this, they fall very obliquely upon the posterior lenticular surface at the edge of the lens, they undergo total reflection. They accordingly do not continue their course into the interior of the eye, but return to the observer, who therefore sees the edge of the lens shine. By transmitted light—in examining with the ophthalmoscope—the edge of the lens for the same reason appears black, because the light that is reflected from the fundus, wherever it traverses the lens near its edge, is so greatly deflected by the strong prismatic action of the latter toward the opposite side of the lens, that it fails to reach the eye of the observer, in case he is stationed straight in front of the eye. Hence the border of the lens appears unillumined. But if the observer's eye is made to pass slowly toward the side of the lens opposite the unillumined edge, a point is finally reached where the rays pass that are transmitted through this edge; and then the latter appear of a shining red, while the rest of the lens appears unillumined (Dimmer).

In making an examination with the inverted image, we may often, in case of dislocation of the lens, see some portion of the fundus—e. g., the papilla—double, and for the same reason that the affected eye itself sees external objects double.

495. Total Luxation.—(*b*) Luxation of the lens consists in its leaving the fossa patellaris altogether, either by prolapsing into the anterior chamber or by receding into the vitreous.

A lens luxated into the *anterior chamber* is readily recognizable from its shape. This is more convex than usual because the lens is no longer kept flat by the tense zonula. It therefore assumes its maximum convexity, as it does upon the strongest effort of accommodation. If the lens is transpar-

ent, its edges appear like a curved line of golden luster, so that it looks as if a great drop of oil were lying in the anterior chamber. The anterior chamber is deeper, especially below, where the iris is pressed backward by the lens.

Luxation of the lens into the *vitreous* occurs more frequently than luxation into the anterior chamber. The anterior chamber then is deep because of the recession of the iris, which is tremulous. The pupil is of a pure black. The lens, itself, if opaque, may sometimes be recognized deep down even with the naked eye; in most cases, however, the ophthalmoscope is required in order to discover it. It is either attached to some spot of the fundus by means of exudates, or it floats about freely in the vitreous (*cataracta natans*).

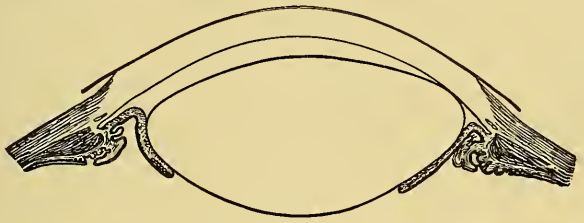


Fig. 240.—Luxation of the Lens into the Anterior Chamber. Magnified 5 × 1.

The lens lies in the anterior chamber and moreover is displaced toward the right side (in the drawing), so that here, on the one hand, it abuts against the posterior surface of the cornea, and, on the other hand, against the ciliary body, the iris being interposed between it and the latter. The pupillary portion of the iris everywhere is jammed against the posterior surface of the lens and thus the posterior is cut off from the anterior chamber. On the other side of the lens the iris is pushed forward and bent at an angle by the aqueous which has accumulated in the posterior chamber.

If the lens has prolapsed into the anterior chamber, it produces through irritation of the iris a spasm of the sphincter iridis. The pupil consequently contracts, so that the return of the lens into the posterior chamber is cut off. It may even happen that on account of this spasm the lens is held tight at the moment when it is endeavoring to make its way through the pupil into the anterior chamber. The lens is then jammed in the pupil, and consequently violent symptoms of irritation are at once set up. But there are also cases in which the lens can slip through the pupil so easily that it is found sometimes in front of, sometimes behind, the iris. Sometimes the patient is able to produce this change of place voluntarily. He can bring the lens into the anterior chamber by bending his head forward and shaking it, while to bring the lens back again behind the iris he has to lie upon his back. In this case, of course, we are always dealing with lenses of diminished diameter, which can pass through the pupil without difficulty. In some instances such movable lenses are still attached to the zonula, which is then greatly elongated. If in such a case we were obliged to extract the lens, we would first bring it into the anterior chamber by the appropriate manœuvre. Then if we cause the pupil to contract behind the lens by employing a miotic, we have, as it were, imprisoned the lens in the anterior chamber, and will be able under ordinary circumstances to remove it with ease. However, these cases in which the lens shows such a great capacity for making excursions belong to the rare exceptions. The rule is, that a lens luxated into the anterior chamber stays there, and, in consequence of the violent inflammation which it excites, becomes attached by exudates to the cornea and iris.

496. Subjective Symptoms.—Every dislocation of the lens entails a considerable *disturbance of vision.* If the lens still lies *within the pupillary area,* the eye becomes very myopic, because owing to the relaxation of the zonula the lens assumes its maximum convexity. Added to this is a considerable degree of astigmatism, arising from the fact that the lens, either from being tilted or being laterally displaced,[7] refracts the light with unequal strength in the different meridians (regular astigmatism); or the refractive power may even vary in different sections of the same meridian (irregular astigmatism). The astigmatism attains its maximum when the lens is so greatly displaced that its edge is visible in the pupil, the latter thus consisting of a portion which does, and one of which does not, contain the lens. In such a case, moreover, double vision—monocular diplopia—is present; for the marginal portions of the lens act like a prism, whose refracting edge corresponds to the equator of the lens. By reason of this the rays passing through the lens are deviated, so that two images (b and b_1, Fig. 239) of one object (O) are formed upon the retina. Neither of these is distinct. The image (b) produced by the aphakic portion of the pupil corresponds to that formed by a very hypermetropic eye, and would require a convex lens to make it distinct. The image (b_1) appertaining to that part of the pupil which contains the lens is that of a myopic eye, and could be made distinct by means of a concave lens. Furthermore, disturbance of vision may be produced by the development of opacity in the subluxated lens.

In luxation of the lens *into the vitreous* the eye acts like an aphakic one, and, if no further complications are present, sees well with the correcting convex glasses. Indeed, in the old method of cataract operation by depression, a luxation of the lens into the vitreous was produced designedly in order to increase the sight.

The disturbance of vision which develops in subluxation of the lens, so far as it consists of myopia and regular astigmatism, can be corrected by glasses, but the irregular astigmatism cannot be. If the dislocation of the lens is so great that a part of the pupil is aphakic, we have the choice of correcting either the aphakic portion of the pupil with a convex glass or the portion of the pupil that contains the lens with a concave glass. We recommend to the patient the form of correction which gives the better sight. Sometimes, for the sake of better correction, it is indicated to enlarge the aphakic portion of the pupil by an iridectomy, and so make the eye like one destitute of a lens.

497. Sequelæ.—Dislocations of the lens usually entail secondary consequences which may be extremely disastrous to the eye. Subluxations often in time change into complete luxations, the vibrating lens constantly pulling upon the zonula and gradually causing it to atrophy. While subluxated lenses often remain transparent for a long time, luxated lenses usually soon become opaque. Moreover, dislocated lenses are often opaque to start with, this being particularly the case in spontaneous luxations. The

[7] [Or from being unequally relaxed (in cases of partial rupture of the zonula).—D.]

worst complications are irido-cyclitis and increase of tension (secondary glaucoma). The most dangerous form of luxation of the lens is that into the anterior chamber. In this case the cornea becomes opaque wherever the lens is applied to its posterior surface, and the eye, for the most part, undergoes speedy destruction through irido-cyclitis or increase of tension. On the other hand, luxation of the lens into the vitreous is the form best tolerated, especially if the lens, as time goes on, becomes smaller through resorption. In fact, in the depression of cataract, one used to count upon this tolerance of the eye toward the lens when depressed into the vitreous.

498. Etiology.—With respect to etiology a distinction is made between congenital and acquired dislocations of the lens.

(*a*) *Congenital* dislocations consist in a lateral displacement (subluxation) of the lens, which is known as *ectopia lentis* (Fig. 241). The displacement is caused by the fact that the zonula is of unequal width in different directions. Most frequently the lens is found to be displaced upward, the fibers of the zonula being shortest above, longest below. For the most part, too, the volume of the lens is somewhat smaller. In after years the ectopia usually increases, and even passes over into a condition of total luxation. Ectopia of the lens is ordinarily present in both eyes and symmetrically in both. Very often it is of hereditary origin.

Fig. 241.—Ectopia Pupillæ et Lentis. Right Eye.

The pupil is pretty wide (5 mm.), not regularly circular and is displaced temporally. The broader nasal portion of the iris displays the contraction furrows. The lens is slightly cloudy, smaller than normal, and displaced in a direction opposite to that of the pupil, that is, nasally.

(*b*) The *acquired* dislocations of the lens develop either as the result of trauma or spontaneously. *Traumatic* dislocations are principally caused by contusion of the eyeball (for the mechanism of the luxation, see page 442). Every variety of subluxation and luxation may be thus produced, according as the zonula is simply torn into or is entirely torn through. If the tunics of the eye are ruptured, the lens may even be expelled entirely from the eye. Among traumatic luxations in the more extended sense of the word may be reckoned those which develop when perforation of a corneal ulcer takes place very rapidly; in this case if the perforation is only large enough, the lens may even be discharged through it externally. *Spontaneous* dislocations take their origin from a gradual softening and disintegration of the zonula. The lens then owing to its weight sinks gradually deeper and deeper, and at length undergoes complete prolapse into the vitreous. The atrophy of the zonula develops as a result of liquefaction of the vitreous, and hence occurs especially in myopia of high degree, in chorioiditis, and in detachment of the retina. Again, the shrinking of a hypermature cataract may cause stretching of the zonula with consequent atrophy of it, and thus give rise to spontaneous dislocation of the lens, so that the sight which has been abrogated by the cataract is restored without an operation. If for any

reason the zonula is already atrophic, the final impulse leading to total luxation is frequently afforded by a very insignificant traumatism—in fact, even by bending over, sneezing, etc.

Spontaneous dislocation of the lens not infrequently occurs in *ectasiæ* either of the eyeball as a whole or of its anterior segments—hence in hydrophthalmus, in staphylomata of the cornea, and in anterior staphylomata of the sclera. The luxation takes place because, as a result of the bulging out of the wall of the eyeball, the space between the edge of the lens and the ciliary body becomes enlarged, so that the zonula is stretched and finally atrophies. It may even happen that the lens has become adherent to a corneal cicatrix, so as to become more and more tilted as the cicatrix expands. So, too, the lens is sometimes drawn out of its place by exudates in the vitreous, which attach themselves to its posterior surface and afterward shrink. Lastly, the dislocation of the lens due to tumors (gliomata and sarcomata) pressing upon it (Fig. 192) may be also mentioned in this connection.

499. Treatment.—*Treatment* in those cases in which the dislocation of the lens entails no further injurious consequences besides the disturbance of vision, consists in the prescribing of suitable glasses. In those cases in which the symptoms of irido-cyclitis or of secondary glaucoma are caused by the displacement of the lens, extraction of the latter, if feasible, is indicated. Extraction is most readily performed in luxation of the lens into the anterior chamber; in this case, too, it is absolutely required, since otherwise the eye is lost. In subluxation, the removal of the lens is often difficult or even miscarries altogether, because prolapse of the vitreous occurs on account of the defective structure of the zonula. Discission of a subluxated lens may be tried, but it is not often successful, because the lens, being imperfectly attached, gives way before the discission needle. [For extraction of a lens floating in the vitreous see § 879.] In cases in which the removal of the lens is difficult or impossible, all we can do is to combat the inflammation or the increase in tension by means of an iridectomy. If an eye which is already blind is the seat of inflammation and pain due to luxation of the lens, enucleation is the best means of relieving the pain and averting the danger of sympathetic affection of the other eye.

Congenital and Other Anomalies of the Lens

500. [Congenital anomalies of the lens include congenital *cataract* (page 545) and *dislocation* (ectopia lentis—page 557). Furthermore, whether ectopic or not, the lens may be smaller as a whole (*microphakia*) or may show a partial defect (*coloboma lentis*), which may take the form of a notching, a flattening of the edge, or an elongation and compression of the lower end of the lens. Coloboma of the lens is often associated with coloboma of the chorioid or iris.—D.]

Lenticonus is a rare, usually congenital anomaly of the lens, which presents a conical prominence upon its anterior or posterior surface (lenticonus anterior vel posterior).

[The zonule of Zinn may present congenital (usually triangular) defects (*coloboma of the zonula*).—D.]

CHAPTER IX

DISEASES OF THE VITREOUS

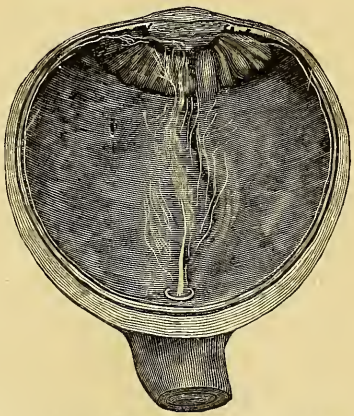
Anatomy

501. The vitreous (corpus vitreum) is a transparent, colorless, gelatinous mass which fills the posterior cavity of the eye. On its anterior aspect it has a depression (the fossa patellaris), in which rests the posterior surface of the lens. By its other aspects the vitreous is applied to the inner surface of the ciliary body and the retina.

The vitreous consists of a clear liquid substance inclosed in the meshes of an equally transparent reticulum—the framework of the vitreous. In the fetal eye the vitreous is traversed from behind forward by a canal, its central canal (canalis hyaloideus, canalis Cloqueti), which begins at the papilla of the optic nerve and extends to the posterior pole of the lens, and in which runs the hyaloid artery. In the fully developed eye this canal probably serves as a lymph channel (see page 16).

The vitreous contains cells, vitreous cells, which have a varying (rounded or branched) shape, and are found particularly in its outer layers. They are to be regarded as emigrated white blood corpuscles which have travelled into the vitreous (Schwalbe).

It is only in the fetus that the vitreous has vessels, which are found in its outer layers (see page 392). In the fully developed eye the vitreous is destitute of vessels, and hence is dependent for its metabolism upon the surrounding tissues, principally the uvea. Accordingly, affections of the inner membranes of the eye, such as retinitis and chorioiditis, always result in an implication of the vitreous.



[Fig. 242.—Persistent Hyaloid Artery (Lang and Collins). From Norris and Oliver.

Shrunken globe in which a tag of a persistent hyaloid artery was found adherent to the optic nerve on pathological examination.—D.]

Of the hyaloid artery normally the only thing that is left in the newborn infant is a short and slender cord, which, moreover, disappears during the first year of life. Exceptionally, however, larger remnants of the artery remain for life. A *persistent hyaloid artery* ordinarily appears under the form of a gray filament that stretches from the papilla out into the vitreous, and may even reach to the posterior pole of the lens. In typical cases it is possible to demonstrate the connection between the filament and the central vessels that emerge at the papilla, and in this demonstration is found the surest means of distinguishing between this remnant of fetal life and

pathological opacities of the vitreous, which may in other respects have a similar shape and position. Sometimes instead of a filament a wider tubular structure is observed, extending out from behind forward in the axis of the vitreous. This represents the *hyaloid canal* (also called *Cloquet's canal*), whose walls, owing to some abnormality in their structure, are visible with the ophthalmoscope. This congenital anomaly, as well as the persistent hyaloid artery, is frequently associated with opacities in the posterior portions of the lens (posterior polar and cortical cataract). In many animals —e. g., in the frog and in many snakes and fishes—the vessels of the vitreous persist during life.

Diseases of the Vitreous

502. (1) **Opacities.**—These are sometimes small and sharply circumscribed, sometimes of large size. The former, which make their appearance under the form of dots, flocculi, threads, or membranes, are what are called opacities of the vitreous in the narrow sense (*opacitates corporis vitrei*). The patient himself perceives them entoptically, seeing black specks of various shapes (muscæ volitantes) floating before his eyes (*myodesopsia*).[1] In addition, there is a diminution of the visual acuity, which is the more pronounced the more marked the opacities are.

The *cause* of opacities of the vitreous are generally exudates deposited there in the course of inflammations of the uvea or retina; but hæmorrhages, taking place from the vessels of these menbranes into the vitreous, either spontaneously or as the result of injuries, may also give rise to opacities of the latter.

The *prognosis* depends upon the size and the age of the opacities. Recent opacities of the vitreous may be absorbed, so that the vitreous becomes perfectly clear again. Old opacities, on the contrary, usually resist all treatment. As regards extravasations of blood, the smaller ones may be completely absorbed, but large-sized ones [nearly] always leave considerable and permanent opacities.

The *treatment*, which [generally] is successful only in recent cases, consists in the employment of remedies which accelerate absorption. Among these are potassium iodide or other remedies containing iodine [especially syrup of hydriodic acid (De Schweinitz)], mercury, diaphoretics, and mild purgatives. Of the last named, saline purgatives, especially the saline mineral waters—e. g., of the Kreuzbrunnen of Marienbad—are particularly employed. Repeated paracentesis of the anterior chamber may also be of service by stimulating the tissue metamorphosis of the eye; and subconjunctival injections of a 5- to 10-per-cent salt solution (one-half or a whole syringeful) [or of mercury oxycyanide] act in the same way. [In a recent case of vitreous hæmorrhage, rest in bed, kept up for several days is very important, and, if the hæmorrhage is due to injury, iced applications should be added. Dionin is also sometimes helpful. If the patient is anæmic iron should be given. Constipation should be relieved, the diet regulated, and

[1] From μυῖα, a fly, and ὄψις, vision; hence properly written myiodesopsia.

any remote source of infection sought for and removed. In arteriosclerosis the blood pressure should be reduced when this can be done safely. The iodides and other absorbent remedies mentioned above should be kept up for a long time. If this is done, sometimes old and even massive hæmorrhages may be absorbed.—D.]

[In persistent bleeding the subcutaneous injection of a 2-per-cent solution of gelatin or of defibrinated human blood may be tried (Leber).—D.]

Large-sized exudates which sometimes fill the vitreous originate in severe inflammations of the ciliary body, retina, and chorioid (see page 415, et seq.). They may be seen, if the media are otherwise clear enough, by lateral illumination under the form of gray or yellowish masses situated behind the lens. The plastic exudates become organized, shrink up, and thus lead to atrophy of the eyeball, while the purulent exudates for the most part are transformed into a panophthalmitis, i. e., are discharged externally after perforating the sclera, and terminate in phthisis bulbi.

The embryonic vitreous contains a great abundance of cells, and is hence opaque. The cells afterward disappear, but opaque remnants of them remain in the vitreous, and may be perceived entoptically as *muscæ volitantes*. These physiological opacities of the vitreous appear under the form of transparent filaments or of strings of pearls or of small flocculi, which move not only with the eye, but also spontaneously. We see this very readily if we look suddenly upward and then hold the eye still, when the opacities sink slowly down. They are thus distinguished from the entoptic images which are produced by opacities in the lens, as these always remain in the same place in the field of view. Physiological muscæ volitantes are not at all obvious, so that most men are not aware of their existence in their eyes. To perceive them we look at a uniformly illuminated surface—for instance, the sky—through a stenopæic aperture (a minute hole made by sticking a needle through a piece of black paper). They are usually better perceived by myopic eyes. As soon as such muscæ volitantes become so distinct as to continually force themselves upon the attention and to become troublesome to the patient, they excite the suspicion of their being pathological opacities of the vitreous. To discover them the ophthalmoscope is used.

When the opacities are faint, it is necessary to use a plane mirror and place behind it a strong lens in order to enable us to get as near as possible to the eye we are examining. Generally, too, artificial dilatation of the pupil is required. [Opacities of the vitreous are often best seen by the direct method, convex glasses of different strength being successively used so as to bring into view the opacities at different depths. From the strength of the glass that makes an opacity distinct, an approximate idea can be formed of the actual distance of the opacity in front of the retina.—D.] Seen with the ophthalmoscope, opacities of the vitreous appear like dark dots or filaments or membranes floating about in the vitreous. Very minute opacities afford the picture of an extremely fine stippling of the vitreous ("vitreous dust"). If the opacities are still more minute, they can no longer be perceived as discrete points; nothing but a uniform obscuration of the fundus is observed (diffuse opacity of the vitreous). The more numerous the opacities are the more hazy the fundus appears, the pupil at the same time looking redder than usual (as any bright background appears red behind a cloudy medium—e. g., the rising sun on a cloudy morning). With very dense opacities, nothing is got with the ophthalmoscope but a feeble red reflection from the pupil, or the latter may even be perfectly dark.

In *synchysis scintillans* particles are seen that look like golden spangles floating about in the vitreous, and which fall like a shower of gold to the bottom of the eye when the eye is held still. These are formed of crystals whose surfaces being smooth reflect the light strongly. They usually consist of cholesterin, sometimes also of tyrosin, margarin, and phosphates. These crystals are sometimes found in eyes that are otherwise healthy (especially in elderly people), without causing any essential disturbance of vision.

[Pathological] opacities are the *remains of exudates.* The more minute opacities consist of masses of cells or pigment granules or filaments. The larger exudates, so far as they do not undergo resorption, become organized into membranes, cords, or even pretty large masses of connective tissue. In this way a new formation of blood-vessels may even take place, which run from the retinal vessels into the vitreous, and can be made out there by means of the ophthalmoscope. Exudates in the vitreous do not originate in the latter itself, but from an inflammation of the membranes (uvea and retina) surrounding it. The disturbance of vision which is set up by a recent cyclitis, chorioiditis, or retinitis, is in large part attributable to the opacity of the vitreous, which is present at the same time. Primary inflammation (*hyalitis*) of the vitreous, which not only is devoid of vessels, but also contains scarcely any cellular elements, cannot be assumed to exist.

Opacities also form as the result of *hæmorrhages* into the vitreous. These occur after injuries and also spontaneously in chorioiditis, retinitis, and myopia of high degree, and, furthermore, are not infrequent in old people with atheromatous vessels. Sometimes, too, in eyes which are otherwise healthy hæmorrhages into the vitreous are observed, which appear spontaneously, recur repeatedly, and permeate the vitreous so thoroughly that even quantitative perception of light is abrogated. This affection, which is often associated with demonstrable retinal hæmorrhages, is observed chiefly in young men, sometimes in conjunction with frequent attacks of epistaxis. In some cases tuberculous disease of the sheaths of the retinal vessels has been shown by dissection to be the cause of the trouble. [It is due often to tuberculosis and occasionally to oxaluria (Leber); and constipation (Eales) and even minor errors of diet (Ziegler) have been held responsible.—D.] If the hæmorrhages recur often, the vitreous never clears up perfectly, but masses of connective tissue ultimately form in it which may vascularize, and detachment of the retina may occur. The sight is thus seriously and permanently affected or even absolutely annihilated (cf. Retinitis Proliferans, page 582 and Fig. 256).

After extensive opacities of the vitreous there may occur a brownish or greenish discoloration of the iris, due to the diffusion of blood coloring-matter.

The *disturbance of vision* caused by opacities of the vitreous is determined by their total amount. Isolated flocculi in the vitreous may coexist with normal visual acuity. When the opacities are numerous, the statement is often made by the patients that their sight shows great variations within short periods of time. This fact is also noticed when tests of the vision are made. While at first the patient, when placed before the card with the test types, does not begin to see the large letters, he can sometimes distinguish even the small letters after he has had his gaze fixed upon them for a pretty long time. Then all at once he sees much worse again. This comes from the mobility of the opacities, which, when the gaze is kept steadily fixed for a long time, sink to the bottom of the vitreous, so that the central portion of the latter becomes clear; then any great movement of the eye stirs them up again.

503. (2) **Liquefaction of the Vitreous (Synchysis[2] Corporis Vitei).**—When observing opacities of the vitreous with the ophthalmoscope, we see

[2] From σύν, together, and χέω, I pour.

that most of them float about freely in the vitreous. It follows from this that the framework of the vitreous must have been destroyed, so that this body itself is converted into a perfectly liquid mass. In operations we often have an opportunity of directly convincing ourselves of the liquefaction of the vitreous, which we see flowing out under the form of a viscid, usually yellow-colored liquid. Liquefaction of the vitreous occurs as a simple senile change, but when of greater extent it is the result of disease of the adjacent membranes, which are concerned in maintaining the nutrition of the vitreous—that is, it occurs chiefly in disease of the ciliary body, also in retinitis, chorioiditis, myopia of high degree, ectatic eyes, etc.

The most important consequence of liquefaction of the vitreous consists in the gradual diminution in volume, which the altered vitreous may undergo and which manifests itself in a diminished tension of the eye. In such cases, detachment of the retina and afterwards even atrophy of the eyeball may supervene. Another consequence of the liquefaction of the vitreous is that the zonula becomes softened and atrophic. By this a tremulous condition of the lens, and later on even its spontaneous dislocation are produced.

504. (3) **Foreign Bodies in the Vitreous.**—These usually excite violent inflammation—irido-cyclitis or panophthalmitis—by which the eye is destroyed. In exceptional cases it happens that a foreign body is tolerated, so that it may be seen for years, either free or enveloped in an exudate, within the otherwise clear vitreous. Even in these cases, however, inflammation may still set in, even after a long time has elapsed, and destroy the eye. Foreign bodies which have but recently entered the vitreous we try to remove as soon as possible. The chief ones that afford a prospect of doing this successfully are chips of iron, since magnets may be employed for their removal (see § 881), while the removal of other foreign bodies is usually effected only by a happy accident. If violent inflammation has already set in, there is usually nothing left to do but to perform enucleation to avert a sympathetic affection.

Among foreign bodies in the broader sense may also be reckoned lenses luxated into the vitreous and also the cysticercus, both of which, like foreign bodies in the proper meaning of the word, give rise to severe inflammation. The cysticercus may be removed by a section made in the sclera. If this is not done in season, or not done successfully, the eye is gradually destroyed by irido-cyclitis, and eventually has to be enucleated on account of the constantly occurring inflammatory attacks.

The *entozoa* occurring in the vitreous are the filaria, the echinococcus, and the cysticercus cellulosæ. Of the two former but very few cases have so far been known. The *cysticercus* was formerly frequent, but now, thanks to better meat inspection, has also become a rarity. It is the scolex of the Tænia Solium. Before a patient can have a cysticercus, the eggs must first get into his stomach. This may take place from the fact

that the patient himself harbors in his intestine a tænia, joints of which find their way into his stomach. Here they are digested and the eggs contained in them set free. Most patients, however, who suffer from the presence of a cysticercus do not have a tænia themselves. Hence, the eggs of the tænia must get into the stomach from outside along with the food (most frequently with the drinking water). In the stomach embryos develop from the eggs, having hooklets, by means of which they penetrate the stomach walls and get into the blood-vessels. The blood current then carries them into different parts of the body, where they again leave the vessels, bore into the tissues, and there grow into cysticerci. In the eye the cysticercus most often gets into the vessels of the chorioid and when it leaves there passes at once beneath the retina, detaching it from the chorioid (see Fig. 261). When it has reached a certain size it perforates the retina and gets into the vitreous. But the cysticercus may also find its way into a vessel of the retina or the ciliary body, and from there may enter the vitreous directly, without a preceding detachment of the retina. In the vitreous the cysticercus becomes visible as a bluish-white bladder. If the head and neck are drawn in, they appear under the form of a brilliant white spot; but if they are protruded they can be recognized quite distinctly, and it is even possible to make out in the head the suctorial disks and the crown of hooklets. The animal makes spontaneous, often very active, movements. It is rare, however, for the cysticercus to be seen with perfect distinctness when in the vitreous. For opacities very soon form in the shape of membranes, which so envelop it that nothing can be discerned through them but a dense white mass. In such cases the diagnosis of a cysticercus is difficult, and can indeed be made with certainty only when upon long and attentive observation we make out the existence of spontaneous movements taking place in the white structure. Ultimately the eye undergoes destruction from irido-cyclitis.

505. (4) **Detachment of the Vitreous.**—This consists in the accumulation of liquid, either in front of it between the lens and vitreous, or behind it between the vitreous and retina. (In the region of the ciliary body the vitreous is so firmly adherent that it never becomes detached.) Such detachment is much more rare than was formerly supposed, when observers were misled by the presence of artefacts produced by the hardening of the specimens (Greef, Elschnig).

506. (5) **Congenital Anomalies.**—[The congenital anomalies of the vitreous include the persistent hyaloid artery (page 559) and coloboma of the vitreous. The latter is a notch in the lower part of the vitreous, sometimes extending all the way from the optic disk to the ciliary body. It is caused by the projection of vascular connective-tissue strands into the interior of the eye.—D.]

CHAPTER X

DISEASES OF THE RETINA

Anatomy and Physiology

507. The retina is a thin membrane which in the living eye is perfectly transparent and of a purplish-red color. This latter depends upon the visual purple contained in the rods (Boll). After death the retina becomes very rapidly opaque, and, as at the same time the visual purple is bleached out under the influence of light, the retina in the eye of a cadaver appears under the form of a very frail white membrane. In the living retina, too, pathological changes manifest themselves at once by a loss of transparency, just as is also the case with the other transparent tissues like the cornea, lens, and vitreous. Thanks to this property, we discover even minute alterations in these organs very early.

There are two points that are particularly prominent in the retina when in situ. One is a small white disk, which lies to the inner side of the posterior pole of the eye, and from which the vessels of the retina emanate; this is the point of entrance or head of the optic nerve, the *papilla nervi optici*. The second spot occupies precisely the posterior pole of the eye, and is distinguished by its faint yellow color. It is hence called the yellow spot, the *macula lutea*. In its center is found a small depression, the fovea of the retina, or *fovea centralis* (*f*, Fig. 144).

A striking anomaly, which by tyros is frequently considered pathological, is the *presence of medullated nerve fibers* in the fiber layer of the retina. The normal retina is perfectly transparent, because the optic-nerve fibers lose their medulla before traversing the lamina cribrosa, and hence when inside of the retina itself are transparent; but in exceptional cases after traversing the lamina cribrosa they regain their medulla for more or less of their extent. (In many animals—e. g., in rabbits—this is the rule.) Since the medullated fibers are opaque, there is found in these places a brilliant-white spot adjoining the edge of the papilla and splitting at its periphery into white fibers, so as to have a flame-like look. Such spots are found most often at the upper and lower borders of the papilla (Fig. 243), but may surround the papilla completely, in which case the latter, by contrast, appears of a strikingly dark red. In rare cases white spots, formed of medullated fibers, lie within the papilla itself or conversely far from it in the transparent retina. The retinal vessels are in places covered by the masses of white fibers. The vision of such eyes is often reduced, and Mariotte's blind spot enlarged. [Yet many cases have normal vision, nor does the presence of medullated nerves necessarily cause a corresponding enlargement of the blind spot (Leber). This anomaly, though often called congenital, is not so in fact. See page 41.—D.]

508. If we try to lift the retina from the subjacent chorioid by means of a forceps, we see that it is connected with its bed only at two places. One of these is the head of the optic nerve, the other is the anterior border

of the retina. The latter is formed by a zigzag line, and hence bears the name of *ora serrata* (*o o*, Fig. 144). The same line also represents the boundary between the chorioid and ciliary body, and extends farther forward on the nasal than on the temporal side. Except at the two spots just named, the retina everywhere simply lies upon the chorioid without being attached to it.

A *histological examination* of the retina shows that it arises from the optic nerve, the fibers of which spread out in all directions and form the innermost layer of the retina, the fiber layer (Figs. 24, 1)[244 B, *X*]. The most external layer, that of the rods and cones (Fig. 24, 8) [Fig. 244, *II*],

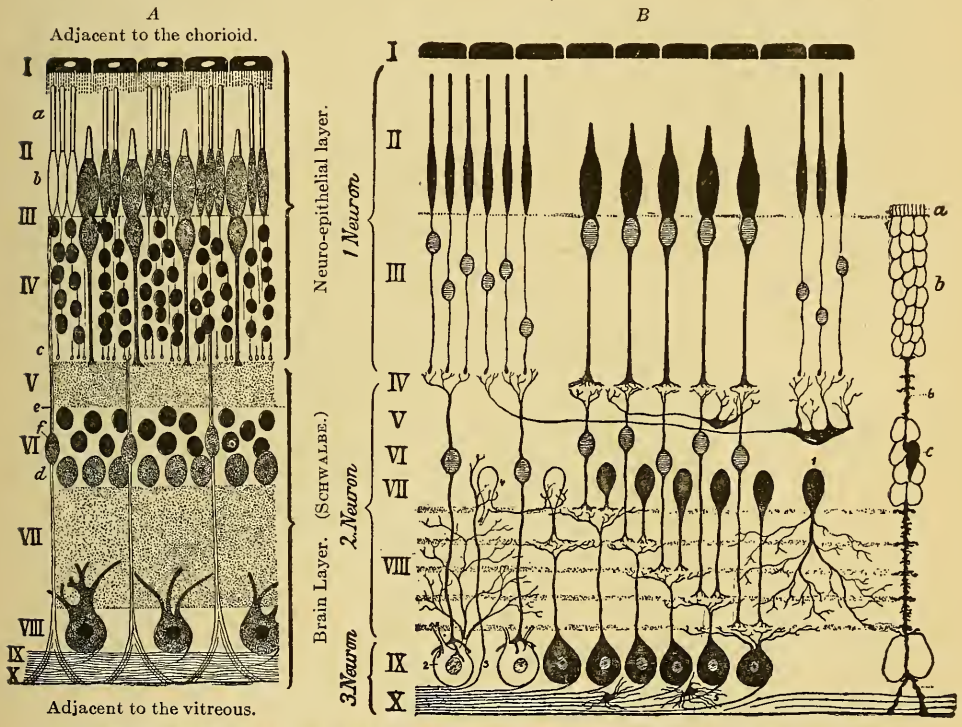
Fig. 243.—Medullated Nerve Fibers. (After Jäger.)

The papilla shows in its center a whitish coloration, representing the physiological excavation. The temporal border of the papilla is surrounded by an irregular chorioidal ring, while the upper and lower borders are concealed by the white fibrous masses that arise from them. These in places cover the retinal vessels, and especially the two arteries running outward and downward. At their peripheral borders the white masses break up into fibers.

is the light-perceiving stratum of the retina. For the rays of light to get to it, they must pass through all the other layers, since these are placed in front of it. Vision, therefore, can be perfect only when these layers are absolutely transparent, so that light undergoes regular refraction on its way to the most posterior (most external) stratum. All opacities of the retina consequently affect the sight, even though the terminal percipient elements may be perfectly healthy.

With regard to the minute structure of the retina, which is very complicated, reference must be made to the text-books of anatomy and histology. It need only be mentioned here that the retina is composed of two kinds of tissue, the nervous tissue and the supporting tissue. The function of the latter is to maintain and support the extremely delicate nervous tissue in the proper position, and also to insulate

the nervous elements from one another. The relative proportion of the two tissues changes in inflammation and particularly in atrophy of the retina, the nervous elements being destroyed while the supporting tissue becomes increased in quantity, so that the retina ultimately consists entirely of the latter.



[Fig. 244.—Scheme of the Structure of the Human Retina. (After Souter in Posey and Spiller.)

A. Horizontal section, hæmatoxylin stain. I. Pigment epithelial layer. II. Layer of rods and cones: *a*, external; *b*, internal elements. III. External limiting membrane. IV. External molecular layer: *c*, fiber layer. V. External granular layer. VI. Internal molecular layer: *d*, spongioblasts; *e*, supporting fibers of Müller; *f*, nuclei of the same. VII. Internal granular layer. VIII. Layer of ganglion cells. IX. Nerve-fiber layer. X. Internal limiting membrane.

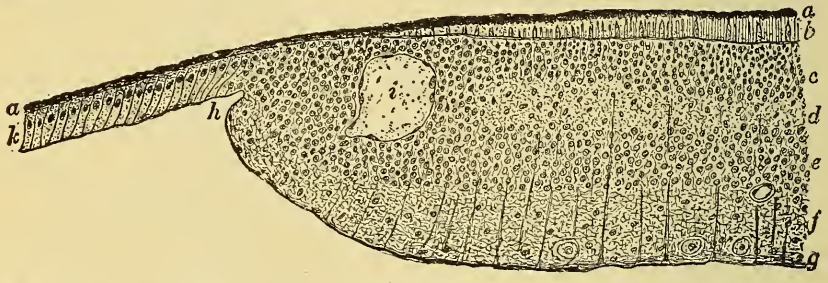
B. Demonstration after the method of Golgi. I. Pigment epithelial layer. II. Layer of rods and cones. III. Molecular and visual cells. IV. External plexiform layer. V. Layer of horizontal cells. VI. Layer of bipolar cells. VII. Layer of amacrine cells. VIII. Internal plexiform layer (fiber layer). IX. Layer of ganglion cells. X. Nerve-fiber layer: 1, diffuse amacrine cells; 2, diffuse ganglion cells; 3, centrifugal nerve fibers; 4, association-amacrine cells; 5 neuroglia cells; 6, supporting fibers of Müller. —D.]

509. Fovea Centralis.—The depression at the site of the fovea centralis arises from a thinning of the retina, the inner layers of the latter being here entirely absent. Furthermore, the retinal fovea is also distinguished by the fact that the most external layer here consists only of cones. The rods do not begin until at the border of the macula lutea, and as we pass toward the ora serrata, grow more and more numerous, while the number of cones diminishes in like proportion.

The fovea centralis is the part of the retina that has the most delicate

perception. And so when we wish to get a precise perception of an object, we so adjust our eye that the image shall fall upon the fovea; we ["sight" or] "fix" the object.

510. Pigment Epithelium.—The membrane here described—the retina, in the narrower sense of the word—develops from the inner layer of the secondary ophthalmic vesicle (page 390 and Fig. 161, *r*). From the outer layer of the vesicle (*p*, Fig. 161) is developed the *pigment epithelium*, which therefore must, on embryological grounds, be counted in with the retina (in the wider sense of the term). It lies upon the chorioid along the outer side of the retina, and, because it remains behind when the retina is removed from the chorioid, was formerly regarded as belonging to the latter. The connection between the retina and pigment epithelium consists in the fact that the cells of the latter send minute cilia-like processes in between the rods and cones; in these processes lie the minute crystals of the brown retinal pigment.

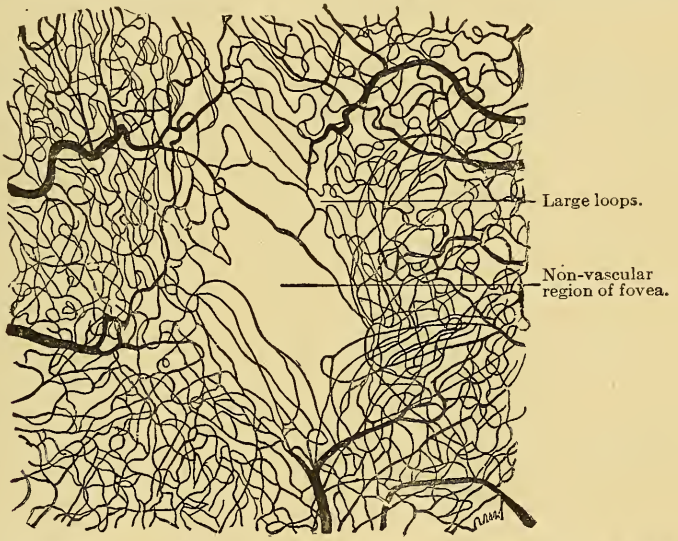


[Fig. 245.—Section of Human Retina at the Ora Serrata. (After Piersol in Norris and Oliver.) Shows the abrupt termination of the usual retinal layers and the continuation of the retinal sheet as the pars ciliaris.—*a*, pigment layer; *b*, rods and cones; *c*, outer nuclear layer; *d*, outer plexiform; *e*, inner nuclear; *f*, inner plexiform; *g*, ganglion cells; *h*, point of transition into inner stratum (*k*) of pars ciliaris; *i*, section of cyst. Magnified 165 diameters.—D.]

511. Continuation on Ciliary Body and Iris.—The cessation of the retina at the ora serrata is apparent only; the microscope shows that under a simpler form it extends still farther, even up to the edge of the pupil. It therefore lines the inner surface of the ciliary body and the posterior surface of the iris. The portion of the retina lying upon the ciliary body is called the *pars ciliaris retinæ* [Fig. 142, *pe* and *pc*]. Wherever this extends, the external layer of the retina, or pigment epithelium (Figs. 145 and 146, *P*), is more darkly pigmented, so that this division of the interior of the eye is characterized by a particularly dark color (*or*, Fig. 144). The inner layer of the retina—the continuation of the retina, in the narrower sense of the word—is here reduced to a single stratum of cylindrical cells (Figs. 145 and 146, *C*) [Fig. 245, *k*]. At the spot where the two layers of the retina pass over upon the iris, the difference between them becomes even less marked than before, since now the cells of the inner layer too are filled with pigment granules. Thus the two layers in conjunction form a uniformly pigmented stratum, which, as the *pars iridica retinæ* (retinal pigment layer of the iris),

covers the posterior surface of the iris up to the margin of the pupil, about which the two layers turn and then unite.

512. Retinal Vessels.—The retina has its own *system of blood-vessels*, which is almost entirely separate from the adjoining system of ciliary vessels. It is formed by an expansion of the arteria and vena centralis nervi optici, which break up into branches in the optic papilla. These branches subdivide in the retina as far as the ora serrata without anastomosing together (see Fig. 153, a, a_1, and b, b^1; Fig. 20 represents the branching of vessels in the retina as seen with the ophthalmoscope). At the papilla alone, minute communications exist between the retinal and the ciliary vessels (see page 382). The retinal arteries are hence to be regarded as terminal arteries



[Fig. 246.—Blood-vessels of the Yellow Spot Injected. After Böhm and Von Davidoff. (Norris and Oliver.)—D.]

(Cohnheim). Consequently, disturbances of circulation in the retina, arising from contraction or plugging of a vessel, cannot be compensated for by means of a collateral circulation.

Within the retina, the vessels lie only in the inner layers, so that the external layers of the retina are non-vascular, and are hence in part dependent for their nutrition upon the neighboring chorio-capillaris. This is especially true of the fovea centralis, the central part of which contains no vessels at all, while, on the other hand, the vascular network of the chorio-capillaris is here particularly dense.

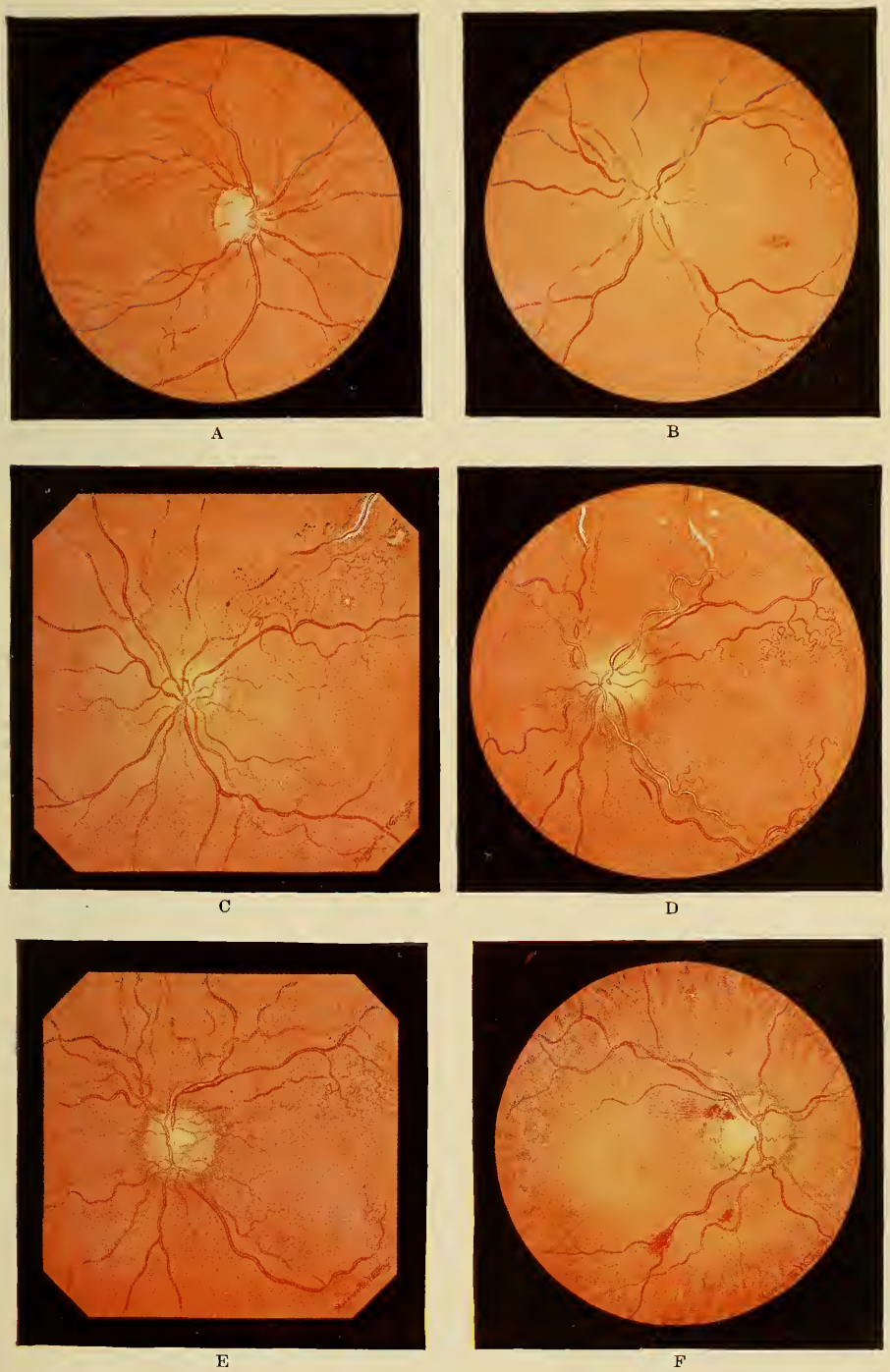
513. Function of the Retina.—The objects of the outer world throw their images upon the retina. It is the function of the latter to convert the rays of light, of which the images are composed, into nervous stimuli. What takes place, accordingly, is a transformation of one sort of motion—

the vibrations of the luminous ether—into another, namely, nervous excitation. This is without doubt simply another sort of motion of such a nature as to be transmissible within the nerve fibers to the brain, a property which is not possessed by the vibrations of the luminous ether. The place in which the conversion of luminous vibrations into nerve excitation occurs is the rods and cones. In what way this conversion takes place is not known, but we do know that a part of the vis viva, which the luminous vibrations represent, is used up in the production of chemical and physical changes, which we are able to follow. The chemical changes consist in the transformation of the visual purple, contained in the rods and which was discovered by Boll, into a colorless substance by the action of light (Kühne). It is very probable that besides the visual purple still other "visual substances"—i. e., substances which undergo chemical changes under the influence of light—exist in the retina, but that such changes are not accompanied by alterations of color, and have accordingly thus far escaped discovery. The physical changes partly consist in variations in the strength of the electric current that normally passes from the retina to the brain (Holmgren), partly are motile phenomena of a less subtle sort, which we perceive both in the cells of the pigment epithelium, and in the rods and cones. In the cells of the pigment epithelium, the pigment granules, when the eye is in the dark—that is, in a condition of rest—lie in the most posterior part of the cell close to the nucleus; but if light impinges upon the retina, these granules push their way forward into the cilia-like processes which extend between the rods and cones. In the rods and cones themselves, a process of contraction combined with shortening takes place under the influence of light.

I. Vascular Disturbances of the Retina

514. The retina is often the seat of disturbances of circulation, such as anæmia and hyperæmia, which latter frequently gives rise to hæmorrhages into it. The most extreme degrees of disturbance of circulation occur in consequence of embolism and thrombosis of the central vessels.

Beginners often fall into the mistake of considering a very vividly red eyeground as hyperæmic. The aggregate tint of the eyeground, however, depends on its pigment content, and the background of slightly pigmented eyes is always much redder than that of the darkly pigmented ones. Moreover, great dilatation of the retinal vessels is in itself no certain proof of hyperæmia of the retina, for it often occurs physiologically, especially in the young. Exceptionally, too, cases of very marked [congenital] tortuosity of the retinal vessels occur in healthy eyes. [Cf. Pseudoneuritis, page 98.] But in all these cases the condition of the retinal vessels in the two eyes is alike. Dilatation and tortuosity of the retinal vessels, therefore, can be regarded as certainly pathological only if unilateral or if confined to certain districts of the fundus, or if there is a faint cloudiness of the retina near the papilla, indicating permeation with serum, or, and more especially, if hæmorrhages can be made out.



[Fig. 247.—Changes in Arteriosclerosis. (After De Schweinitz.)

A, Normal fundus. B to F, successive changes occurring in arteriosclerosis, including pallid arteries (B), later assuming a silver-wire appearance (C); indented veins (B, C), afterward showing ampulliform enlargements (D, E); corkscrew capillaries (C, D); corkscrew arteries and veins (D, E); perivasculitis (C, D); sclerosis of vessels (F); œdema of disk (B, C, D, E), hæmorrhages (C, F).—D.]

515. Hyperæmia.—Hyperæmia of the retina may be either arterial or venous. The former accompanies all inflammations of the retina and the neighboring tissues, particularly the uvea, and is characterized by a pronounced distention and tortuosity of the arteries. Venous hyperæmia manifests itself by dilatation and great tortuosity of the veins, while the arteries are often thinner than usual. It is produced most often by plugging of the veins (see Thrombosis, page 575) or by their compression. The compression is mostly located in the papilla, as in glaucoma, where the increased intra-ocular tension squeezes the veins down into the excavated papilla, or in optic neuritis, in which the swelling of the papilla compresses the veins. In orbital tumors it is the trunk of the optic nerve that is compressed. Venous hyperæmia also occurs as a symptom of a general venous congestion, particularly in heart disease [arteriosclerosis (see below), juvenile recurrent hæmorrhages (page 562), and polycythæmia.—D.]

[Very rarely the retinal arteries and veins show sac-like dilatations (*aneurysms*) or varicosities. Miliary aneurysms of the retinal arteries occur, especially in old persons and most often in conjunction with extensive arteriosclerosis and atheroma (less often with heart disease and nephritis). There is a peculiar, usually very chronic and progressive condition, in which multiple aneurysms are associated with extensive infiltration of the retina (Leber).—D.]

[Conditions of *arteriosclerosis* and high vascular tension are often accompanied by visible changes in the retinal vessels. As summarized by De Schweinitz these consist of: (1) Corkscrew appearance of arterial twigs, while the arteries from which they spring may be normal. (2) Flattening of veins, where crossed by arteries. Later on, this flattening becomes an indentation and the veins beyond the point of compression become ampulliform. (3) A dull red congestion or actual œdema of the optic disk. (4) In the later stages the formation of white streaks along arteries and veins (perivasculitis) and hæmorrhages. These changes (see Fig. 247) are important because of their effect on the sight, because of their liability to give rise to retinal hæmorrhages, and particularly because they form a frequent and early sign of general arteriosclerosis, especially of the brain and kidney. Ocular arteriosclerosis may be associated with persistent asthenopia which is relieved only by general treatment (De Schweinitz).—D.]

[*Cyanosis* of the retina may occur in congenital heart disease (patent foramen of Botallo), in polycythæmia, and from the action of certain poisons (dinitrobenzol).—D.]

516. Retinal Hæmorrhage.—Extravasations of blood into the retina are frequent and occur in all sorts of sizes and shapes. They form dark-red patches contrasting with the brighter red of the fundus.

If in the fiber layer of the retina they have striate or flame-like shapes, because the exuded blood spreads along the fibers (Figs. 252 and 254). Hæmorrhages in the deeper layers of the retina or between it and the chorioid are of rounded or irregular shape (Fig. 253). In the region of the macula large disk-shaped extravasations sometimes occur, situated not in the retina, but between it and the vitreous (preretinal or subhyaloid hæmorrhage) (Fig. 248). The retinal tissue then is not injured by the hæmorrhage, so that after resorption of the blood vision becomes normal again. [Sometimes, however, preretinal hæmorrhages are not absorbed but form large membranes (retinitis proliferans), see page 582.]

The extravasations are most frequently located in the neighborhood of the larger vascular trunks. The causes of retinal hæmorrhages are—

1. General fragility of the vessel walls. This is found very often in old people with atheromatous vessels, particularly if they have a heart lesion, too. In such cases retinal hæmorrhages are often the precursors of cerebral apoplexy. [See page 571.]

2. Local disease of the retinal vessels or of the adjacent vessels of the chorioid. Under this head must be reckoned those hæmorrhages which occur so frequently in excessively myopic eyes in the region of the yellow spot. With the occurrence of such a hæmorrhage central vision is often permanently destroyed.

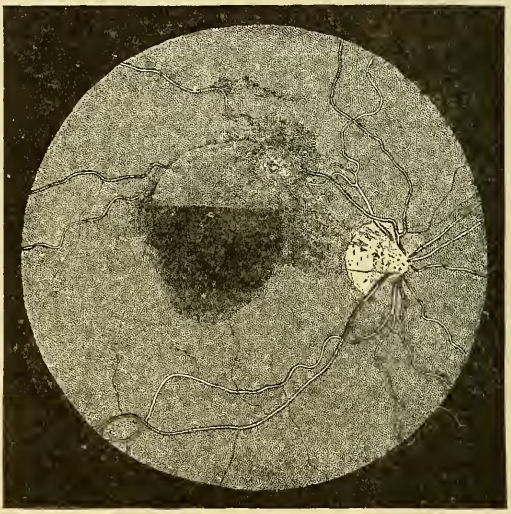


FIG. 248.—PRERETINAL HÆMORRHAGE.

This represents the right eye of an elderly woman. The eye, in consonance with its myopia, shows a broad white crescent at the temporal border of its papilla; and the latter also has a pretty large physiological excavation. The retinal arteries are very tortuous, the veins normal. The middle of the fundus is occupied by a large hæmorrhage, covering the region of the macula lutea, and extending upward as far as the superior temporal vessels, which are partially concealed by it. The lower part of the hæmorrhage is dark red, and is separated by a sharp horizontal line from the upper, pale-red portion. This division into two parts is caused by the settling of the blood corpuscles to the bottom of the still fluid blood. In the vicinity of the large hæmorrhage, especially at its upper and inner borders, lie numerous small spots of blood. These extend up to and upon the white crescent adjoining the optic nerve and up to the superior temporal vessels. From these vessels is derived the extravasated blood, which, after breaking through the limitans interna of the retina, gets between the latter and the vitreous, and sinks down to the region of the macula lutea, where even in the normal eye the connection between the retina and the vitreous is the least.

3. Over-distention of the blood-vessels by circulatory disturbances, such as active and passive hyperæmia of the retina and occlusion of the central artery and vein or their branches. In new-born infants retinal hæmorrhages are often found as a result of the disturbances of circulation occurring within a child's skull during birth. [They are present in from 21 to 32 per cent of the new-born, occurring especially in protracted labors, and in cases of instrumental delivery and constriction of the umbilical cord. They absorb very rapidly (Leber).—D.] Many cases of so-called congenital amblyopia (§ 567) may possibly be explained by them; i. e., the extravasated blood is absorbed and leaves no trace, but the lacerated retinal tissue never again becomes entirely normal. The retinal hæmorrhages which very often occur in glaucomatous eyes as a result of iridectomy are the result of sudden reduction of the intra-ocular tension, so that an unwonted amount of blood pours into the retinal vessels and distends them unduly. Moreover, the retinal hæmorrhages adduced under the next head may in part be caused by blocking of the smaller vessels—e. g., the hæmorrhages in sepsis by emboli composed of masses of fungi.

4. Altered composition of the blood affecting the vessel walls. Here belong retinal hæmorrhages in extreme anæmia, particularly pernicious anæmia, in leuchæmia, scurvy, purpura, sepsis, albuminuria, diabetes, oxaluria, intermittent fever, relapsing fever, [influenza], jaundice, phosphorus poisoning, extensive burns of the skin, etc. [Tuberculosis is often the cause of the recurrent retinal hæmorrhages which, together with recurrent hæmorrhages into the vitreous, sometimes occur in young persons (page 562). Often, however, these happen without assignable cause.—D.]

5. Rupture of the blood-vessels due to trauma.

Retinal hæmorrhages absorb very slowly, requiring for this weeks and months, during which they are often seen to take on a white color (Fig. 250). They ultimately disappear, leaving no trace of their presence or decolorized whitish, rarely pigmented spots in the fundus. [When large they may become organized into plaques or masses of connective tissue.] Whether a scotoma is left at the affected spot or not depends on the degree to which the hæmorrhage has lacerated the retinal tissue.

[For the treatment of retinal hæmorrhages see remarks under treatment of vitreous hæmorrhages (page 560).]

517. Anæmia of Retina.—Anæmia of the retina may be of sudden or gradual development. The former may follow occlusion or compression of the arterial vessels, as occurs in sudden increase of tension and especially in embolism (518). Spasm of the retinal arteries also occurs, particularly in acute quinine poisoning (page 627). Much more frequent than acute anæmia of the retina is that form which sets in gradually in consequence of retinal atrophy.

The retinal vessels then either become simply attenuated (Fig. 258) or surrounded by white bands, the result of thickening of the vessel walls, the blood column being simultaneously narrowed (*perivasculitis retinæ*). [See C and D, Fig. 247 and B and F, Fig. 251.] Ultimately the vessels may vanish altogether from the retina or be transformed into white bloodless strands. [Spasm causes sudden and great attenuation of the retinal arteries and transient or permanent blurring of sight over part or all of the visual field. Repeated attacks may occur. Inhalations of amyl nitrite relieve the spasm, and may restore the sight. (See also page 575).—D.]

518. Embolism of Central Artery.—This was first observed by Von Graefe. The patient is made aware of his disorder by sudden and complete blindness, which appears at once upon the obliteration of the artery. If immediately afterward an examination is made with the ophthalmoscope, the signs of an extreme arterial anæmia of the retina are found (Fig. 249) [Fig. 251, A and B]. The larger arteries are narrowed down to thin filaments, the smaller ones become invisible. The veins, on the other hand, are not markedly contracted except upon the papilla itself; the latter appears paler. Within a short time, often within a few hours, the retina, as it dies, loses its transparency. It becomes of an opaque milky white, most markedly so in the vicinity of the papilla and of the fovea centralis. Owing to this opacity the outlines of the papilla are obscured, and, on the other hand, fine ramifications of the retinal vessels, such as ordinarily are not visible with the ophthalmoscope, come into view in the vicinity of the fovea, where they stand out with great distinctness upon the white background.

At the center of the fovea a vivid red spot stands out on the cloudy white background, because the inner layers of the retina and hence also the retinal cloudiness are lacking at this spot, and consequently the fundus appears of its normal red color, which looks even more pronounced from contrast with the turbid white area surrounding it. In this region also small retinal hæmorrhages sometimes occur.

After some days the retinal vessels fill up again. Then a peculiar phenomenon is sometimes observed. In some sets of the vessels (particularly the veins) the blood column appears to be broken up into short sections separated by clear interspaces, and the whole column makes jerking movements, sometimes in the sense of the normal blood current, sometimes in the reverse direction. In the course of the following

Fig. 249.—Embolism of the Central Artery, Developing Eight Days Previous to the Date of Observation in a Woman Affected with Aortic Aneurysm.

The whitish haze over the retina obscures the outlines of the papilla and the initial portions of the vessels arising from it. The arteries are already better filled than they were, although they still are below the normal in this respect. The veins have a very uneven caliber, the latter in general increasing toward the periphery. In the large veins, running upward and outward and downward and outward, the blood column is seen to be broken up into short separate sections. The vicinity of the fovea centralis is covered with a dense whitish haze, upon which the finest ramifications of the vessels stand out distinctly, although the connection between them and the main vessels is in places concealed by the haze. The mid point of the fovea centralis is occupied by a spot which is dark red with a light center, and which represents the chorioid showing through the haze.

weeks the cloudiness of the retina vanishes; the retina regains its transparency, but becomes perfectly atrophic. The optic papilla is now white and sharply outlined, the blood-vessels both upon the papilla and in the retina are scanty, thin, filamentous, and often bordered by white lines; many of the finer vascular twigs become completely invisible. The blindness persists and is permanent.

Instead of affecting the central artery, the embolism may effect only a *branch* of it. In that case the visible alterations are limited to that portion of the retina which draws its blood supply from the obliterated vessel. The blindness, too, corresponds then simply to the part of the retina that is diseased—that is, appears under the form of a defect in the visual field, one half of the latter or a sector of it being destroyed. Moreover, even in embolism of the central artery itself a small portion of the retina

may retain its functional power. This is the case when cilio-retinal vessels from the vascular circle of Zinn are distributed to the retina (see page 382). In ophthalmoscopic examination such vessels can be recognized from the way in which they are seen to arise, which is by a hook-shaped extremity from the border of the papilla (Fig. 155). These vessels, which receive their blood from the short posterior ciliary arteries, are of course unaffected by embolism of the central artery, and hence the region of the retina that is supplied by them, and which lies between the papilla and macula, retains its functional power. [See Fig. 251 B.]

Embolism occurs in all those disorders which give rise to the entrance of clots into the circulation, and above all in affections of the heart. The possibility of a cure exists only in very recent cases, before the death of the retina has taken place. The retina might then regain its function if we could succeed in restoring the circulation in it. The only way in which this can be accomplished is for us to try to drive the plug lodged in the central artery on into its smaller branches, where it will do less harm. With this object in view, we draw off the aqueous by paracentesis of the cornea. In consequence of the sudden diminution of intra-ocular tension thus produced, the blood tends to flow into the eye in increased quantity, and may thus push the embolus forward if it is not too tightly fixed in its position. In addition, we try to favor the washing away of the embolus by massaging the eye [(see page 58), and dilate the retinal arteries by giving inhalations of nitrite of amyl.—D.] In this way it has been possible to restore the circulation in the retina, and with it the sight in some few cases, in which the lesion was still very recent.

What is known clinically under the name of embolism of the retinal arteries corresponds to those cases in which there are no infected emboli in question; hence, no inflammation occurs, but only the results of the mechanical cutting off of the blood supply. The retina, being no longer nourished, simply dies. It does not indeed become necrotic, because it still obtains a supply of nourishment from the adjacent chorio-capillaris of the chorioid, although this does not suffice to maintain the function of the retina. But infectious emboli also may get into the retinal arteries, as sometimes happens in pyæmia. Then a suppurative retinitis develops, the suppuration from which soon extends to the other structures of the eyeball, so that the clinical picture of panophthalmitis is produced (metastatic ophthalmia; see pages 420 and 471).

The ophthalmoscopic picture of embolism is the expression of changes which set in when the supply of arterial blood is cut off from the retina. It is hence not confined to embolism of the central artery, but is found in occlusion of it in general, due to other causes as well. Such occlusion may be produced by endarteritis, by thrombosis of the artery, and also by spasm of it, in case it lasts long enough. [Evidence of extreme spasm with occlusion can sometimes be furnished by the ophthalmoscope. We see then a thread-like bloodless vessel which as we look at it (perhaps under the influence of amyl nitrite) dilates to full size once more. Some would ascribe to spasm many of the cases regarded as embolism; particularly the cases in which the complete occlusion has been preceded by repeated attacks of transient obscuration.—D.] The ophthalmoscopic picture of embolism may also be caused by compression of the artery within the trunk of the optic nerve consequent upon hæmorrhage or inflammatory infiltration in the latter; and by a solution of the continuity of the artery occurring when the optic nerve is cut or torn through in front of the point where the central vessels enter it.

519. Thrombosis of Central Vein.—Thrombosis, which was first anatomically demonstrated by Michel, is characterized by an enormous distention of all the retinal veins with blood, while the arteries are so attenuated as to be scarcely discoverable (Fig. 250). [See also Fig. 251 C and D.] The

blood escapes from the turgid veins at many spots so that the entire fundus is covered with hæmorrhages. These keep recurring again and again, and with their recurrence the sight which from the start was greatly reduced is at length utterly destroyed.

Thrombosis may be confined to a *branch* of the central vein, in which case the changes in the fundus are present in that portion only of the retina which represents the area of distribution of the occluded vein. The prognosis then is more favorable than in occlusion of the main trunk. The disturbance of the circulation may be compensated for by the capillary vessels of collateral channels—capillary vessels in this case dilating to form larger vessels which can be seen with the ophthalmoscope.

FIG. 250.—THROMBOSIS OF THE CENTRAL VEIN. PRESENT FOR FOURTEEN DAYS IN A MAN FIFTY-TWO YEARS OF AGE.

The outlines of the papilla are concealed partly by a gray striate opacity, partly by radially disposed hæmorrhages. The retinal arteries are attenuated; the retinal veins are unusually broad and tortuous, and are filled with blackish blood. In many places the vessels are covered by extravasations of blood, and hence appear interrupted. The hæmorrhages are extremely numerous, have a dark-red, almost black, color, and are either radiately striate or irregularly rounded in shape. A few of the hæmorrhages have taken on a brilliant-white hue in their center. This is particularly visible in the large patches of blood lying outside of the macula lutea. The retina, wherever it is not occupied by hæmorrhages, is clouded and grayish.

The marked venous stasis, which must occur as a result of a venous thrombosis, makes it easy to see why *increase of tension* frequently occurs in such cases. This so-called hæmorrhagic glaucoma gives a bad prognosis, since it is generally incurable, even by iridectomy.

Thrombosis of the central vein occurs for the most part in elderly persons suffering from *arteriosclerosis*; but an *inflammation in the orbit* may also lead to thrombosis of the central vein, probably through the development of the orbital veins of thromboses, which subsequently extend into the central vein. In this way cases of blindness are sometimes produced in the course of facial erysipelas. The erysipelatous inflammation of the skin has a tendency to penetrate in spots into the deeper parts, and there set up either infiltrations or phlegmons. Hence, abscesses of the lids, abscesses in the orbit, and, through transmission to the brain, even purulent meningitis, are observed as sequels of facial erysipelas. If, then, a case of erysipelas is complicated with inflamma-

tion of the cellular tissue of the orbit, it is sometimes found, after the erysipelas has run its course and the swelling of the lids has abated, that the eye is blind. The ophthalmoscope shows atrophy of the optic nerve with extreme attenuation of the blood-vessels. According to an observation of Knapp's, we are dealing in this case with a thrombosis of the central vein, consequent upon the inflammation of the retrobulbar cellular tissue. The blindness that is due to erysipelas may affect both eyes.

II. Inflammation of the Retina

520. Symptoms.—Inflammation of the retina (retinitis) is characterized first of all by a diffused cloudiness of the organ. The cloudiness varies very greatly in intensity, although in general it is greatest in the vicinity of the papilla, because here the retina is thickest. Consequently, the outlines of the papilla become indistinct and the vessels in the retina hazy. In addition, circumscribed exudates occur in the retina, usually under the form of brilliant-white, sharply defined patches. Retinitis is always associated with hyperæmia of the retina, evidenced by distention and tortuosity of the vessels, and often also by extravasations of blood. Owing to the passage of the exudate from the retina into the vitreous, opacities of the vitreous are produced.

The function of the retina is impaired in proportion to the intensity and extent of the inflammation. In the lightest cases vision may be normal, so that the patients complain simply of the presence of a light-colored cloud before their eyes. But for the most part vision is very considerably reduced, both because of the changes in the retina itself and because of the accompanying opacities in the vitreous. Circumscribed exudates cause fixed scotomata in the field of vision. [Retinitis affecting the region of the macula lutea is marked by a central scotoma and by retinal metamorphopsia (micropsia, macropsia—see page 125 and cf. page 465).—D.]

The course of the retinitis is always pretty sluggish. Only in the lightest cases does the inflammation abate completely in a few weeks, and then the sight may again become perfectly normal. But for the most part it takes several months for all inflammatory symptoms to disappear from the retina and the sight remains permanently impaired. Severe and, more particularly, recurrent inflammations of the retina lead to atrophy of it (see page 585), pigmentation frequently occurring at the same time (through migration of pigment from the pigment epithelium). When atrophy of the retina has once set in the sight is always abolished, either completely or all but a remnant, and cannot be restored.

A *new formation of blood-vessels* may occur in retinitis proliferans and also whenever a retinitis, particularly a syphilitic retinitis, has lasted a long time. The vessels in this case project from the retina into the vitreous under the form of slender and, often also, of repeatedly convoluted coils.

[For the pathological changes occurring in retinitis, see page 589.]

521. Etiology.—In the etiology of retinitis, general affections play the chief part. Retinitis appears but rarely as a local lesion, e. g., when due to

dazzling; in most cases it is simply the symptom of an internal or general disease, to the discovery of which we are often led just by finding the retinitis. Among such general diseases are, above all, albuminuria, diabetes, leuchæmia, syphilis, uratic gout, and diseases of the vascular system. In these cases, in which a general disease underlies the retinitis, the latter is usually bilateral.

It is very rare indeed for retinitis to remain confined strictly to the retina, the fact being that it usually implicates the head of the optic nerve as well. If this implication is present to a marked degree we call the case one of *neuro-retinitis*. We use the same term when an inflammation that starts from the head of the optic nerve extends out into the retina. With regard to its etiology, therefore, neuro-retinitis is at one with retinitis on the one hand and with neuritis on the other. A similar relation exists between retinitis and chorioiditis. An inflammation that affects predominantly the outer layers of the retina, adjoining the chorioid, will scarcely run its course without implicating the chorioid too. Of this, syphilitic retinitis affords a very evident example. Conversely, it is obvious that chorioiditis cannot exist without the portions of the retina immediately adjacent participating in the process. From an anatomical standpoint every chorioiditis is a *retino-chorioiditis* (or *chorio-retinitis*), although we generally do not use this term unless ophthalmoscopically visible signs of inflammation can be demonstrated to exist both in the retina and in the chorioid.

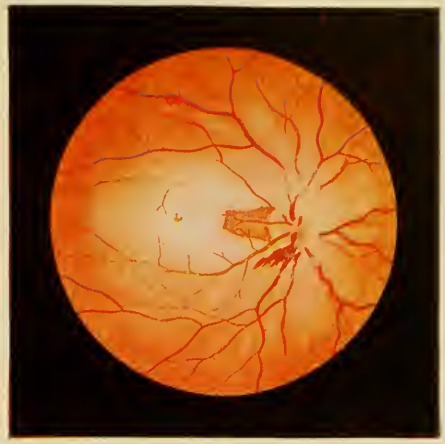
522. Varieties.—The main varieties of retinitis are as follows:

1. **Retinitis Albuminurica.**—This of all inflammations of the retina is the one best characterized. In addition to the general signs of retinitis, such as haziness of the retina and of the outline of the papilla, distention of the retinal arteries, and hæmorrhages, it is particularly distinguished by the white patches in the fundus (Fig. 252). [Fig. 255, C and D.] The white spots are found chiefly in two places—in a certain area surrounding the papilla and in the macula lutea. In the former situation we find rather large white patches, which may be so numerous as to form a speckled zone, which is usually interrupted at a point corresponding to the macula lutea. The latter, on the other hand, is occupied by small white stipplings radially disposed, so that a pretty radiating crown is formed with the fovea centralis at its center. Very often the crown is incomplete, the rays being fully formed only on certain sides of it.

Besides this form of retinitis, affections of the fundus occur in albuminuria that present no such characteristic appearance. Such affections comprise simple retinal hæmorrhages, retinitis hæmorrhagica (Fig. 254), and neuritis (including even choked disk). On the other hand a picture very similar to that of typical retinitis albuminurica may be produced by other causes, such as brain tumor, diabetes, and arteriosclerosis. [See Retinitis Stellata, page 579.] Hence an examination of the patient's urine should be made in every case of retinitis.

All forms of kidney disease that result in albuminuria may be complicated with retinitis, but the one most frequently thus complicated is the atrophic kidney. The severity of the retinitis bears no fixed proportion to the intensity of the kidney disease nor to the amount of albumin in the urine. The like is true of the subsequent course; the retinitis may improve while the kidney lesion grows worse, or vice versa. Nevertheless, retinitis albuminurica is, on the whole, of evil prognostic significance. Even if it does occur sometimes with benign kidney lesions (e. g., with scarlatinal nephritis and the renal affection of pregnancy), yet it far more frequently is associated with the serious chronic cases, and it is a matter of experience that most patients suffering with retinitis albuminurica succumb from their renal disorders within a few years. (For the connection between the retinitis and the renal disorder, see page 28.)

A



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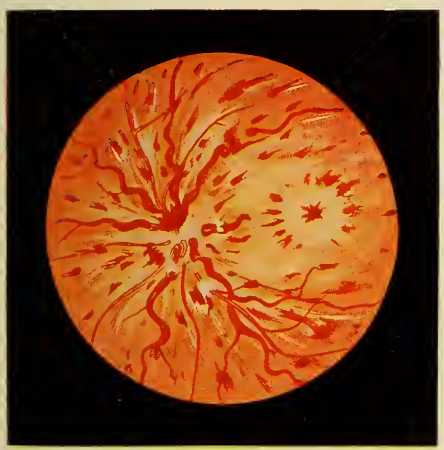

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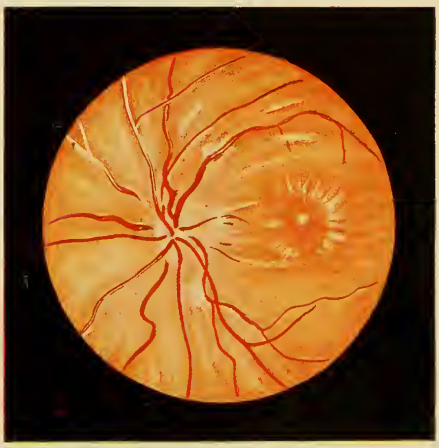


D

E

F

[FIG. 251.—CHANGES IN RETINAL VESSELS. (After Würdemann in Posey and Spiller.)

A, Embolism central artery; partial, affecting only inferior branch (Haab). B, Embolism central artery; total within nerve; a cilio-retinal vessel supplies a small area of retina in which function is preserved (Würdemann). C, Thrombosis of central vessels from mumps (Würdemann). D, Same case six months later, showing sclerosis and atrophy (Würdemann). E, Hæmorrhages from retinal vessels (Magnus). F, Perivasculitis luetica (Magnus).—D.]

In nephritis disturbance of vision may occur also under the form of a transitory blindness without any retinitis being present. The patient declares that everything suddenly becomes dark before his eyes; the disturbance of sight increases so quickly that the blindness gets to be complete within a few hours or a day. Even, however, when the blindness is absolute, the results of examination of the eye are usually negative. After one or more days the sight is gradually restored. Simultaneously with the attack of visual disturbance other nervous symptoms are found, such as headache, vomiting, dyspnœa, loss of consciousness, and convulsions—in short, the symptoms of uræmia. The blindness is therefore known as *uræmic amaurosis*. The fact that the reaction of the pupil to light is in most cases preserved in spite of the complete blindness proves that the location of the affection cannot be in the eye or in

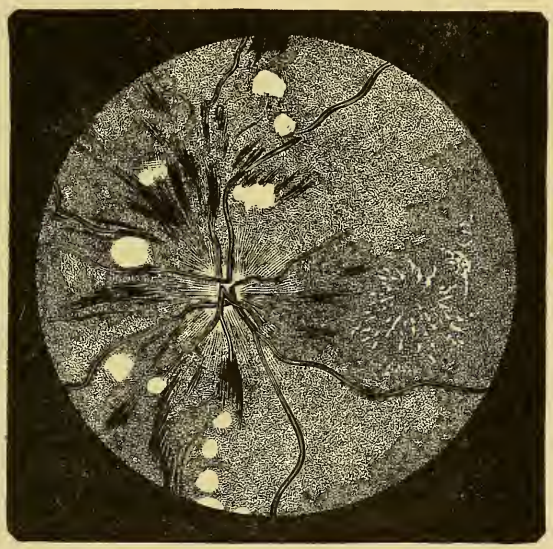


Fig. 252.—Retinitis Albuminurica.

The clouding of the retina is most pronounced in the region of the papilla, where it exhibits a fine radial striation, and completely veils the margin of the nerve. Furthermore, even at a considerable distance from the papilla, the retinal clouding covers isolated portions of the vessels and especially the distended veins, with a delicate haze, so that the vessels in these places look lighter. Surrounding the papilla are found rounded, brilliantly white spots of exudation and numerous dark-red, radially striate hæmorrhages. The latter lie mainly in the neighborhood of the larger retinal vessels, and in part cover them. From this fact and from their striate appearance, it can be inferred that they belong to the most anterior layer of the retina—the nerve-fiber layer. In the region of the macula lutea is seen a group of small white specks, which combine to form the stellate figure characteristic of retinitis albuminurica. In the present case this is not very regularly formed, and above it is a somewhat larger spot, produced by the coalescence of several small dots.

the optic nerve, but is higher up—that is, in the brain, which is poisoned by the excretory matters retained in the blood. Uræmic amaurosis is distinguished from the disturbance of vision due to retinitis albuminurica partly by the negative results of ophthalmoscopic examination, partly by the course. Uræmic amaurosis sets in suddenly and is complete, while in retinitis albuminurica the sight is reduced gradually and is seldom annihilated entirely. The blindness, however, is permanent, while the blindness due to uræmia gives place to normal vision again, provided the patient does not succumb to the uræmic attack. Of course, the possibility of a uræmic amaurosis occurring in a patient already suffering from retinitis is not excluded.

[2. **Retinitis Stellata.**—This is also called *pseudo-nephritic retinitis*, because it resembles albuminuric retinitis in having the characteristic star figure at the macula,

but is not due to nephritis. One form occurs as a unilateral or bilateral affection, especially in young people and without discoverable cause. It may or may not be associated with a slight papillitis. The visual disturbance is often moderate, and after a long time complete recovery may take place. Other cases are more acute in onset, being preceded by violent pain in the eyes and head and accompanied by marked neuro-retinitis with great narrowing of the arteries. These cases are helped by salicylic acid. Other cases occur with optic neuritis due to influenza, syphilis, or chlorosis; and a star figure in the macula also accompanies some cases of choked disk due to brain tumor, meningitis, or hydrocephalus. Other cases, marked by sudden blindness or sector-like limitations of the field are caused by circulatory disturbances (probably embolism) in the retina. Finally some occur in severe injuries of the skull, produced by blunt force (Leber).—D.]

[3. **Retinitis Cachecticorum** is a form, also more or less resembling albuminuric retinitis, occurring in carcinomatous subjects (Leber).—D.]

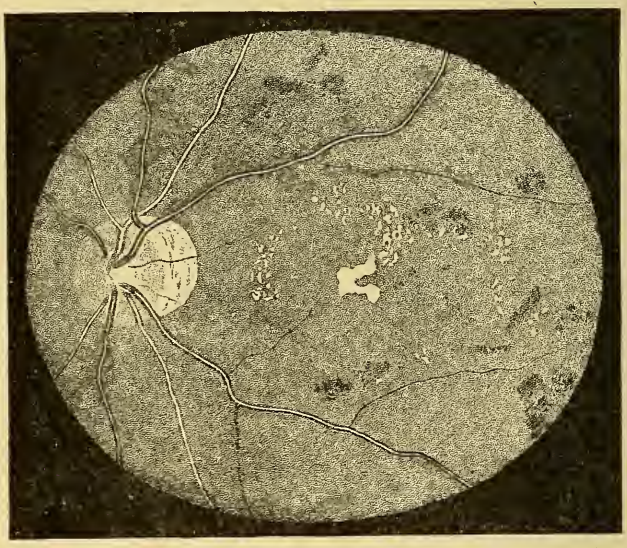
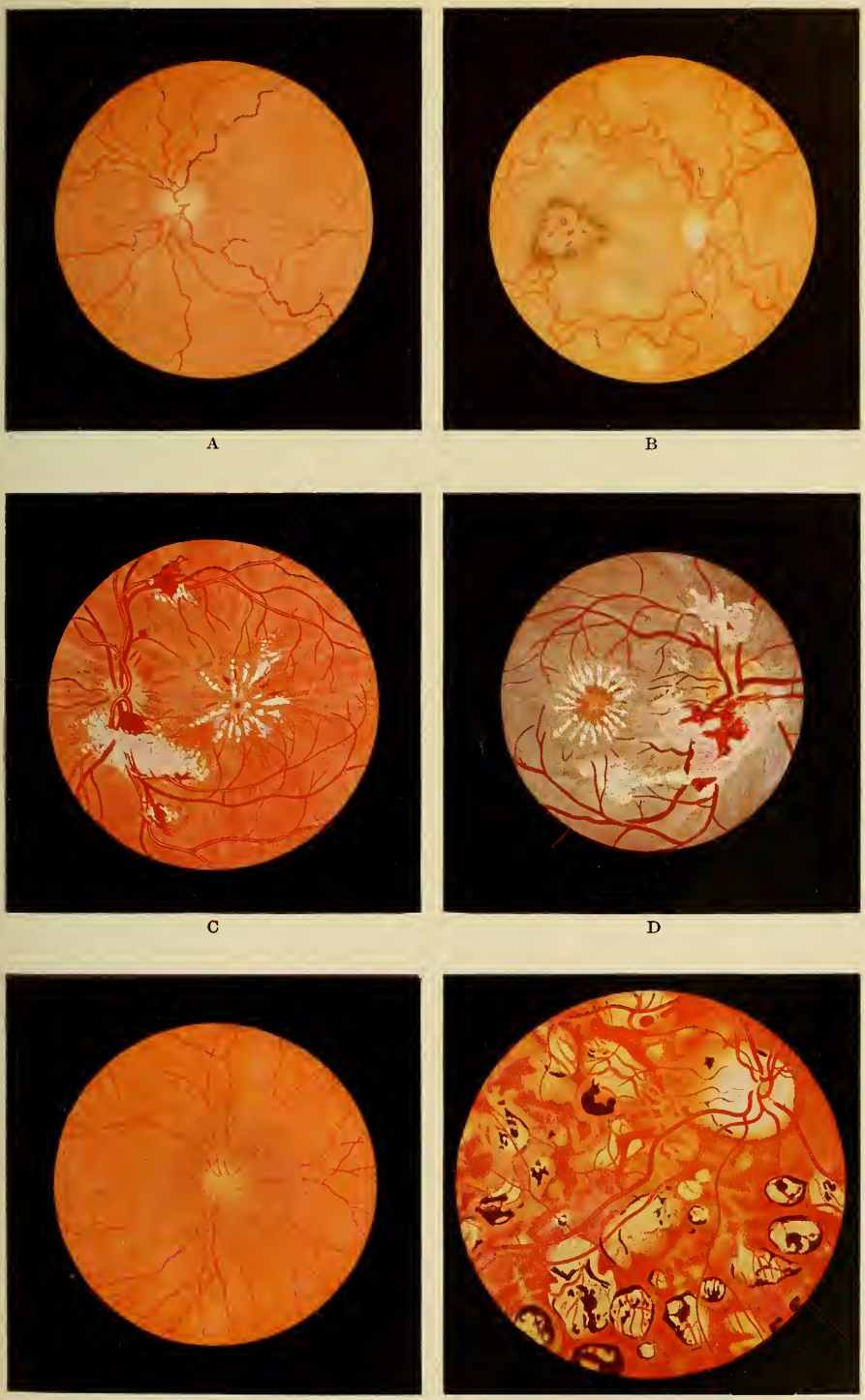


Fig. 253.—Diabetic Retinitis in a Man Sixty-nine Years of Age.

When the diabetes was discovered eleven years previous six per cent of sugar was present, while for some years past, under the influence of suitable treatment, the sugar has almost disappeared. The disturbance of sight has lasted for nine months, and is caused mainly by a central scotoma for blue—a scotoma which corresponds to the exudate in the macula. In keeping with the myopia of the eye there is an atrophic crescent, about half the width of the papilla, adjoining the optic nerve. In its posterior section the retina shows numerous punctate hæmorrhages, which are generally disposed in groups, and frequently coalesce to form rather large patches. In addition, small, brilliantly white dots are present of irregular shape and sharp outline. These are disposed quite irregularly in a large circle surrounding the macula. In the macula itself is found quite a large exudate having a structure like that of the small dots.

4. **Retinitis Diabetica.**—This is characterized in many cases by the presence of small, brilliantly white spots in the retina, which chiefly occupy the region of the macula lutea and its vicinity, without, however, presenting a stellate arrangement, as in albuminuric retinitis (Fig. 253). Sometimes by the confluence of small dots one or two large patches are found, which show by their crenated border that they are composed of smaller spots. Between the white patches lie punctate extravasations of blood. The rest of the retina is transparent, and the papilla, too, is unaltered.

In other cases this characteristic picture is not present, in fact, diabetic retinitis may actually appear under the guise typical of albuminuric retinitis [or closely resemble a retinitis circinata (see page 584). Very rarely in young diabetics, especially when



[Fig. 255.—Inflammations of the Retina. (After Würdemann in Posey and Spiller.)

A, Œdema in pernicious anæmia (Oliver). B, Leuchæmic retinitis (Oliver). C, Albuminuric retinitis and neuritis of pregnancy (Würdemann). D, Albuminuric retinitis in the negro (Würdemann). E, Syphilitic retinitis (Haab). F, Atrophy of retina, chorioid, and nerve following chorio-retinitis luetica (Oeller).—D.]

nearing death, *retinal lipharmia* is found, i. e., a condition in which on account of the large amount of fat in the blood, the retinal vessels appear reddish-white or pure white.—D.]

Retinitis is also observed in *oxaluria*.

5. **Retinitis Leuchæmica.**—In this, superadded to the general symptoms of inflammation of the retina (namely, retinal cloudiness and hæmorrhages), there is found, as a characteristic feature, a light hue of the blood in the retinal vessels, which ordinarily are greatly dilated. Furthermore, since in leuchæmia the blood that flows in the chorioidal vessels is of lighter color than normal, the entire fundus, even where there is no retinitis present, is of a much lighter red than usual, and shows a yellowish tinge.

FIG. 254.—HÆMORRHAGIC RETINITIS IN A WOMAN OF FORTY-EIGHT, WHO SUFFERED FROM CHRONIC NEPHRITIS AND CARDIAC HYPERTROPHY.

The outlines of the grayish-red papilla are obscured, the arteries of the retina are somewhat contracted, and in places are concealed by the cloudiness of the retina. The veins are very tortuous, and from the character of their reflex streaks it can be seen that not all their convolutions lie in the same plane. The retina, as a whole, shows a faint striate opacity, and is filled with very numerous, partly striate and partly rounded hæmorrhages. To the left and below, between the two branches of a vein, is a white spot which has originated from a hæmorrhage.

Likewise characteristic of leuchæmic retinitis are white patches with a red rim (consisting of white corpuscles surrounded by red ones); but such patches are present in only a few cases of retinitis leuchæmica. [See Fig. 255, B.]

6. **Retinitis Septica.**—In this the changes affect mainly the posterior section of the retina, in which both hæmorrhages and white patches are found. The papilla is unaltered. The disease occurs in connection with sepsis, and that not only in the fatal forms, but also in the slighter cases.

7. **Retinitis Hæmorrhagica.**—This is diagnosticated when, along with the presence of numerous hæmorrhages in the retina, the latter itself is hazy and the papilla is obscured (Fig. 254). Hæmorrhagic retinitis is due for the most part to diseases of the retinal vessels; many of these cases are probably identical with those of thrombosis of the central vein, as described on page 575.

8. **Retinitis Proliferans.**—Manz described under this name an affection in which dense masses of connective tissue extend out from the retina into the vitreous and cover a portion of the fundus—in fact, even the papilla itself (Fig. 256). Into these masses run new-formed vessels from the retina. For a number of these cases it is probable that these masses of connective tissue have been preceded by hæmorrhages, which were poured out from the retina into the vitreous and afterward became organized (see page 571). [Much the most common cause of these are the spontaneous hæmorrhages occurring in the retina and vitreous in young persons particularly as a result of tuberculosis (see pages 562 and 573). Other causes alleged are anæmia, chlorosis, menstrual congestion, syphilis, nephritis, diabetes, arteriosclerosis, oxaluria, etc. Other cases are

FIG. 256.—RETINITIS PROLIFERANS.

In a man thirty-five years of age, who suffered frequently from palpitation of the heart and nose-bleed, sudden obscuration of the sight occurred in both eyes five years ago. This obscuration has recurred repeatedly since, being due to recurrent hæmorrhages into the vitreous. In the vitreous of the left eye close to the papilla lies a shining white mass having a structure partly fibrous, partly granular. From this mass pass strands in different directions, some extending as far as the periphery of the fundus. No vessels are recognizable in the white mass, which covers the upper half of the papilla. The other half of the papilla and the rest of the fundus are somewhat hazy (owing to the presence of a faint opacity of the vitreous), and besides appear indistinct, because the ophthalmoscope is adjusted not for them, but for the connective-tissue mass, which is situated farther forward. Indistinctly visible are indications of the retinal vessels (above and to the outer side) and indications of a tessellation of the fundus (below and to the inner side).

traumatic. Such are those in which we find dense white masses encapsulating a foreign body or an entozoön or dense cicatricial masses traversing the vitreous as the result of a penetrating or non-penetrating injury (especially that inflicted by some projectile) (Leber). Some of these cases retain surprisingly good vision.—D.]

9. **Retinitis Syphilitica.**—Acquired syphilis is one of the most frequent causes of retinitis. Syphilitic retinitis is usually associated with disease of the uvea, and above all of the chorioid; often also of the iris, which then exhibits the picture of syphilitic iritis. In the retina inflammation appears under two forms, *diffuse* and circumscribed. In the former case the entire retina is clouded and faintly gray [see Fig. 255, E and F]; here and there, especially in the macular region, more densely gray spots may be found. Later on in the disease, in proportion as the cloudiness of the retina abates, changes in the pigment epithelium set in, and ultimately migration of

pigment from the latter may take place into the retina, so that a picture is formed resembling that of retinitis pigmentosa. This form of syphilitic retinitis consequently coincides in part with the syphilitic chorioiditis described by Förster (page 459).

In the *circumscribed* form a bulky white exudate is found either in the region of the macula lutea or more frequently close to one of the larger retinal vessels. In the latter case it is often possible with the ophthalmoscope to recognize that an affection of the wall of the vessel is the cause of the circumscribed exudation. Later on the exudate is transformed into bluish-white scar tissue, which by shrinking may give rise to detachment of the retina.

Hereditary syphilis, too, leads to retinitis, which may be observed in children or may be even congenital. Ordinarily we see only the evidences of inflammation after it has run its course; such evidences occurring either under the form of numerous small, light or black spots, or under the form of an old, bulky exudate, that has been transformed into connective tissue.

10. **Retinitis due to Dazzling.**—This is produced most frequently by looking at the sun [*solar retinitis*] or at the electric arc light [*electric retinitis*]. The ophthalmoscope shows pigment changes in the macula lutea, upon which the image of the sun has been cast. Corresponding to this spot there is a central scotoma, which for the most part remains permanently (see page 22). [This is often combined with metamorphopsia. —D.] We must not confound with the retinitis due to dazzling that variety of inflammation of the eyes which is produced by the action of snow (snow blindness) or by the electric arc light. This, in addition to transitory symptoms of dazzling, consists mainly in a violent conjunctivitis (see page 24).

11. **Retinal Changes at Macula.**—The macula lutea has the most delicate anatomical structure of any of the tissues of the eye, and is hence also specially vulnerable. We find it therefore diseased particularly often, e. g., in myopia where it participates in the affection of the subjacent chorioid (page 463). But the macula is also often injured in cases in which it is not in any way directly affected by the agent causing the injury. Thus a circumscribed affection of the macula often develops after contusion of the eyeball, or in the case of foreign bodies in the vitreous, or in compression of the eye by orbital tumors. From rarefaction of tissue at this spot [*retinitis atrophicans centralis*] an actual *hole* may be produced in the retina. Under the ophthalmoscope, this hole looks like a deep red disk at the site of the macula (Kuhnt, Haab). [This is usually one-third to one-half a disk diameter in width, and 0.3 to 0.5 mm. deep. The vision is very much reduced (Leber).—D.]

In the last-mentioned forms of retinitis—namely, retinitis syphilitica and retinitis due to dazzling—the inflammation has its seat mainly in the outer layers of the retina, as is proved by the changes simultaneously taking place in the pigment epithelium and, indeed, in the chorioid itself. But in the forms of retinitis first described it is mainly the inner layers of the retina that are affected.

Often enough cases of retinitis occur in which even a careful examination of the patient is unable to demonstrate any determining etiological factor. Among these is—

12. **Retinitis Exudativa,** so called because it is characterized by particularly massive exudation into the retina (Coates). In the latter there are numerous large and small white spots, which are often arranged in groups, and later may become confluent, or there are extensive white areas, usually somewhat elevated above the level of the retina. [They may, indeed, project like a veritable tumor.—D.] Frequently the blood vessels take a prominent part in the process either under the form of a general dilatation or a localized aneurysmal widening or the formation of numerous new vessels. These cases are described under the name of *Hippel's retinitis* or *angiomatosis retinæ*. [This is often classed as a distinct disease—angiomatous degeneration or primary angioma. The arteries and veins are greatly distended, and there is a rounded prominence into

which one of the distended vessels empties (Leber). Both conditions are very chronic and in both] detachment of the retina usually sets in later, followed by increase of tension or irido-cyclitis and complete blindness. Exudative retinitis is rare and its cause unknown. It usually attacks young persons, and more frequently males, and commonly affects but one eye. [Coates, who ascribed it to hæmorrhages in the outer layers of the retina, called it *retinitis hæmorrhagica externa*.—D.]

13. **Retinitis Circinata.**—Several forms, which are, to be sure, of rare occurrence, are distinguished by characteristic changes in the fundus from which they get their names—e. g., retinitis circinata from the circle of white spots.

14. **Amaurotic Family Idiocy.**—This very rare affection of the retina occurs in children in the first two years of life with the following symptoms: The region of the macula lutea is occupied by a grayish-white patch of the size of the papilla, having in its center a small vividly red spot like that found in embolism of the central artery. The

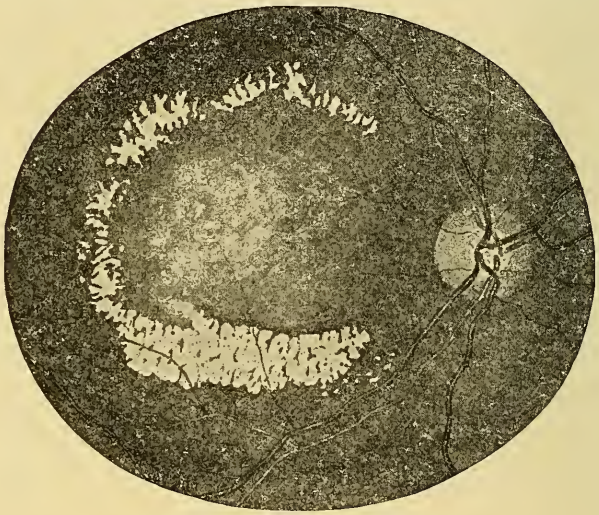


FIG. 257.—RETINITIS CIRCINATA.

Right eye of a woman 70 years of age. Erect image. The papilla and the retinal vessels are normal. The macula lutea and its vicinity are transformed into a yellowish-gray, not sharply limited area which is enclosed by a zone composed of white spots. The spots run together, to form map-like figures. On the side toward the papilla the spots are absent, so that the ring here is open.

rest of the fundus is normal, although the papilla becomes paler and paler all the time and finally altogether atrophic. The changes are always found in both eyes, and are alike in both. The children gradually become blind, and exhibit apathy and paralytic weakness of the muscles. These symptoms increase until after the lapse of many months the children die. Autopsy shows changes in the cerebral cortex [and ganglion cells of the retina (Holden)] and descending degeneration in the spinal cord.

The cause of the disease, which sometimes affects several children of the same family, is unknown. [The disease attacks particularly children of Jewish parentage.—D.]

523. Treatment.—The treatment must be directed both against the causal lesion and against the local affection of the retina. The first indication is most readily fulfilled in cases of syphilitic retinitis, where energetic mercurial treatment in most cases results in rapid improvement. In albuminuric retinitis due to pregnancy artificial interruption of pregnancy may

be considered. The symptomatic treatment consists in fully safeguarding the eye by forbidding all work and by protecting the eye from glaring light, either by means of dark glasses or in severe cases by confinement in a darkened room. To combat the inflammation, and also to cause resorption of the exudate and restoration of the transparency of the vitreous, mercury, potassium iodide (both remedies being used in non-syphilitic as well as in syphilitic cases), saline purgatives, diaphoretic treatment, and subconjunctival injections of salt [or mercury oxycyanide] are employed.

III. Atrophy of the Retina

524. Atrophy of the retina is the result of its protracted inflammation, or is the final outcome of an embolism or a thrombosis of the retinal vessels.

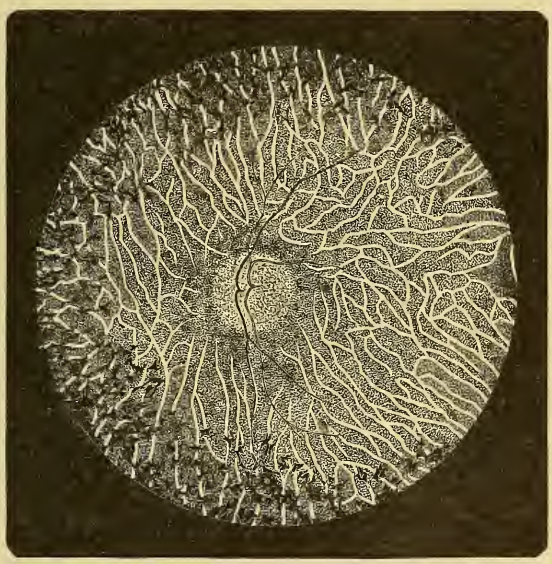


Fig. 258.—Retinitis Pigmentosa. (In part after Jäger.)

Owing to the disappearance of the pigment epithelium, the stroma of the chorioid is exposed, so that the bright-red chorioidal vessels with the darkly pigmented intervascular spaces are everywhere visible. For the same reason numerous branched, interconnected pigment spots are found in the periphery of the retina. This pigmented zone extends in a circle, although it comes closer to the papilla on the nasal than on the temporal side, where in fact, it lies so far to the periphery that it is not represented in the drawing at all. The papilla is of a dirty grayish-yellow color and ill defined. Of the retinal vessels, only the main trunks are visible, and these, especially the arteries, are greatly contracted.

Ophthalmoscopically, atrophy is characterized above all by the stenosis of the retinal vessels (Fig. 258; 251,D), which in severe cases may amount to complete obliteration, so that the vessels are either transformed into white strands or have become altogether invisible. The retina may look otherwise unchanged and transparent, or it may bear traces of the antecedent inflammation. In every case the signs of a secondary atrophy can be made out upon the papilla as well; its outline is indistinct and it is of a pale, dirty-gray color (retinitic atrophy of the papilla).

525. Retinitis Pigmentosa.—A special variety of atrophy which runs a very chronic course is the *pigmentary degeneration of the retina* (also called *retinitis pigmentosa*). This is marked by such characteristic subjective symptoms that the diagnosis can be made almost from them alone. The persons affected with this disease, even when still young, complain that they see worse whenever the illumination is reduced, and particularly at night (hemeralopia). This state of things increases with the age, so that finally the patients are no longer able to go about alone at night, while in the daytime they still see quite well. The cause of this phenomenon is disclosed by the examination of the field of vision.

In the beginning of the disease the visual field, when taken with good illumination, shows a broad blind zone between the center and the periphery, i. e., an annular scotoma (see page 123). Central vision is good, because the periphery of the retina functions, orientation is good too, so that the ring-shaped defect in the visual field is not noticed. The peripheral portion of the retina, however, is undersensitive, and when the illumination is diminished it no longer functions, and then the periphery of the visual field disappears just like the portion that corresponds to the ring scotoma, so that the patient has left only the small central portion lying inside of the latter. In feeble illumination, therefore, orientation is no longer possible for him. And even the central portion of the retina is damaged, because the light sense in general (page 126) is reduced, and hence the liminal stimulus is increased, i. e., the minimum quantity of light required to stimulate the retina is greater than in a normal eye. As the disease advances, the periphery of the retina becomes entirely blind, so that even in good illumination nothing but a small central portion of the visual field is left, and the patient can scarcely guide himself alone even in daytime. At the same time direct vision may still be so good that the patient is able to do fine work. Finally, central vision too is lost, so that complete blindness supervenes. This ordinarily is not the case until late in life (in the sixth decade or later).

[In some cases of retinitis pigmentosa the main disturbance of sight, especially for reading, is caused by a cortical cataract. The densest part of this lenticular opacity lies right in the center of the pupillary area, and hence these patients see worst when the pupil is contracted, i. e., in a strong light. They therefore form an exception to the rule that persons with retinitis pigmentosa see worst when the illumination is reduced. These cases can be recognized by the fact that dilatation of the pupil with atropine or homatropine enhances considerably their ability to read.—D.]

526. Ophthalmoscopic examination shows, as the most prominent symptom of the disease, the presence of small black spots in the retina ("speckled retina," Fig. 258). These are of branched shape, so that they have been compared to bone corpuscles or spiders; they are connected with one another by their processes, and are found especially along the veins.

In the beginning of the disease they occupy only the most anterior portion (periphery) of the visible fundus corresponding to the equatorial region of the eyeball, but as time goes on, new spots keep forming farther and farther back, until at length they reach the macula lutea and the papilla. As fast as the retina becomes pigmented, the pigment epithelium becomes decolorized, so that the chorioidal vessels get to be more and more visible. With the increasing pigmentation of the retina the signs of atrophy of the retina and the papilla become more and more prominent. Accordingly, what takes place is a gradual degeneration of the retina associated with a migration of pigment from the pigment epithelium into the retina (Fig. 259). The degeneration begins in the equatorial region and thence advances toward the periphery and toward the center. In the same way, too, the retina gradually loses its function; the affected portions of it at first are simply less sensitive than before, being still stimulated to action by a pretty strong light; later on they become completely insensitive.

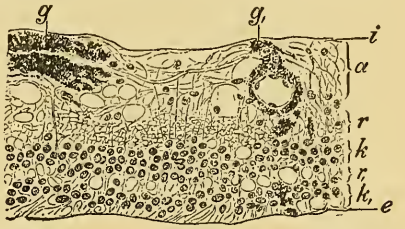


FIG. 259.—RETINITIS PIGMENTOSA. SECTION THROUGH THE RETINA. Magnified 170 × 1.

The retina is bounded on its anterior surface by the membrana limitans interna, *i*, upon its posterior surface by the membrana limitans externa, *e*; the layer of rods and cones that succeeds the latter has disappeared altogether as a result of atrophy. Succeeding the limitans interna is a coarse-meshed network, *a*, which has originated from the supporting tissue of the retina. The nerve fibers and ganglion cells, which normally are inclosed in this supporting tissue, have been completely destroyed. The vessels, however, can still be seen, and are inclosed in pigment. One of them, *g* (very greatly contracted), has been divided longitudinally; another, g_1, transversely by the section. The succeeding layers of the retina—namely, the inner molecular layer, *r*, the inner granular layer, *k*, the outer molecular layer, r_1, and the outer granular layer, k_1,—are altered, and here and there contain pigmented cells.

The black spots in pigmentary degeneration of the retina are not always like bone corpuscles, but sometimes are also rounded or irregular, like the black spots in chorioiditis. Their characteristic distinguishing mark lies not so much in their shape as in their situation, which must be assigned to the retina. This is recognized from the fact that the retinal vessels, wherever they run by the black spots, are covered by them; hence, the spots must lie in front of the vessels—i. e., in the inner layers of the retina. (In the case of pigment spots in the chorioid we can distinctly follow the retinal vessels in their course over the spots.) Spots of pigment in the retina are not, however, limited to pigmentary degeneration of the latter; on the contrary, the migration of pigment into the retina may take place ultimately in every case of retino-chorioiditis. This is particularly the case in syphilitic retino-chorioiditis, in which the pigment in the retina can, moreover, assume the bone-corpuscle shape, so that a picture quite similar to that of pigmentary degeneration may be produced (Förster). In chorioiditis, however, there are also usually present atrophic changes (white spots) in the chorioid, which are wanting in retinitis pigmentosa. [See Fig. 255, F.] Nevertheless there are cases in which the diagnosis is very difficult, and can only be made with the aid of the previous history and the careful testing of the function. Compare also remarks on Ophthalmia Hepatica, page 459.

Not only is pigmentation of the retina not confined to pigmentary degeneration of this part, but, on the other hand, such degeneration is not necessarily associated with the presence of pigment. There are cases of what are called *retinitis pigmentosa sine pigmento*, in which the same gradual attenuation of the retinal vessels, progres-

sive atrophy of the optic nerve, hemeralopia, and ultimate blindness are observed as in retinitis pigmentosa, and only the migration of pigment into the retina is absent. These cases resemble *congenital hemeralopia*, in so far as in this also there is hemeralopia without any pigmentation of the retina being present. A further point of resemblance is that congenital hemeralopia frequently occurs in several members of the same family. The distinction between these cases and retinitis pigmentosa sine pigmento is that in the former the fundus presents no signs of atrophy of the retina or optic nerve, and vision remains good throughout life. The condition, therefore, is a stationary one as opposed to the slowly but surely progressing retinitis pigmentosa.

An affection allied to retinitis pigmentosa is *retinitis punctata albescens* (Gayet, Nettleship). This in all the rest of its symptoms agrees with retinitis pigmentosa, but instead of the pigmentation of the retina shows hundreds of small white dots, which are distributed pretty uniformly over the whole fundus. [These dots closely resemble druses (see page 457 and Fig. 198), with which, indeed, Leber believes they are identical.—D.]

527. Retinitis pigmentosa attacks both eyes. It develops in childhood, and would seem to be congenital in many cases, although it usually is not discovered until some time after birth. Inheritance plays a great part in its production; retinitis pigmentosa occurs frequently in brothers and sisters, and also in several successive generations. The female members of the family are less frequently affected than the males. It is often found at the same time with other congenital anomalies, like deafness, mental weakness, harelip, or supernumerary fingers or toes, or with malformation of the eye, such as persistent hyaloid artery, posterior polar cataract, etc. After it has lasted a pretty long time, posterior cortical cataract usually develops. In almost a fourth of the cases the disease occurs in individuals descended from consanguineous parents. Herein apparently lies the explanation of the fact that pigmentary degeneration of the retina is so frequently associated with other congenital anomalies, since these latter also occur as a result of the consanguinity of the parents.

Treatment is powerless against pigmentary degeneration of the retina, and the prognosis, therefore, is bad, since complete blindness inevitably supervenes—though not, to be sure, until after the lapse of many years.

For treatment we may try mercury, potassium iodide, hypodermic injections of strychnine, the constant current, diaphoresis, and the like. We do this more for the satisfaction of the patient than anything else, for, although we do sometimes obtain an improvement of the sight, it is only a transient one. [Considering the extreme slowness with which the disease progresses, a hopeless prognosis should not be given in these cases, to these patients, who can often get adequate vision for a good many years by properly selected glasses, changed when required. If a cortical cataract develops, causing impairment of sight, this can be removed, sometimes with quite satisfactory results.—D.]

[**528. Other Forms of Retinal Degeneration.**—Allied to retinitis pigmentosa are the conditions knows as *central* and *diffuse tapeto-retinal degeneration* (Leber). In the former, which is nearly always bilateral and usually occurs in several members of the same family, white spots (druses) and varying pigmentary changes occupy the maculo-papillary region, and in the latter there are fine diffuse pigmentary changes. This central form develops usually after the age of ten, but sometimes not till after middle life or

later, and causes progressive diminution and ultimate loss of central vision. The diffuse form causes total or nearly total blindness, which may begin at or about birth or not till later in childhood. It often occurs in several members of the same family and is frequently associated with cerebral degeneration producing idiocy (*amaurotic family dementia*—a condition not to be confounded with the amaurotic family idiocy, described on page 584, in which the site and nature of the lesions are different). It is a not infrequent cause of congenital blindness.—D.]

[Another form of degeneration is characterized by a very extensive atrophy of the pigment epithelium and the chorioid, so that the normal red of the fundus is replaced by a great glistening white area. This may occupy nearly the whole fundus, a small portion round the macula and nerve being alone exempt (*atrophia retinæ totalis*); or may form a peripherally situated, lobulated white ring, surrounding a more or less intact central red area (*atrophia retinæ gyrata*). Pigmentation usually occurs as in retinitis pigmentosa, and, as in the latter, the condition often is found in several members of the same family (Leber).—D.]

[*Cystoid degeneration* of the retina, perhaps due to œdema of the latter, occurs as a senile change near the ora serrata and in younger subjects in other parts of the retina, in eyes affected with a variety of diseases. Sometimes it occupies the macula, producing a hole there. Rarely, if ever, is cystoid degeneration diagnosticated with the ophthalmoscope, being discovered only with the microscope in enucleated eyes (Leber).—D.]

A special form of pigmentation of the retina are the *angioid pigment streaks*. In this condition a brownish or reddish stripe surrounds the papilla, at some distance from it, and sends off radiating striæ toward the periphery. These look like vessels, but are more ribbon-like and varicose and not of as pure a red as blood-vessels. They lie in the outermost layers of the retina; their cause and the way in which they develop have not yet been certainly determined.

529. Anatomical Changes in Inflammation and Atrophy.—In inflammation the signs of inflammatory œdema exist, or those of a cellular infiltration due to extravasated white blood corpuscles; also extravasations of blood. The changes at the same time observed in the tissue elements of the retina itself are: 1. Fatty degeneration both of the nervous elements and of the supporting tissue of the retina. 2. Thickening (sclerosis), especially in the nerve fibers of the fiber layer. 3. Free exudate under the form of homogeneous masses interposed between the tissue elements. The three changes just mentioned constitute the principal cause of the brilliant-white spots occurring in many cases of retinitis (particularly in retinitis albuminurica). 4. Hypertrophy of the supporting tissue, which becomes the more prominent in proportion as the inflammation passes over into atrophy. 5. Thickening of the walls (sclerosis) of the blood-vessels, leading to the contraction of their lumen or even to their obliteration. 6. The migration of pigment cells into the retina. These are ordinarily regarded as derivatives of the pigment epithelium from which they emigrate into the retina, where they may undergo spontaneous multiplication. But according to Krückmann they are glia cells of the retina itself, which have simply taken up pigment from the cells of the pigment epithelium (Fig. 259).

When, after protracted inflammation, the retina has become perfectly atrophic, it consists of a reticulum which is derived from the supporting tissue and which contains pigment cells, but from which the nervous elements have disappeared without leaving a trace of their presence. The blood-vessels are in great part obliterated and converted into solid strands of connective tissue.

Many pathological changes in the retina do not originate in it, but are derived from an affection of the chorioid, which contributes so greatly to the nourishment of the retina. If we divide the ciliary vessels in rabbits and thus interfere with the

circulation in the chorioid, degeneration of the retina ensues, with migration of pigment into the latter (Wagenmann). [So, too, chorioidal exudates are associated with inflammation and adhesion of the overlying retina.—D.]

IV. Detachment of the Retina

530. Objective Signs.—Detachment of the retina (*ablatio* sive *amotio retinæ*) is diagnosticated by means of the ophthalmoscope, which shows the detached retina under the guise of a delicate gray membrane that rises above the level of the normal fundus and projects forward into the vitreous (Fig. 260). Exteriorly the eye looks normal, only the anterior chamber is often strikingly deep and the tension is also diminished.

Fig. 260.—Serous Detachment of the Retina in Myopia.

A woman of sixty-two, having previously been very myopic, had for four years suffered from a cataract in the right eye. After the removal of the cataract by operation, the lower half of the retina proved to be detached, thrown into folds, and tremulous. The upper border of the detached retina lay upon the lower border of the papilla, and concealed it. To the outer side the detachment is sharply demarcated from the normal fundus, while to the inner side it spreads out quite gradually into two or three flat folds. On the crests of the folds the detached retina looks lighter than in the depressions between them. The retinal vessels running downward from the papilla soon after they start disappear behind the overhanging edge of the detached portion of the retina, and are apparently interrupted at this spot. In their subsequent course they are distinguished by the remarkably sharp bends they make and which follow the folds of the detached retina. The outer side of the optic disk is bordered by a white atrophic crescent, which is about half the width of the papilla, and is attributable to the myopia pre-existing in the eye. The outlines both of this papilla and the crescent are hazy. The rest of the fundus is tessellated—i. e., displays the chorioidal vessels and the dark intervascular spaces.

The detached postions of the retina, being pushed forward, exhibit a lower degree of refraction than the rest of the fundus; they are, in fact, generally very *hypermetropic*. On account of this difference of refraction one cannot, using the erect image, see the detached and the adjoining portions of the retina distinctly at the same time; it being possible to do this only by means of the indirect method. In order to examine with the erect image, we had best hold the mirror some distance off when looking into the eye; and at the same time we may place a convex lens (e. g., one of + 3 D) behind the mirror. If the retina is pushed very far forward it can actually be seen by lateral

illumination when the pupil is dilated; a gray membrane, with the characteristic retinal vessels, being recognized deep down in the eye.

The *ophthalmoscopic appearance* of a detachment differs according as the case is one of serous detachment or of detachment due to a tumor or a cysticercus.

In *serous* detachment of the retina from the chorioid, the pigment epithelium remains in its place upon the latter. The retina, therefore, is at first transparent, but very quickly becomes cloudy, because it is separated from the chorioid, which in great part provides for its nutrition. The detached retina, accordingly, has a light, rather transparent gray color and a dull luster. If some blood is mixed with the subretinal serum, the detachment acquires a greenish tinge. The retina lies in folds of greater or less size, whose tops show a whitish sheen; and it shakes all over when the eye is moved. The gray hue and dull luster, the folds, and the tremulousness of the detached retina justify the comparison made between it and a gray silk or satin fabric.

A thing that especially characterizes a detached retina is the appearance presented by the blood-vessels that run over it. Inasmuch as these follow the folds of the retina, they are very tortuous, and some of their bends are entirely concealed between the folds. The blood-vessels are dark red, indeed almost black, as though the blood circulating in them had been altered in character. This, however, is not the case, the dark color being really due to the fact that the blood-vessels are in part at least seen by transmitted light, since some light always passes through the detached retina and is then reflected from the more posteriorly placed chorioid. The blood-vessels, consequently, look dark for the same reason that opacities in the media appear black.

The sides of the detachment may merge by a gradual slope into the surrounding retina, or they may be baggy and overhanging. In extensive detachments the papilla is partly or wholly concealed by the overhanging retina. Very flat detachments are rather hard to diagnosticate. In this case the red hue of the fundus shows a slight gray cloudiness in the parts affected, and is traversed by low folds of a rather lighter gray; but the main thing that enables the diagnosis of detachment to be made is the unusual tortuosity and the dark color of the vessels. When there is a flat detachment in the region of the macula, we can sometimes see at a point corresponding to the macula a pale-red spot in the detached retina.

The detached retina sometimes exhibits white patches, extravasations of blood, or pigmented spots. Particularly often there is found a rent in it (*ruptura retinæ*). The rent lies generally in the periphery of the fundus, and most frequently in its upper part. The edges of the tear gape and are frequently everted, so that we can see between them for quite a long distance the more posteriorly placed chorioid. The latter owing to its vivid red color forms a marked contrast with the gray of the detached retina. According to Leber, rupture of the retina plays an important part in producing detachment in myopic eyes. For in myopic eyes delicate membranes develop in the anterior portion of the retina and on its interior surface and by the subsequent shrinking of these membranes traction is made on the retina. This throws the retina into folds, and may even, because of the delicate structure of this membrane rupture it, whereupon liquefied vitreous passes through the rupture and beneath the retina. This accounts not only for the detachment itself, but also for its sudden development.

For detachment due to *tumor*, see pages 474 and 596.

In *cysticercus subretinalis* a rounded, rather sharply circumscribed detachment is found, beneath which may be recognized the bluish-gray cysticercus bladder with its lighter colored margin (Fig. 261). The detached retina is not tremulous, but spontaneous movements may be made out through it taking place in the bladder. [*Other entozoa*, found very rarely in the retina, are the echinococcus, filaria, and larvæ of diptera (Leber).—D.]

531. Symptoms.—The subjective symptoms of detachment of the retina consist in the disturbance of vision that it causes. This is characterized above all by a limitation of the field of vision, which is often perceived as a positive phenomenon by the patient. A dark cloud lies over a part of the field of vision, corresponding in location to the detached portion of the retina, which has partially or entirely lost its sensitiveness to light. If, as is so frequently the case, the detachment lies below, the patient complains of a dark curtain which veils from him the upper part of objects. For instance, he does not see the head of a man standing in front of him. Hence,

FIG. 261.—CYSTICERCUS SUBRETINALIS IN A WOMAN TWENTY-SIX YEARS OF AGE, WHO HAD NOTICED A SUDDEN DIMINUTION IN HER SIGHT TWO MONTHS PREVIOUS.

The papilla is encompassed on its outer and lower side by an irregular crescent. The region of the posterior pole of the eye is occupied by a bladder-like detachment of the retina. This detachment has a faint-gray hue, and allows the red of the fundus to appear through it, although dully. The edge of the bladder shows a light-gray, silky luster, while in the center of the bladder a bright yellowish-white speck is visible, which corresponds to the animal's head. The retinal vessels run up upon the bladder without showing any marked bend as they pass its edges. The bladder displays active spontaneous movements, in which the central white speck changes its position, shape, and size. Between the papilla and the inner edge of the bladder is an irregular, light-colored speck in the retina lying beneath a small retinal vessel. The upper part of the fundus is of a uniform red, the lower is somewhat albinotic, so that the dark chorioidal vessels stand out distinctly upon the bright-red background.

the examination of the field of vision is of great importance for the diagnosis of detachment of the retina (see also page 593). Direct vision is preserved as long as the detachment has not yet extended to the site of the macula lutea.

In the beginning of a detachment of the retina, objects frequently appear crooked (*metamorphopsia*), in consequence of the oblique position of the percipient retinal elements. [See page 125.] Photopsia, too, is often caused by the traction upon the retina, and often gives the first warning of the advent of the detachment.

532. Course.—The detachment of the retina is at first partial—i. e., is confined to one portion of the retina. It may develop at any spot whatever of the retina, but most often at its upper part and usually (in case it is

caused by fluid) changes its place afterward, for, as the subretinal fluid sinks on account of its weight, it depresses the detachment gradually to the lower part of the eye; hence detachments of the retina are most frequently found low down, although their original situation very often was at some other spot in the fundus.

Every detachment of the retina has a tendency to enlarge and finally becomes total. In the latter case we find the retina pushed forward en masse, and connected with its bed at two points only—at the papilla and at the ora serrata. Then the detached retina forms a plaited funnel, beginning at the papilla and opening out in front, a shape which Arlt has well compared to that of the flower of a convolvulus (see Figs. 176, 207 and 208).

In its later stages a detachment of the retina, whatever its origin, is frequently made inaccessible to observation with the ophthalmoscope, owing to turbidity of the media, especially of the lens and vitreous, and hence the diagnosis is rendered difficult or impossible. In such cases the diagnosis must be based upon two factors—upon the *field of vision* and the *intra-ocular tension*. If qualitative vision is lost on account of the turbidity of the media, the field of vision must be tested with the candle flame in a darkened room (see page 115). In detachment of the retina a corresponding limitation of the field will then be found. The intra-ocular tension in detachment of the retina is generally reduced, because the volume of vitreous is diminished; for the same reason the anterior chamber is often found to be deepened, because the lens has sunk backward. When in advanced cases of irido-cyclitis, irido-chorioiditis, or complicated cataract we find limitation of the visual field and reduction of the tension, we are warned that complete blindness due to total detachment of the retina and subsequent atrophy of the eyeball is imminent.

In that form of detachment of the retina which is produced by active propulsion of the retina away from the chorioid (see page 595) the tension is not diminished, but rather is increased. Hence, increase of tension with detachment of the retina is in doubtful cases an argument that an intra-ocular tumor is the cause of the detachment (Von Graefe).

The retina, when but recently separated, retains for some time its sensitiveness to light, and, if it soon becomes re-attached, may resume its function perfectly. Thus there is a possibility afforded of a *cure of the detachment* in respect to function as well as in other regards. Moreover, it sometimes happens that the sight improves very much, although the detachment remains and is not diminished in size. Such an apparent cure is brought about, whenever the detachment, after occupying the site of the macula lutea, afterward settles lower down, so that the macula resumes its function, and nothing is left but a peripheral contraction of the visual field, that causes but little disturbance. But a real spontaneous cure of retinal detachment by *re-attachment* of the retina does occur. This, to be sure, is very rare. Moreover it is imperfect in this regard; first, that the attachment is ordinarily not complete and, second, that the attached portion of the retina is so greatly damaged as to have very little functional power. [Re-attachment is more likely to take place in the comparatively infrequent cases due to active propulsion by exudation or hæmorrhage (see page 595). A cure in the sense of re-attachment occurs in about 8.5 per cent of the cases, but restoration of even moderately useful vision occurs in from 3 to 6 per cent only (Leber).—D.] As a rule a re-attachment can readily be made out with the ophthalmoscope. The fundus in the region concerned is spotted, as in old chorioiditis, owing to partial disappearance of the pigment epithelium, and is quite frequently traversed by rectilinear striæ, which are either pure white

or lined with pigment, and which lie back of the retinal vessels (retinal strands, *retinitis striata*). The whole affected area is demarcated from the remaining, normal fundus by a sharp, usually curved line, which is yellowish or gray and is encompassed by pigment.

After a detachment has lasted some time, the retina becomes entirely atrophic. It has then lost its sensitiveness to light, and, moreover, becomes again transparent. At the same time the recognition of the detachment by means of the ophthalmoscope becomes more difficult, being now made mainly from the anomalous characters presented by the vessels.

533. Etiology.—The retina simply lies upon the chorioid without being connected with it anywhere except at the papilla and the ora serrata. In the dissected eye it can be lifted from its bed with the greatest ease. In the living eye the retina is kept pressed against the chorioid by the vitreous. A detachment of the retina, therefore, is possible only when either the pressure exerted by the vitreous ceases to act, or when the retina is pushed from its bed by a force greater than this pressure.

(*a*) The former variety of detachment occurs when through *disease of the vitreous* the pressure exerted by it is diminished or becomes absolutely negative—i. e., is converted into a traction. This occurs: 1. When a pretty large quantity of vitreous has escaped, in the case of injuries or of operations. 2. When shrinking of the vitreous is produced on account of some disease of it. The most frequent cases of this sort are those in which the formation of exudates has taken place in the vitreous in irido-cyclitis or irido-chorioiditis. When these exudates become organized and shrink they draw the retina, to whose surface they are attached in places, away from the chorioid. This form of detachment, it is true, cannot be seen with the ophthalmoscope, inasmuch as the media are to cloudy, but can be readily diagnosticated by means of the softening of the eyeball and the contraction of the field of vision.

When the retina is detached from the chorioid by alterations taking place in the vitreous, fluid transuded from the chorioidal vessels collects between the retina and the chorioid, owing to the negative pressure set up beneath the retina by reason of its detachment. This subretinal fluid is a quite albuminous, usually rather yellow serum, and hence detachments of the retina of this sort are called *serous*.

In high myopia a serous detachment of the retina, which can be seen with the ophthalmoscope (Fig. 260), often occurs without there having been any antecedent inflammation. It constitutes by far the greatest of the dangers that threaten very near-sighted eyes. The way in which the detachment is produced has not yet been altogether cleared up. Most probably in this case we have to do with the result of changes in the vitreous. Such changes, consisting of floating opacities and liquefaction, can actually be made out with the ophthalmoscope in the myopic eye. The same is conjecturally true of the so-called senile detachment of the retina, which sometimes occurs in elderly people without any special cause

and which should probably be attributed to senile changes taking place in the vitreous.

(*b*) Much less frequent are those cases in which the separation takes place in consequence of an *active propulsion* of the retina away from the chorioid. The causes of such a propulsion are: 1. An acute process of exudation from the chorioid, as occurs in purulent chorioiditis and in phlegmons in the orbit. 2. Hæmorrhage from the chorioidal vessels, whether spontaneous or due to injury. 3. Tumors of the chorioid or of the retina, and also a cysticercus developing beneath the retina.

Among the causes of detachment of the retina must be mentioned *scars* that remain after perforating wounds in the region of the sclera. Such scars may be produced by accidental traumatism or by operation (page 350). They attach the retina to the chorioid and the sclera, and by their subsequent contraction cause a strain upon the retina that leads to its detachment from its bed.

534. Treatment.—Treatment in serous detachment of the retina must seek to secure the absorption of the subretinal fluid. This may be accomplished by purely medicinal means, i. e., by diaphoresis, by the use of purgatives or preparations of iodine, and by subconjunctival injections of salt solution mercury oxycyanide, or dionin (see page 66); also, in case it is tolerated by the eye, by a pressure bandage applied with moderate firmness. Of operative procedures, may be mentioned the superficial cauterization of the sclera with the galvanocautery, after the sclera at the site of the detachment has been exposed by dissecting up the conjunctiva; also puncture of the sclera (see § 880) for withdrawing the subretinal fluid directly. We undertake puncture of the sclera when the other measures have failed us, or when at the start we are dealing with a baggy detachment produced by a large amount of fluid. The puncture is made at the spot where the detachment is most pronounced, for which purpose the site and extent of the detachment must have been precisely determined beforehand with the aid of the ophthalmoscope. Only as much fluid is allowed to escape as will flow off spontaneously. Puncture, may if necessary be frequently repeated. [Puncture may be combined with cauterization of the sclera or the puncture may be made with the galvanocautery. Recently good results have been had by combining puncture with a trephining by Elliot's method, (Curtin) —D.]

In every form of treatment the patient should keep to his bed for several weeks, since the detachment is increased in size by movements of the body. Hence it is that patients with detachment of the retina generally state that they see best in the morning after prolonged rest in bed, and that their sight gets worse again as the day passes. [Occasionally rest in bed, alone, will effect a cure. It is most serviceable in recent cases when the detachment is still in the upper part of the retina. Later, when the detachment has settled it is useless (Leber).—D.]

By these methods of treatment it is generally possible in recent and not too excessive cases of separation of the retina to obtain an improvement of the sight by partial attachment of the retina, and in especially favorable cases even to cause the detachment to disappear completely. Unfortunately, it is only in the rarest cases that these good results are lasting. As a rule, after some time the separation develops anew, and ultimately in spite of all our therapeutic endeavors becomes total, so that the prognosis of retinal detachment in general must be characterized as very unfavorable. The cause of the recurrences lies in the fact that no treatment is able to do away with the lesion which usually lies at the bottom of the trouble—namely, the altered state of the vitreous. In inveterate cases or in total detachment of the retina, we had better abstain from any form of treatment. In total detachment of the retina, cataract usually develops later on, the eye becomes soft, and a slight degree of atrophy of the eyeball supervenes. Moreover, iritis is not infrequent in eyes with detachment of the retina.

When the retina is detached by means of a neoplasm, enucleation of the eye must be performed. A cysticercus occurring beneath the retina, may be extracted by an incision into the sclera, and the eye may thus be preserved in a condition serviceable for vision.

[Deutschmann has reported remarkably good results from division of the vitreous bands by a double-edged knife, or, when this fails, by the injection of an artificial vitreous. Birch-Hirschfeld withdraws the subretinal fluid and injects this (pure or mixed with salt solution) into the vitreous.—D.]

[Patients in whom a one-sided detachment has already occurred or in whom, because of their high myopia, detachment may occur at any time, should be warned to abstain from near work or straining eye work in general and to avoid excessive physical exertion.—D.]

V. Tumors of the Retina

535. Glioma.—Glioma is [almost] the only neoplasm which occurs in the retina. It is found only in children. In a case of it the parents notice that a bright, whitish, or golden-yellow reflex emanates from the pupil, which sometimes even is noticeable at a distance. For this reason the disease since the time of Beer has been known as *amaurotic cat's eye*; amaurotic, because the eye is blind; and cat's eye, because it shines like cats' eyes in the dark. If such an eye is examined by focal illumination, we recognize as the cause of the reflex a light-colored nodular mass (the degenerated retina) situated behind the lens and covered over with minute vessels.

The *subsequent course* of the glioma shows the same stages that we have learned to recognize in the case of chorioidal tumors (see page 474). In the first stage, inflammatory symptoms are absent; the disease manifests itself only by the light colored reflex and the blinding of the eye. The second stage is characterized by the development of increase of tension. The eye becomes irritated and painful, and the child begins to suffer. [Occasionally

in this stage, the eye instead of becoming glaucomatous, shrinks and takes on temporarily the characters of phthisis bulbi (cf. page 476).—D.] Afterward, in the third stage, the tumor grows out from the eye, first of all along the optic nerve, then in other places as well, particularly through the cornea or in its vicinity. The eye at length is transformed into a large, ulcerated, painful, and readily bleeding mass [exophthalmia fungosa], which fills the whole orbit and projects out between the lids. In the fourth stage the tumor spreads to remote organs. Through transfer by continuity it passes along the optic nerve to the brain; and by way of metastasis it spreads to the neighboring lymphatic glands and also to the most various internal organs (most frequently to the liver). [According to Leber, metastases are uncommon so long as the glioma remains confined to the eye, and when they do occur later usually occur in the bones of the skull or face—rarely in the liver.—D.] The children finally die either from exhaustion or from the spread of the neoplasm to vital organs, especially the brain. The course of the disease from its very outset to its fatal termination usually extends over several years.

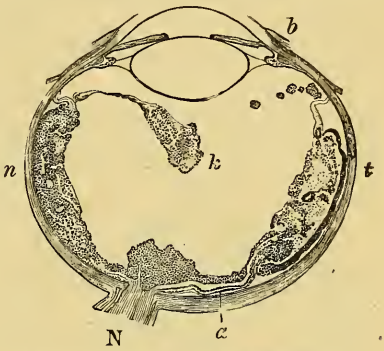


Fig. 262.—Glioma of the Retina. Magnified 2 × 1.

The glioma has spread over the entire extent of the retina. The latter on its nasal side, *n*, has been completely consumed in the formation of the new growth, while on the temporal side, *t*, the external retinal layers are still in places preserved (at *a*). The pseudoplasm also covers the optic papilla, *N*, into the excavation of which it penetrates. In the anterior segment of the vitreous lie isolated nodules, the largest of which, *k*, is connected with the ora serrata. The anterior segment of the eyeball shows the effects of the increase of tension; that is, shows on the nasal side the apposition of the root of the iris to the sclero-corneal junction, while at the point corresponding to this on the temporal side the precursor of a staphyloma intercalare can be perceived under the form of an excavation, *b*.

Glioma, as a rule, attacks only one eye, although numerous bilateral cases have also been seen. It is found in children only, and mostly before the fifth year of life. [Most cases develop before the age of three and probably over half before the age of two. Less than 2 per cent have been observed after the age of ten, and the oldest authentic case known was between fifteen and sixteen.—D.] Often it is observed at such an early age that its beginning must be dated back to fetal life. This, as well as the fact that several children in the same family are one after another affected with glioma, would argue that the cause of it is in many cases to be looked for in a congenital vice of development.

Treatment consists in the promptest possible removal of the neoplasm. So long as the growth is still confined to the eyeball, it is sufficient to enucleate the latter, in doing which we take care to divide the optic nerve as far back as possible. In such cases we may hope for a permanent cure. When the tumor has perforated the eyeball and is growing outside of it, but is still

confined to the orbit, complete removal of the neoplasm can still be attained through exenteration of the orbit (see § 897). But in this case rapid recurrences both in loco and in the neighboring lymphatic glands seldom fail to occur. Nevertheless, even in such cases the operation is indicated, because by the removal of the local focus of disease the child is spared much suffering.

From what has been said, the *prognosis* is favorable only when the operation is performed very early. [Almost 40 to 50 per cent of cases operted on early are cured (Leber). Three cases of spontaneous recovery by permanent shriveling have been reported. In inoperable cases, actinotherapy with X-ray or radium may help the process or relieve pain, and in a bilateral case, has caused disappearance of the tumor in one eye, after enucleation of the other (Axenfeld).—D.]

Glioma retinæ (Virchow), in contradistinction to intra-ocular sarcomata, is never pigmented. It probably develops from remains of the embryonic retina, that have not been differentiated into distinct layers. The tumor is composed of small cells and a very soft basement substance (Fig. 263). The cells consist of a nucleus surrounded by a very scanty amount of protoplasm which in many spots possesses minute processes.

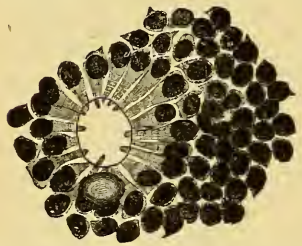


Fig. 263.—Glioma Retinæ. (After Wintersteiner.) Magnified 500 × 1.

The tumor consists of cells which have a single nucleus, and surrounding the latter a very thin, often scarcely visible, protoplasmic body. The left half of the drawing is occupied by a structure which resembles the cross section of a tubular gland. Surrounding its lumen are long cylindrical cells, whose nuclei lie at their peripheral extremities (rod and cone granules). The centrally directed extremities of the cells are bounded by a clear-cut line, the membrana limitans externa. From this latter short conical processes of protoplasm (rudimentary rods and cones) project into the lumen of the gland-like structure. Right below the latter lies a rather large, elliptical, very lustrous body—one of those hyaline concretions that sometimes occur in gliomata.

In many cases we find cells having the shape of cylindrical epithelial cells, arranged about an open space, so that the whole is like the cross section of a gland tubule (rosettes of Wintersteiner; Fig. 263). The multiplication of the tumor cells takes place mainly in the immediate vicinity of the numerous wide blood-vessels. Here, therefore, are situated the youngest cells, which keep pushing the older ones further and further away from the nutrient vessels. The older cell-layers consequently undergo necrosis. Thus in many cases the structure of the tumor becomes tubular, the vessels being surrounded by envelopes of living cells and lying with these cells in the midst of a necrotic mass. The process of overgrowth of the retina leads to its irregular thickening, and consequently to folding and detachment of it; but in many cases, as Fig. 262 shows, the detachment may for a long time remain confined to a small circumscribed spot. Neoplasm germs pass from the degenerated retina both into the chorioid and into the vitreous, where they subsequently develop into small independent nodules (*k*, Fig. 262).

That a congenital morbid disposition very often lies at the bottom of glioma is shown by the following interesting observation which I once made: A mother brought her four-year-old son into the clinic with a glioma of the right eye. According to her statement, this had existed for only a year, but it was already far advanced; the eyeball, as a whole, was very much enlarged, and the pseudoplasm was growing out from it into the orbit. The entire orbital contents were removed, but nevertheless the child died half a year afterward with brain symptoms, while at the same time a new tumor

could be felt in the orbit. Some months afterward the mother brought the next child, a two-year-old boy, with the statement that he had been blind in the right eye since birth, although it was only recently that she had noticed an enlargement of the eye. This child likewise had a glioma of the right eye, and also died of a recurrence about a year after the operation had been performed. Soon afterward the woman brought me her last child, then only a few months old, full of fear lest this one, too, might be the victim of the same frightful disease, because she had noticed in its left eye an appearance varying from the usual. This child, however, did not have a glioma, but a typical, congenital coloboma of the iris downward as well as a coloboma of the chorioid.

The features of amaurotic cat's eye may be produced not only by glioma, but also by exudate into the cavity of the vitreous [suppurative chorioiditis (page 468), retinitis exudativa (page 583), massive tubercle of the chorioid, also by primary atrophy of the retina, persistent hyaloid artery and a congenital anomaly in which layers of connective tissue cover the posterior surface of the lens (Leber).—D.] Such cases are often hard to distinguish from glioma, and are hence designated by the name of pseudoglioma.

536. [**Other Tumors of the Retina.**—Carcinoma and sarcoma (primary and secondary) have been described as occurring, though very rarely, in the retina. Tumor-like products of inflammation (cysts and granulomata) also occur. For the so-called angioma of the retina, see page 583.—D.]

537. Injuries of the Retina.—*Ruptures* of the retina occur in consequence of contusions of the eyeball, even without perforation of the remaining tunics of the eyeball; but these cases of isolated laceration of the retina are extremely rare. The retina is much harder to tear than the chorioid, since in ruptures of the latter the retina is generally found to be uninjured. Less infrequent are the spontaneous ruptures of the retina in detachment of the latter.

A transient alteration of the retina after contusion of the eyeball is the *commotio retinæ*, described by Berlin. This is characterized by a milk-white cloudiness of the retina, which occupies either the vicinity of the papilla or that part of the retina which corresponds to the point at which the effect of the blow was felt. In many cases also the spot diametrically opposite is found to be clouded. At the same time a moderate reduction of central vision and often also a contraction of the field of vision are present. The clouding of the retina disappears after some days, and with it also disappears the disturbance of vision that is produced. The condition, according to Berlin, is an œdema of the retina; according to Leber, it is a stretching of the retina with minute lacerations.

538. [**Congenital Anomalies of the Retina.**—Congenital aplasia or absence of the retinal vessels (with aplasia of the optic disk) has been observed in anencephalia and microphthalmus, and as a very rare anomaly in eyes that exteriorly appear normal. Other anomalies are sac-like protrusions of the retina at the macula and coloboma of the retina (see page 481). For medullated nerve fibers (which are not strictly a congenital anomaly) see page 565.)—D.]

CHAPTER XI

DISEASES OF THE OPTIC NERVE

Anatomy

539. The optic nerve (nervus opticus) collects its fibers from the retina, and passes from the eye through the orbit and through the optic foramen into the cavity of the skull. Hence, three divisions are distinguished in the optic nerve: (*a*) The intra-ocular termination, which is found within the sclera; (*b*) the orbital portion from the eyeball to the optic foramen; and (*c*) the intra-cranial portion from the optic foramen to the chiasm.

540. Intra-ocular Portion.—To get from the retina to the exterior of the eye, the optic nerve must pierce the chorioid and sclera. The spot where this takes place lies a little to the inner side of the posterior pole of the eye (Fig. 144). The opening in the sclera through which the optic nerve leaves the eye is called the *foramen scleræ*, and really consists of a short canal (sclerotico-chorioidal canal); the segment of the optic nerve lodged in this is its intra-scleral portion. Accurately speaking, a complete aperture for the optic nerve exists neither in the sclera nor in the chorioid, but the two membranes conduct themselves as follows: The external lamellæ of the sclera, which occupy about two-thirds of its thickness, are not perforated by the optic nerve at all, but are reflected backward upon it to form its exterior sheath (Fig. 264, *D*). The innermost lamellæ of the sclera, on the contrary, stretch over the foramen scleræ, and are perforated by numerous

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Exflanation of Fig. 264.—Longitudinal Section Through the Head of the Optic Nerve. Magnified 60 × 1.

In its passage through the sclerotico-chorioidal canal the optic nerve shows an irregular conical contraction. The fibers of the nerve are collected into bundles, *n*, separated by septa, *s*. Under the form of rows of nuclei, which belong to the neuroglia cells, the continuation of the septa can be followed to the head of the optic nerve. The axis of the nerve is occupied by the central vein, *v*, and the central artery, *a*, which is situated to the nasal side of the vein. The optic nerve is traversed transversely by the lamina cribrosa, which separates the trunk from the head of the nerve. The fibers of the lamina cribrosa arise from the wall of the scleral canal, traverse the nerve in a slightly concave arch (the concavity being directed to the front), and are inserted into the connective tissue that accompanies the central vessels. About at the level of the inner layers of the chorioid, the nerve fibers diverge like a sheaf, so as to form a funnel-shaped depression—the vascular funnel, *G*. More fibers pass to the nasal than to the temporal side of the papilla, for which reason the former side is the higher. The fibers of the optic nerve pass over into the fiber layer (*1*), of the retina. Succeeding this are the other layers of the retina, namely the layer of ganglion cells (*2*), the inner granulated or plexiform layer (*3*), the layer of inner granules or bipolar cells (*4*), the outer granulated or plexiform layer (*5*), the layer of outer granules or of the bodies of the visual cells (*6*), the limitans externa (*7*), and the layer of rods and cones (*8*). The layers of the retina stop short at the head of the optic nerve, the outermost layer, *8*, extending the furthest in. The innermost fibers of the sclera, which form the wall of the scleral canal, accompany the optic nerve backward and form its pial sheath, *P*, which is in intimate relation with it. At a point further back from the nerve-head the outer layers of the sclera are reflected backwards and form the dural sheath, *D*, which envelops the nerve loosely. Between these two sheaths lies a third, the arachnoid sheath, *A*, which divides the intervaginal space of the optic nerve into the subdural space, *sd*, and the subarachnoid space, *sa*. Anteriorly both end by a cul-de-sac in the substance of the sclera. *b* is the cross section of one of the numerous subarachnoid trabeculæ which connect the arachnoid to the pial sheath. In the wall of the scleral canal is seen the cross section of some blood-vessels, belonging to Zinn's scleral circle. Between the sclera, *S*, and the retina, *R*, lies the chorioid, *Ch*. The innermost layer of the latter, the lamina vitrea, *10*, is the one that extends the furthest in toward the nerve-head, and the fibers of the nerve are constricted by the edge of the lamina. Upon the lamina vitrea lies the pigment epithelium, *9*, which belongs to the retina

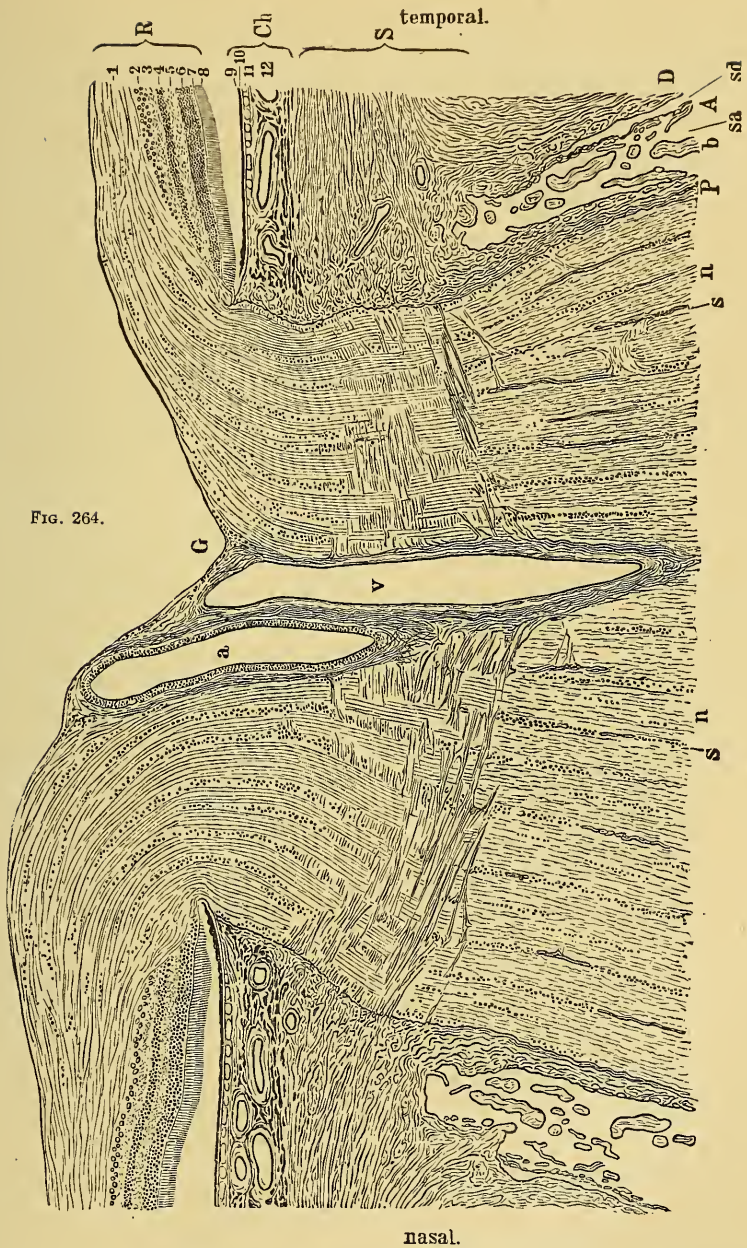


FIG. 264.

and which on the nasal side extends as far as the lamina vitrea, but on the temporal side stops somewhat short of it. On both sides the pigment epithelium gets to be thicker and more pigmented toward its edge—a state of things which answers to the chorioidal ring that can be seen with the ophthalmoscope. The succeeding layers of the chorioid, the chorio-capillaris, *11*, and the layer of medium and large sized vessels, *12*, do not extend quite up to the optic nerve on the temporal side, because a layer of connective tissue representing a continuation of the sclera juts in between the two.

openings designed for the passage of the separate funiculi of the optic nerve. In a similar fashion, a few fibrous bands from the chorioid are also continued over the foramen scleræ. Within the latter, therefore, the optic nerve is traversed by numerous septa of tough connective tissue. These form the *lamina cribrosa*, which bridges over the foramen scleræ, and is so called because it is everywhere pierced by funiculi of the optic nerve. (Seen in sagittal or longitudinal section in Fig. 264, in frontal or cross section in Fig. 267.)

If we look at the optic-nerve entrance in longitudinal section, we see that at its point of entrance into the sclera it is narrowed down in conical shape (Figs. 264, 265), so that the spot corresponding to the lamina cribrosa is the slenderest portion of the optic nerve. This narrowing of the optic nerve appears still more marked when we consider that at the site of the lamina cribrosa the connective-tissue septa are particularly numerous and of large size. The space left for the nervous constituents of the optic nerve is hence very considerably reduced at this spot. (Compare Fig. 266 with Fig. 267.) How, then, is it possible that the bundles of nerve fibers can go through this narrow passage? A glance at the longitudinal section of a fresh optic nerve gives the explanation of this. Such a section shows the nerve to be white as far as the lamina cribrosa, while in front of it it is of a translucent gray. The white hue of the optic nerve in its extra-ocular portion depends upon the fact that the optic-nerve fibers here are medullated, and therefore opaque (Fig. 265). In their passage through the lamina cribrosa the nerve fibers lose their medulla, and consequently become transparent; hence, the translucent, gray appearance of the head of the optic nerve. With the loss of the medulla, the diameter of each individual nerve fiber diminishes very considerably, so that their aggregate bulk finds room in the narrow foramina of the lamina cribrosa.

The lamina cribrosa plays an important part in *pathological* processes. In the first place, it is the weakest spot of all the tunics of the eye, being here constituted only by the innermost layers of the sclera (together with a few fibers of the chorioid), which, moreover, are perforated by the foramina for the bundles of fibers of the optic nerve. Hence, in case of increase of tension, this spot is the first to give way. In the normal eye the lamina cribrosa runs straight, or with but a slight backward curvature, across the optic nerve. With increase of tension, it recedes more and more, and thus forms the glaucomatous excavation. A second reason for the production of pathological changes lies in the fact that within the foramen scleræ, and particularly within the limits of the lamina cribrosa, the optic nerve is tightly inclosed between firm, fibrous walls, a thing that occurs at no other spot in its course. Here, therefore, when swelling of the optic nerve takes place, constriction and strangulation of it may readily occur. The foramen scleræ, accordingly, in this case play a part like that which the fibrous ring of the hernial orifice does for the viscera lying in front of it.

That portion of the optic nerve situated in front of the lamina cribrosa, in the interior of the eye itself, is the *head of the optic nerve* (papilla nervi optici). It is the part of the optic nerve which even during life can be seen by means of the ophthalmoscope. The name papilla was selected by the older

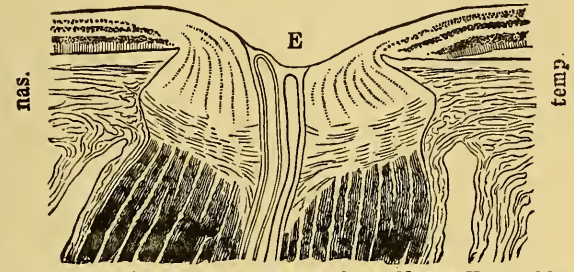
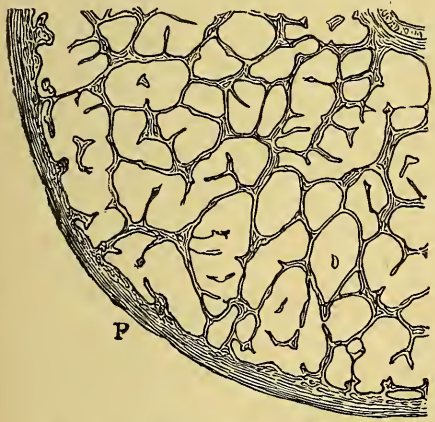


Fig. 265.—Longitudinal Section through the Optic Nerve Head. Magnified 20 × 3.

The nerve fibers of the trunk of the nerve, whenever they are medullated, are stained black by Weigert's hæmatoxylin stain. The medullary coating extends as far as the lamina. Proceeding from behind forward, the sclerotico-chorioidal canal shows first a dilatation, then a contraction. *E*, physiological excavation.

Fig. 266. a b



b a Fig. 267.

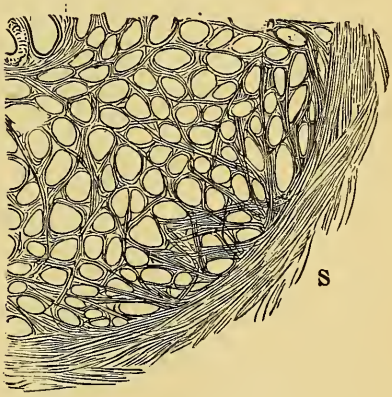


Fig. 266.—Frontal Section through the Trunk of the Optic Nerve behind the Lamina Cribrosa. Magnified 50 × 1. The septa start from the pial sheath, *P*. They traverse the optic nerve branching repeatedly and forming numerous interconnections, and join with the connective tissue, *a* which surrounds the central vessel, *b*.

Fig. 267.—Frontal Section through the same Nerve at the Level of the Lamina Cribrosa. Magnified 50 × 1. The septa, *S*, starting from the wall of the scleral canal are compact and broad and run, forming a dense plexus, through the entire optic nerve as far as the connective-tissue investment, *a*, of the central vessels. In the vicinity of the latter the gaps between the septa are larger than at the periphery of the nerve, because in the former spot the section falls somewhat in front of the densest portion of the lamina. For, since the lamina as a whole curves slightly backwards, it can never completely coincide with the plane of the section.

authors under the erroneous impression that the head of the optic nerve represented a projection into the interior of the eye. This, however, is the case only in pathological conditions—e. g., in inflammatory swelling of the papilla. In the normal state, the latter is perfectly flat, so as to lie in the same plane as the retina, or it actually has a central depression (*G*, Fig. 264). The way in which this latter is produced is that the fibers of the optic nerve begin to separate from each other, not at the level of the retina but

below it, so that a funnel-shaped depression is produced from which emerge the central vessels of the optic nerve. This is the normal vascular funnel, which quite often expands into a pretty extensive depression, the physiological excavation [see page 485].

The *fibers of the optic nerve* upon entering the interior of the eye spread out like a sheaf to form the most internal (most anterior) layer of the retina. The fibers which are situated along the margin of the papilla terminate in the vicinity of the latter. The nearer the fibers lie to the axis of the optic nerve the greater is the distance that they have to traverse in the retina in order to reach the level of the next layer of the retina—the ganglion-cell layer—with which they are continuous. Accordingly the following may be enunciated as representing the state of the case: Those fibers which come from the peripheral portions of the retina lie in the center of the optic nerve, while those which rise from the central regions of the retina lie along the margin of the nerve.

541. Orbital Portion.—The optic nerve on its way from the eye to the foramen opticum makes a bend like an italic *f* (*O*, Fig. 144). Owing to this, the eyeball can move freely within wide limits. For the movements of the eye take place about a center of rotation which lies nearly in the center of the eyeball. Hence, when the cornea is turned toward one side, the posterior pole of the eye goes about as far to the opposite side. And so for all excursions of the cornea there are corresponding ones, as large in extent but in the opposite direction, of the posterior pole, for which reason the latter must be freely movable. If now the optic nerve were stretched in a straight line between the eyeball and the optic foramen, it would keep the posterior segment of the eyeball fast in its place, and hinder the movements of the whole eye. We see a confirmation of this in those cases in which the optic nerve is put on the stretch by protrusion of the eyeball from the orbit. The more pronounced the exophthalmus, the greater is the restriction of motility of the eyeball. In the normal state, the optic nerve, on account of its S-shaped curvature, is longer than the distance between the eye and the optic foramen, so that by straightening out it can follow the changes of place of the posterior pole of the eye.

The orbital portion of the optic nerve consists of the trunk of the nerve and the sheaths enveloping it.

(*a*) The *trunk* of the optic nerve is composed of nerve fibers and connective tissue. The nerve fibers vary greatly in caliber and are extremely numerous, their amount being estimated at half a million or more. Most of them are centripetal, but there are also some centrifugal fibers in the optic nerve. Lying between the fibers as a supporting and insulating substance is the neuroglia tissue. The nerve fibers are combined into bundles (*n*, Fig. 264) which run parallel to one another, and anastomose by a mutual interchange of fibers. Between the bundles lies the connective tissue which furnishes the supporting framework for the entire optic nerve. It forms thick or thin septa which are everywhere connected and traverse the entire

optic nerve (Fig. 264, *s*; Fig. 266). Between the outer surface of a nerve bundle and the inner surface of the septa is found a space which acts as a lymph cavity.

(*b*) The *sheaths* of the optic nerve are three—an interior, a middle, and an exterior one. As these originate from the three enveloping membranes of the brain, they are designated by the names of the pial, arachnoid, and dural sheaths (Axel Key and Retzius). The inner or pial sheath (*P*, Figs. 264 and 266) closely embraces the trunk of the optic nerve. From it the bands of connective tissue, which form the septa, pass into the interior of

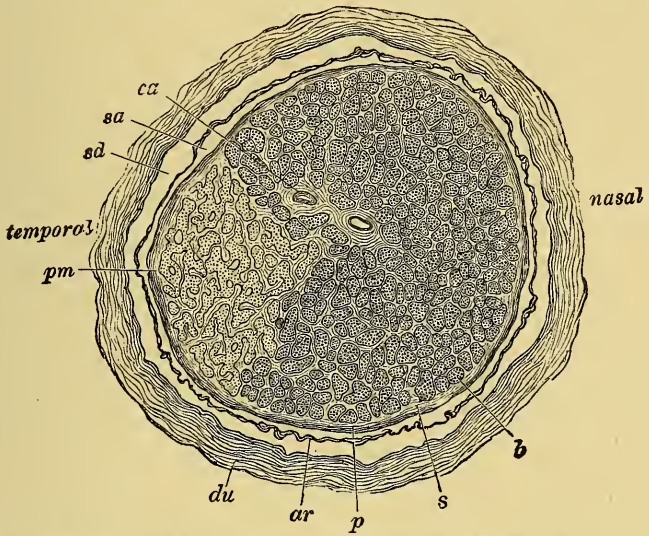


FIG. 268.—CROSS SECTION OF THE OPTIC NERVE, WITH ATROPHY OF THE PAPILLO-MACULAR BUNDLE (SECTION MADE 4 MM. BEHIND THE EYEBALL). Magnified 15 × 1.

The optic nerve is enveloped in the dural sheath, *du*, the arachnoid sheath, *ar*, and the pial sheath, *p*. Between the first and second is found the subdural space, *sd;* between the second and third, the subarachnoid space, *sa*. On the outer and upper side of the center of the section is seen the central artery, *ca;* and more centrally is seen the central vein. These are surrounded by the cross sections of the nerve bundles, *b*, which are separated from each other by the septa, *s*, of connective tissue. At the temporal side, a wedge-shaped segment, *pm*, is distinguished from the rest of the cross section of the nerve by its paler color. This represents the atrophic papillo-macular bundle. Within the confines of it the cross sections of the nerve bundles are narrower, and the septa of connective tissue are correspondingly broader.

the nerve; and with them pass the blood-vessels. The exterior or dural sheath (*D*, Fig. 264 and *du*, Fig. 268) is much thicker than the interior sheath, and surrounds the nerve loosely. By reason of this, a pretty broad space—the intervaginal space—is left between it and the internal sheath. The middle or arachnoid sheath (*A*, Fig. 264 and *ar*, Fig. 268) is a very delicate pellicle which for the most part is intimately attached to the dural sheath. It is united by manifold trabeculæ of connective tissue to the external and internal sheaths. It divides the intervaginal space into two portions, the subdural (*sd*) and subarachnoid (*sa*) spaces, which communicate with the cerebral spaces of the same names. These appear particularly prominent in Fig. 276, where they are pathologically dilated by an accumu-

lation of fluid. The surfaces of the sheaths that are turned toward these spaces are provided with an endothelial coating, so that these spaces are lined completely with endothelium, and must be regarded as lymph channels (Schwalbe).

Upon the eyeball the three sheaths become united to the sclera. The exterior and middle sheaths pass into the outer two-thirds of the sclera; the inner sheath goes to the innermost lamellæ of the sclera which form the lamina cribrosa, and it is also connected with the chorioid. The intervaginal space ends by a cul-de-sac situated within the sclera. Posteriorly the three sheaths are continuous with the corresponding membranes of the brain.

The *blood-vessels* pass from the pial sheath into the optic nerve. In addition, in the anterior portion of the orbital division there are found the central vessels of the optic nerve. The central artery is a branch of the ophthalmic artery; the central vein empties into the superior ophthalmic vein or directly into the cavernous sinus. Both vessels enter the optic nerve on its lower and inner side at a distance of 10 to 20 mm. behind the eyeball (Fig. 144, *e*) and run in the axis of the nerve as far as the papilla, where they divide into the retinal vessels.

Those fibers which supply the retina from the macula lutea to the papilla (the so-called *papillo-macular region*) take on a special form of arrangement. In that division of the optic nerve which adjoins the eyeball, they are aggregated in the form of a sector whose apex is directed toward the center of the nerve, while its base corresponds to the outer margin of the latter (the paler-looking bundle, *pm*, in Fig. 268). Farther back the arrangement changes, so that these fibers get to lie in the axis of the nerve. The sector occupied by the papillo-macular bundle amounts to about one-third of the entire cross section of the optic nerve. This is very large, when we consider that the corresponding region of the retina constitutes but a small fraction of the entire retinal surface (being commensurate with the central scotoma shown in Fig. 278). This relation, in fact, is in harmony with the predominant importance of this region of the retina. It gives support to the hypothesis that each one of the terminal elements in the macula lutea is connected with the brain by a nerve fiber of its own, so that excitations of this element are conveyed to the brain, isolated from those of other elements, while in the peripheral portions of the retina probably a number of terminal elements are continuous with one fiber.

542. Intra-cranial Portion.—The optic nerve leaves the orbit through the optic foramen. The latter really forms a short bony canal (canalis opticus), which contains besides the optic nerve only the ophthalmic artery (lying on the inner side of the optic nerve). Owing to the fact that the optic nerve within the canalis opticus is tightly inclosed by the bony walls of the latter, this section, just like the intra-scleral portion of the optic nerve, has a particular predisposition for morbid affections. Such affections consist in inflammation, in compression of the nerve through thickening of the bone, and in its contusion and laceration in case of fracture of the bony wall of the canal.

The intra-cranial portion of the optic nerve extends from the optic foramen to the chiasm; is therefore short (scarcely one centimetre). It is flattened and is enveloped only by the pial sheath, since the other two sheaths after passing through the optic foramen become united with the two outer membranes of the brain.

543. Continuation of Fibers to Cortex.—The two optic nerves join together in the chiasm, where they form an intimate anastomosis, and then on the posterior side of the chiasm make their appearance again as the optic tracts. The chiasm lies in the optic groove of the body of the sphenoid bone, directly in front of the infundibulum and above the hypophysis. Starting from the chiasm the optic tracts pass backward, diverging as they go, and, winding about the crus cerebri, arrive at the primary subcortical optic centers. The most important of these are the external geniculate body, the anterior corpus quadrigeminum, and the thalamus opticus. From these centers fibers pass to the various parts of the brain, and of these tracts of fibers two are of particular importance—the fibers (Fig. 269, *m*) which go to the nuclei of the oculomotorius, *K*, and the fibers, *S*, which pass to the cerebral cortex, *B*. The former regulate the movements of the ocular muscles and the reflex action of the pupils; the latter effect the perception of the object seen. The fibers of the optic tract which are destined for the cerebral cortex terminate in the cortical ganglion cells, within a district which is known as the optical area of the cortex, or the *visual sphere* (Munk), and which corresponds mainly to the parts surrounding the calcarine fissure. Within the ganglion cells the excitation set up in the optic-nerve fibers is transformed into sensation (sensory perception), so that it is here that the object seen comes within the domain of consciousness. In the ganglion cells which have once been subjected to excitation permanent changes remain (memories), which become so intense, particularly upon a pretty frequent repetition of the same excitation, that by means of them we are able to reproduce in our consciousness an object formerly seen (optical memory-pictures). Upon destruction of the percipient center in the occipital cortex excitations of the optic-nerve fibers fail to reach the consciousness at all. Upon destruction of the area devoted to optical memory they no longer evoke the recollection of anything already known, because the optic memory-pictures are demolished. Objects in that case are seen, to be sure, but are not recognized (*psychic blindness*).

[Wilbrand, Henschen, and others think that each portion of the retina and particularly the central portion (macula) has a definite, quite localized representation in the occipital cortex. To account for the fact that in complete hemiopia macular vision is often preserved (see page 614), Wilbrand believes that the macula has a representation in both occipital lobes. This view that the calcarine cortex is a projection of the retina is rejected by Monakow and others, who assert that the macula finds its representation in the whole occipital cortex. On the whole the balance of recent evidence, especially as afforded by very circumscribed occipital lesions, is in favor of the theory of Wilbrand.—D.]

[The process by which the two distinct visual impressions conveyed from the right and left eyes to the cortex are merged into a single sensory impression is called fusion (§ 649). Barchold thinks that the *fusion center* by which this process is performed

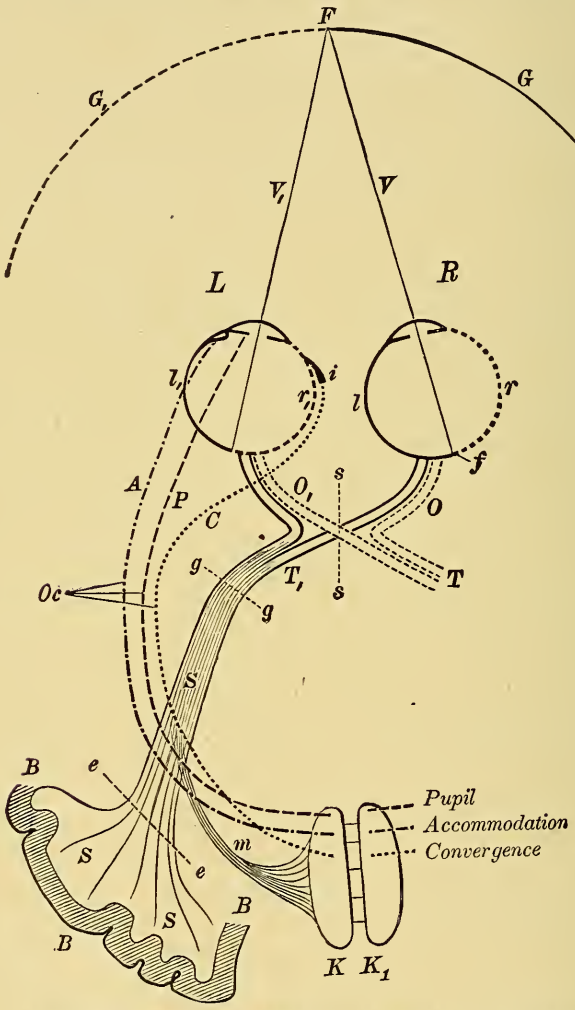


FIG. 269.—SCHEMATIC REPRESENTATION OF THE OPTIC PATHWAY.

The field of vision common to the two eyes is composed of a right half, *G*, and a left half, G_1. The former corresponds to the left halves, *l* and l_1, of the two retinæ, the latter to the right halves, *r* and r_1. The boundary between the two halves of the retina is formed by the vertical meridian. This passes through the fovea centralis, *f*, in which the visual line drawn from the point of fixation, *F*, impinges upon the retina. The optic-nerve fibers arising from the right halves, *r* and r_1, of the two retinæ (indicated by the dotted lines) all pass into the right optic tract, *T*, while the fibers belonging to the left halves, *l* and l_1, of the two retinæ pass into the left optic tract, T_1. The fibers of each optic tract for the most part pass to the cortex of the occipital lobe, *B*, forming Gratiolet's optic radiation, *S;* the smaller portion of them, *m*, goes to the oculo-motor nucleus, *K*. This consists of a series of partial nuclei, the most anterior of which sends fibers, *P*, to the pupil (sphincter iridis); the next one sends fibers, *A*, to the muscle of accommodation; and the third sends fibers, *C*, to the converging muscle (internal rectus, *i*). All three bundles of fibers run to the eye in the trunk of the oculo-motor nerve, *Oc*. Division of the optic tract at *g g* or at *e e* produces right hemiopia; and in the former case there would be no reaction to light on illuminating the left half of either retina. Division of the chiasm at *s s* produces temporal hemiopia. Division of the fibers, *m*, abolishes the reaction of the pupil to light, but leaves the sight and also the associated contraction of the pupil in accommodation and convergence unaffected.

is in the occipital lobe, the interconnection between the two centers being made by the posterior fibers of the corpus callosum.—D.]

[Psychic blindness (mind-blindness, object-blindness) includes *word-blindness, letter-blindness,* and *number-blindness,* or inability to recognize or learn printed or written words, letters, or numbers. It or any of its sub-varieties may be congenital.—D.]

544. Decussation of Optic Nerves.—We have still to study more exactly the course of the fibers of the optic nerve in the *chiasm* itself. It is not a complete but only a partial decussation (*semi-decussation*) of the fibers that takes place here. In order to understand the arrangement of the fibers it is best to start from the eyeball in making our examination. Let us suppose a vertical plane (*V*, Fig. 269) to be drawn through the retina and the visual line of the right eye. This will pass through the fovea centralis, *f*, and will divide the retina into two halves, a right or temporal half (*r*) and a left or nasal half (*l*). The fibers (indicated by dotted lines in the figure) which spring from the right half pass backward into the optic nerve (*O*), and go, keeping all the while upon the right side, to the right optic tract (*T*). The sum of these fibers is hence known as the non-decussating bundle. But the fibers which proceed from the left half (*l*) of the retina of the right eye pass over to the left side in the chiasm, so as to be found in the left optic tract (T_1). They form the decussating bundle. The like is true of the fibers belonging to the left eye. They all lie together in the left optic nerve (O_1), and become separated in the chiasm; the fibers coming from the left half of the retina pass into the left optic tract, those from the right half of the retina into the right optic tract. Each optic tract therefore contains fibers from both eyes. The right optic tract consists of the non-decussating fibers from the right half of the retina of the right eye and the decussating fibers from the right half of the retina of the left eye. Accordingly, the right halves of both retinæ (*r* and r_1), and thus the left halves of both visual fields (G_1), belong to the right tract. Hence, the perception of all objects situated to the left of the median line is conveyed along the right optic tract to the cortex of the right hemisphere; the latter is thus designed for the apprehension of the left half of the external world. The converse is true of the left hemisphere. Thus the nerve subserving the sense of sight is in harmony with other nerves, all of which terminate in the hemisphere of the opposite side. This is the case both with centripetal and centrifugal nerves. What we touch with our left hand becomes an object of consciousness through excitation of the cortex of the right side of the cerebrum; and destruction of a certain portion of the latter entails a loss of the voluntary movements of the left arm. The sense of sight appears to form an exception to this rule, since each eye is directly connected with both hemispheres. This exception ceases to exist if we distribute the visual sensations in accordance with the halves of the field of vision to which they correspond. *Everything which the*

observer sees on the left side of him becomes an object of consciousness through excitation of the right occipital cortex, and vice versa.

[It is evident from the above statement that the right eye is not governed by the left brain in the sense that the right hand is. The visual apparatus corresponding to the left brain is a forked sensorio-motor organ, consisting of: (1) the right half of the cornea and lens in the two eyes forming the two ends of the fork. These collectively are the true sensory organs and may be called the right lens-terminal. (2) The left half of the retina in both right and left eyes. This double organ, being an outlying portion of the left brain (see page 396), may be called the left brain-terminal. (3) The crossed bundle of the right and the uncrossed bundle of the left optic nerve. (4) Left optic tract. (5) Lower and (6) higher optic centers in left cortex. (7) Efferent fibers from 5 and 6 to (8) motor centers in left cortex. (9) Fibers from 8 to (10) nerve nuclei of right side (motor decussation). (11) Efferent fibers from 10 to muscles attached to lens terminal (1). The right eye, then is not a visual representative of the left brain, but is composed of the fused representatives of both brains. A man who is left-brained and consequently right-handed is not "right-eyed." At most he is "right-seeing," i. e., sees and sights objects on the right rather better than on the left, but even this is largely neutralized by the fact that, owing to the bicerebral representation of the macula direct vision is quite alike in the two eyes.—D.]

In the lower vertebrates a complete decussation of the optic nerves exists, but in the higher vertebrates there is only a partial decussation, and Gudden has the credit of having definitely demonstrated by his experiments the precise state of the case in them. He employed for this purpose the method of an artificially induced atrophy. If the right eye is enucleated in a new-born puppy, and some time afterward the animal is killed and examined, the right optic nerve is found to be completely atrophied; it consists of a thin strand of connective tissue without a trace of nerve fibers. If complete decussation of the optic nerves took place in the chiasm, this complete atrophy would necessarily be continued into the optic tract lying on the opposite or left side, while the optic tract of the right side would be perfectly intact. This, however, is not the case; in the left optic tract there is still a slender bundle of nerve fibers which has escaped atrophy. This can originate only from the optic nerve, and must, accordingly be a non-decussating bundle. So also in the apparently normal right optic tract there is found a thin bundle of atrophic fibers which must spring from the right optic nerve, and which correspond to the non-decussating bundle of the right side. Hence, in the dog a semi-decussation does exist, although the decussating is much more pronounced than the non-decussating bundle. In rabbits this disproportion is still more marked. In them the non-decussating bundle is so attenuated that at first it escaped Gudden's notice altogether. On the other hand, in man the non-decussating bundle approximates the decussating one in size, the former containing about two-fifths, the latter three-fifths of all the optic-nerve fibers. In default of experiments, accident rendered the determination of this relation possible in man. Opportunities were had of holding autopsies on men of advanced age who had lost one eye in childhood. In such subjects it was found that the complete atrophy of the single optic nerve was distributed between the two optic tracts in such a way that the tract of the opposite side was always somewhat more atrophied than that of the same side (Fig. 270).[1] Hence the following statement may be made of the facts of the case: *In the lower vertebrates complete decussation of the optic nerve takes place; in many of the higher vertebrates a partial decussation exists, the partial character of which is the more pronounced the nearer akin the animal is to man.*

[1] [It is interesting to learn that an observation of this sort in a case of ascending degeneration, after phthisis bulbi, led Vesalius to argue against the existence of a complete decussation in man.—D.]

We comprehend the reason of the foregoing fact if we start from the law that the optical perception of all objects which are situated on the right side of the body is effected by means of the left cerebral hemisphere, and vice versa. In the lower vertebrates, and in fact even in most birds and mammalia, the eyes are placed so far on one side of the head that the animal is unable to see any point whatever with both eyes at once. The fields of vision of the two eyes are perfectly distinct. The right eye sees nothing but those objects which are situated on the animal's right side; accordingly, the fibers of the optic nerve originating from this eye must all pass to the left hemisphere, for which reason complete decussation of the optic nerve takes place. In the higher vertebrates—e. g., in the dog—we begin to find the eyes placed farther forward. Objects straight in front of them situated in and close to the median line can therefore be seen by both eyes at once, so that in this locality the fields of vision of the two eyes partially overlap, and there exists a small common (binocular) field of vision.

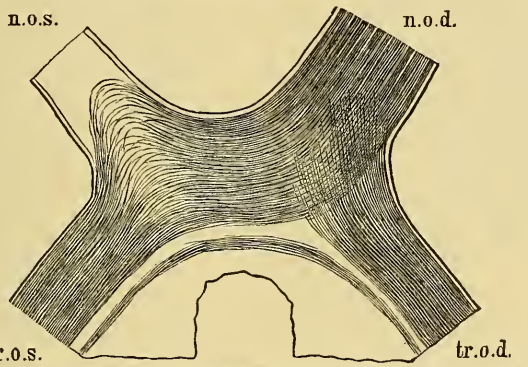


Fig. 270.—Chiasm in Atrophy of the Left Optic Nerve. Magnified 3 × 1.

The specimen is from a man, sixty-six years of age, whose left eye had been blind from childhood because of a total staphyloma of the cornea, due probably to blennorrhœa neonatorum. The cut represents a section passing through the chiasm in the horizontal plane, and stained with hæmatoxylin by Weigert's method, so that the normal (medullated) nerve fibers look black, while the atrophic fibers are unstained. The left optic nerve, *n. o. s.*, is completely atrophic, being both unstained and also considerably narrower than the right, *n. o. d.* The greater part of the fibers composing the latter pass transversely through the chiasm into the left tract, *tr. o. s.* On their way they make a loop-like bend into the left optic nerve. The smaller portion of the fibers of the right optic nerve remain on the right side of the chiasm, and pass over into the right optic tract, *tr. o. d.* Since the non-decussating fibers are less numerous than the decussating, the right tract appears somewhat narrower in cross section than the left. The bundle of nerve-fibers running in a curve along the posterior border of the chiasm, and separated from it by a light-colored zone, is Gudden's commissure (or commissura inferior) which contains no optic-nerve fibers.

The right eye, to be sure, is mainly designed for the vision of objects situated on the right side of the body, but it also, by means of the extreme temporal portion of its retina, presides over a small area to the left of the median line. The optic-nerve fibers springing from this portion of the retina must go to the cortex of the right cerebral hemisphere, because they transmit the impression of objects lying upon the left side of the body. These fibers constitute the non-decussating bundle; and this latter is small because the area of retina corresponding to it is of but slight extent.

Lastly, in man both eyes lie in the frontal plane, so that almost all objects are seen with both at the same time. Accordingly, the visual fields of the two eyes are to a large extent coincident, so that there is formed a large binocular field of vision (the area left white in Fig. 271). Each eye sees objects both upon the right and upon the left side of the body, for which reason one part of the fibers of the optic nerve belonging to it go to the right, another part to the left hemisphere. To be sure, the visual field of each eye extends farther to the temporal than to the nasal side (see page 119 and Fig. 35). Hence, it follows that the nasal portion of the retina is larger than the tem-

poral, and as the fibers which spring from the former pass over in the chiasm to the opposite side, the number of decussating fibers in the optic nerve is necessarily somewhat larger even in man than the number of those which do not decussate.

The way in which the optic nerves decussate, therefore, depends upon the relation of the fields of vision of the two eyes. If the two fields are completely separated, total decussation exists. If there is a binocular field of vision, semi-decussation takes place, and this is the more pronounced the larger the binocular field of vision.

[Animals that have their eyes placed on the side of the head, so that they have no binocular field of vision, may be said to have *lateral vision*, as distinguished from the *frontal vision* enjoyed by man. Some birds are able to move their eyes in such a way as to have either lateral or frontal vision, and some have two foveæ in each eye, one for each type of vision. An animal having lateral vision moves at right angles to his line of sight and hence gets a large parallactic displacement of objects that he passes—just as a man does when looking out of the windows of a railway train at objects whirling by. This large displacement gives the animal a much better idea of the relative distance of the objects seen than it could get by frontal vision, in which the parallactic displacement of the objects toward which it is moving is comparatively slight (Trowbridge). When the animal is at rest this advantage of lateral vision disappears, and even when it is in motion the advantage is partly compensated for by the fact that the animal with frontal vision sees the same object with both eyes at once and from this gets an idea of its spatial relations (see §647).—D.]

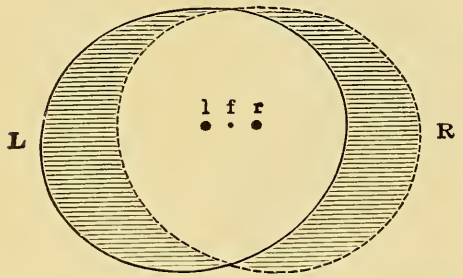


FIG. 271.—BINOCULAR FIELD OF VISION.[1] (After Baas.)

The undotted line, *L*, bounds the visual field of the left eye, the dotted line, *R*, the visual field of the right. The median portion of the two visual fields overlap to an extent shown by the surface left white. This is, accordingly, the binocular field of vision, all objects in which are seen by both eyes at the same time. In its center lies the point of fixation, *f*, and at either side of the latter the blind spots, *r*, and *l*, of the right and left eye. Adjoining either side of the binocular field of vision are the temporal divisions of the two visual fields (the shaded areas in the figure), objects in which are seen with one eye alone.

545. Hemiopia.—The fact of semi-decussation affords the explanation of an important variety of visual disturbances, hemiopia.[2] Let us suppose that the continuity of the left optic tract (Fig. 269, T_1) is interrupted at any spot—e. g., at *g g*. In this case the left halves of both retinæ (*l* and l_1) would be cut off from their connection with the cortex of the left hemisphere. The right half, *G*, of the fields of vision of the two eyes would be wanting, so that only the left halves of all objects sighted would be seen. In like manner, the left halves of both visual fields would be lost upon destruction of the right optic tract. Hemianopia thus originating is called *homonymous* or lateral (Fig. 272). It is called right-sided or left-sided according as the right or the left half of the visual field is wanting. Such a hemiopia would also, of course, occur if the destruction did not affect the optic tract itself, but a spot placed higher up (e. g., *e e*)—in fact, even the cerebral cortex itself. Homonymous hemiopia, therefore, is always indicative of a lesion which lies

[2] From ἥμισυς, half, and ὤψ, vision. By many authors the terms hemianopia or hemianopsia, formed by the interposition of an ἀ privative, are employed.

to the central side of the chiasm and upon the same side as the blind half of the retina. [But see pages 615, 616.]

If the chiasm is divided by a sagittal section (*s s*, Fig. 269) into a right and left half, all the decussating fibers are severed, while the non-decussating bundles remain intact. Since the decussating bundle supplies the inner halves (*l* and r_1) of the two retinæ these portions of the retina would be thrown out of use, and thus the outer temporal half of the two visual fields would be suppressed. This form of visual disturbance is therefore called *temporal* (or *bitemporal*) hemiopia (Fig. 273). It occurs, for instance, when

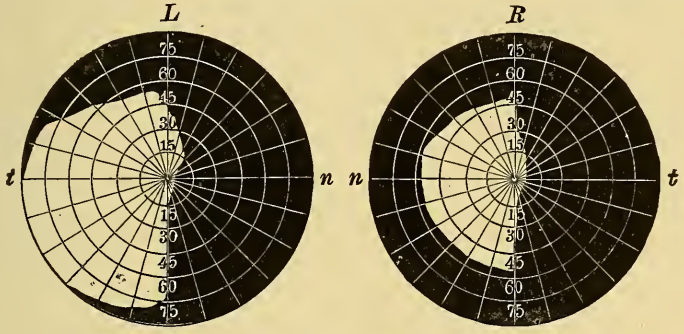


Fig. 272.—Homonymous Hemiopia. (After Schweigger.)
The areas which have been left white correspond to the left halves of the visual fields, *R* and *L*, of the right eye and left eye, which are still intact; *t*, temporal; *n*, nasal side.

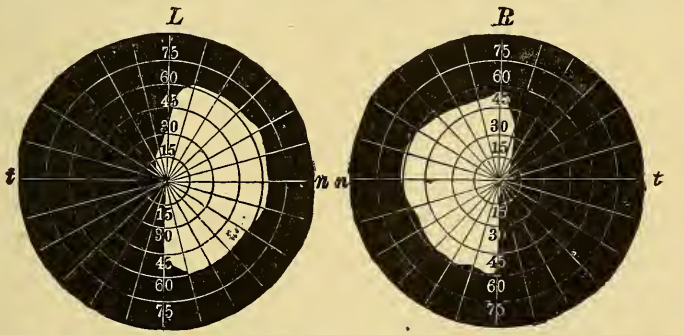


Fig. 273.—Temporal Hemiopia. (After Schweigger.)
The areas left white correspond to the nasal halves of the visual fields, *R* and *L*, of the right and left eye, which are still intact; *t*, temporal; *n*, nasal side.

as the result of an inflammation or a neoplasm the chiasm suffers from a lesion situated mainly in its mesial line. The same thing might also be caused by a lesion existing in the anterior or posterior angle of the chiasm, where decussating fibers alone are situated. [The very rare *nasal* (or *binasal*) hemiopia, in which the nasal half of each field is abolished, is caused by a lesion which involves both the right and left side of the chiasm but leaves its central portions intact.—D.]

Hemiopia in the more extended sense of the term exists not only when an entire half of each visual field is wanting, but also when there is a deficiency which, though

smaller, occupies a symmetrical position in the visual fields of the two eyes (*incomplete hemiopia*, Wilbrand). In this case, too, there is a lesion of the optic fibers above the chiasm, only now simply a portion instead of all the fibers of one tract (or of its continuation to the cortex) is destroyed. [Hemiopia may even be limited to a central or paracentral scotoma, symmetrically placed in the visual field of each eye; and such hemiopia may be central—even cortical—in origin (Wilbrand).—D.] In homonymous hemiopia it is the rule that the field of vision is not divided exactly in half, the vertical border of the field bending out a little at the site of the point of fixation (Fig. 272), so that the portion of the field of vision corresponding to the macula lutea is preserved intact [*exemption of the macula*]. Hence, if, as is very rarely the case, a bilateral hemiopia is produced by a bilateral central lesion, the combination of the visual defects on the two sides does not give complete blindness, but [usually] leaves intact right in the center a very small central visual field, representing the fovea centralis.

546. Site of Lesion Determined by Visual Defects.—Our knowledge in regard to the course of the optic fibers can be applied practically, inasmuch as it enables us to determine precisely the situation of a lesion in the optic paths. In this instance we are dealing with cases in which a defect exists in the visual field without the ophthalmoscope's showing any disease of the deep tunics of the eye, so that the defect must be referred to some break in the conduction.

In all cases in which the defect in the visual field is contained in one eye only or in which, while there are defects in both eyes, they are not symmetrically situated, the lesion must be seated in the *optic nerve* itself—that is, in front of the chiasm—since all interruptions on the farther side of the chiasm result in the production of symmetrical defects in the visual fields. For the same reason complete blindness of one eye, with retention of good sight in the other, must be referred to an affection in front of the chiasm. Central scotomata correspond to an affection of the *papillo-macular bundle*. In temporal hemiopia the lesion is seated in the *chiasm* itself, and is so placed that only the decussating fibers are affected by it. This is the case when the lesion occupies the middle of the chiasm or its anterior or posterior angle. It most frequently occurs as a result of an enlargement of the hypophysis cerebri, which, therefore, is often first discovered by the ophthalmologist. The symptoms vary, depending on whether the degeneration affects the glandular or the nervous portion of the hypophysis, whether it causes increase or diminution of function, and finally whether it begins in the growing period or not till later in life. When it occurs during the growing period, there develop disturbances of growth, viz., either gigantism or more rarely dwarfing of growth, general adiposity, and defective development of the sexual parts or of the hair (beard, pubic hair). When the disease develops later in life, the disturbance of growth manifests itself by an enlargement of the skeleton and the soft parts of the face, hands and feet (acromegaly), impotence and in women amenorrhœa, chilliness, somnolence, and polyuria. A congestive optic neuritis, which is so frequent in brain tumors, is rare in this condition. Ordinarily there is a pallor of the papilla, which does not develop until late and which results from an atrophy of the optic nerve, descending from the chiasm. To make the diagnosis certain, it is necessary to prove, by taking a radiograph of the skull, that the sella turcica is dilated by the enlarged hypophysis. [Pituitary disease is sometimes associated with] homonymous hemiopia, or less extensive but still homonymous defects in the field. But homonymous defects usually depend upon a disturbance *above the chiasm*. If then there is loss of the pupillary light reflex when light is thrown upon the blinded portion of the retina (Wernicke's hemiopic pupillary reaction [or inaction]), the break in the con-

duction must lie below the spot at which the fibers to the oculo-motor nucleus are given off—i. e., lie in the *optic tract;* but if the pupillary light reflex is intact, the lesion is to be located *higher up.* It most often lies in the back part of the internal capsule, or, still higher, in the optic fibers running from capsule to occipital cortex or in the cortex itself.[3] [The Wernicke reaction is difficult to demonstrate and even when refined methods are used, its diagnostic value is contested by competent observers (Walker and others).—D.]

I. Hyperæmia of the Optic Nerve

547. Simple hyperæmia of the optic nerve is marked by increased redness of the nerve and by haziness of its outlines, so that it contrasts but little with the red fundus surrounding it, which shows a radiate striation representing the course of the nerve fibers in the retina. To this are added dilatation and tortuosity of the retinal vessels.

A wrong diagnosis of hyperæmia of the papilla is often made if one forgets that in dark pigmented eyes the papilla looks redder and does so, too, when in the case of a faint uniform opacity of the vitreous it is seen through a turbid medium. Cf. Pseudo-neuritis (page 98).

Hyperæmia of the disk is a frequent occurrence. It is not only constant accompaniment of all inflammations of the retina and chorioid, but is found in connection with violent inflammations of the anterior division of the eye—e. g., in irido-cyclitis and even in corneal ulceration. [It also occurs in connection with refractive errors and arteriosclerosis (see page 571).—D.]

II. Inflammation of the Optic Nerve

548. Inflammation of the optic nerve (neuritis optica) may develop at any spot whatever of the nerve. Of course, it is directly visible in the living eye only when the optic papilla, which is accessible to ophthalmoscopic examination, is involved. Such cases we call neuritis intra-ocularis, or, because of the changes in the papilla, papillitis (Leber). From them are to be distinguished those cases in which the inflammation is located in a portion of the optic nerve situated farther back (neuritis retrobulbaris). Since in this case the focus of inflammation cannot be seen, its existence must be inferred from the other symptoms.

If an inflammation of the retina is associated with pretty marked involvement of the optic nerve, or if, conversely, an inflammation of the optic papilla has extended so as to occupy quite a large area of the retina, the picture of *neuro-retinitis* or papillo-retinitis is produced. Almost all forms of retinitis, as well as of neuritis described in these pages, may appear under the form of neuro-retinitis.

(*a*) *Neuritis Intra-ocularis*

549. Symptoms and Course.—Neuritis of the optic papilla manifests itself externally by no sign except that the pupils are dilated, to correspond with the diminution or absolute loss of sight. Ophthalmoscopic examination shows in the papilla evidences of inflammation (Fig. 274). The color of the papilla is altered, being either white, gray, or reddish, and it is often

[3] [It is, of course, possible for hemiopia, whether temporal, nasal, or homonymous, to be caused by symmetrical lesions affecting both optic nerves or their expansions in both retinæ (cf. page 616).—D.]

mottled with white spots or extravasations of blood (*h*). The papilla is often radially striate and its outlines become indistinguishable, the exudation extending over them into the adjoining retina; hence, too, the papilla appears of greater diameter than normal. The retinal blood-vessels are altered, the arteries (*a a*) being thinner, while the veins (*v v*) are distended, owing to compression of the vessels by the swollen optic nerve. The retinal veins are exceedingly tortuous, especially where they pass down upon the retina over the edge of the swollen papilla; and wherever their coils dip deeper into the clouded tissues they look veiled or cut off altogether. The most important symptom is the swelling of the papilla, shown by its projecting above the surrounding retina (Figs. 276 and 277).

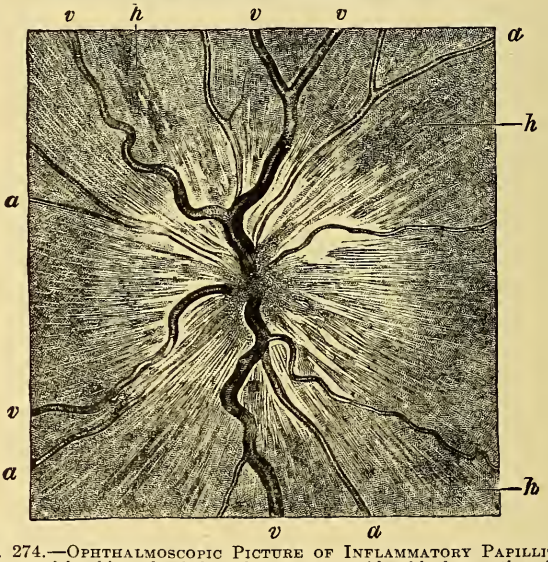


Fig. 274.—Ophthalmoscopic Picture of Inflammatory Papillitis.

The papilla is grayish-white, clouded, and appears considerably larger than it really is because it cannot be demarcated, from the surrounding retina which is likewise clouded and gray. To a diminishing extent the cloudiness and the radial striation extend out into the retina beyond. The retinal arteries, *a*, *a*, are contracted, the retinal veins, *v*, *v*, are exceedingly dilated and tortuous, and both are obscured in places. In the retina, adjoining the papilla, are found radially disposed, striate, red spots (hæmorrhages), *h*.

The subjective symptoms consist in disturbed sight. This in most cases is very great; with severe neuritis complete blindness is usually present. Nevertheless, cases of marked swelling with normal sight do also occur (in choked disk). Characteristic of many cases of neuritis is a sudden and momentary obscuration of sight, repeated many times a day. Contraction of the field of vision is often found, sometimes under the form of hemiopia. [In some cases the only anomaly of the field is an enlargement of the blind spot. It has been thought that interlacing or reversed color fields (see page 645) are characteristic of neuritis due to increased intra-cranial pressure, but this is probably erroneous.—D.]

Neuritis runs a chronic course. It takes months for the inflammatory

signs to disappear, and then they are replaced by the symptoms of atrophy. The papilla grows paler, its outlines become once more clearly visible, and the vessels upon the papilla and in the retina are narrowed. This atrophy (*neuritic atrophy*, as it is called) is the greater the more intense the preceding neuritis; and the degree of the atrophy determines whether the vision grows better again after the inflammation has run its course, or remains permanently enfeebled, or is annihilated altogether. In any case, the prognosis of neuritis is serious.

FIG. 275.—CHOKED DISK IN BRAIN TUMOR. (After Haab.)

The papilla is swollen out into a mushroom-like protrusion so as to appear enlarged but still well defined (in contradistinction to inflammatory papillitis, Fig. 273). The arteries are thinner than normal, the veins somewhat distended and tortuous. Two of the veins show a brief interruption inside of the papilla since here a bend of the vessel that dips down into the tissue of the papilla is concealed. Where the vessels descend over the overhanging margin of the papilla to the retina, they all display a bend or an actual interruption. The retina surrounding the papilla is the seat of a delicate radiating striation.

550. Etiology.—Like the rest of the intra-ocular affections, neuritis is but rarely a local lesion; on the contrary, it usually originates in some deep-seated affection, and for this reason is almost always bilateral in development. The diagnosis of neuritis is therefore of importance, not only for the oculist, but for every physician engaged in treating internal disorders, as it affords him aid indispensable for the diagnosis of many diseases.

551. Brain Disease Causing Optic Neuritis.—1. *Brain diseases* are by far the most frequent cause of optic neuritis. They lead to disease of the optic nerve, either through producing congestion or through transfer of

inflammation. (*a*) *Congestion* is chiefly of moment in those diseases of the brain which lead to an elevation of the pressure within the cranial cavity—i. e., most frequently in tumors of the brain and in hydrocephalus. A brain

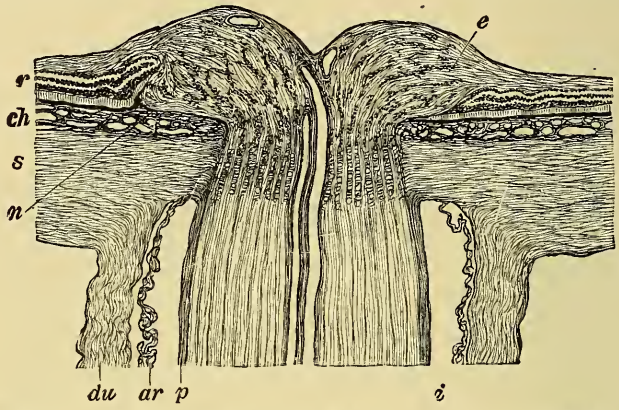


Fig. 276.—Longitudinal Section through the Head of the Optic Nerve in Papillitis (Choked Disk). Magnified 14 × 2.

The disk is greatly swollen, so as to project above the level of the adjacent retina and form at the base an annular tumefaction, the neuritic swelling, *n*. There is a cellular infiltration, particularly along the minuter blood-vessels, *e*, for which reason the latter appear specially prominent. The retina, *r*, is thrown into folds about the circumference of the papilla, in consequence of the swelling of the latter; the chorioid, *ch*, and the sclera, *s*, are normal, as is the optic nerve posterior to the lamina cribrosa. Here there is present simply a dilatation of the intervaginal space, *i*, through accumulation of fluid, by virtue of which the greatly folded arachnoid sheath, *ar*, becomes especially prominent; *du*, dural sheath; *p*, pial sheath.

tumor, as a result of its growth, arrogates constantly more and more space to itself within the cranial cavity. Hence, as the skull is unyielding, an increase in the intra-cranial pressure arises, by virtue of which a portion of the cerebrospinal fluid is squeezed out of the cranial cavity. This fluid finds an egress partly in the direction of the spinal cord, partly in that of the optic nerve. The spaces between the sheaths of the optic nerve which communicate with the lymph spaces between the membranes of the brain are dilated by an accumulation of fluid (hydrops vaginæ nervi optici, Figs. 276, *i*, and 277; Stellwag). Upon this fact is based Schmidt and Manz's theory of the origin of neuritis, which is as follows: In consequence of accumulation of fluid in the intervaginal space, a stasis of lymph occurs in the trunk of the optic nerve itself, particularly in the region of the lamina cribrosa, the lymph spaces of which communicate with the intervaginal space. The œdema

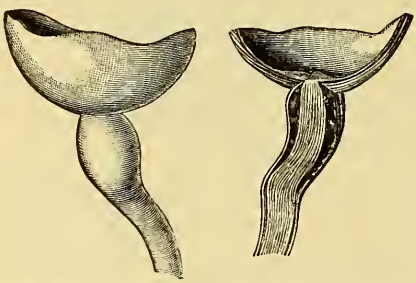


Fig. 277.—Hydrops Vaginæ Nervi Optici. (After Pagenstecher.)

Upon the left side is seen the ampulliform swelling of the optic nerve in its anterior portion. Upon the right are represented the relations as seen after making a longitudinal section of the nerve. One can see how the swelling of the nerve is caused by the distention of the outer sheath, which is separated a good way from the trunk of the nerve; and can recognize the projection of the papilla above the level of the retina.

of the optic-nerve trunk and the lamina cribrosa causes a compression of the central vessels—a compression which makes its influence felt sooner and to a higher degree in the central vein of the optic nerve than in the central artery. As there is constantly pouring into the papilla through the artery a quantity of blood which cannot be completely carried away again by the contracted central vein, venous engorgement of the optic nerve and consequently swelling of the latter are developed. This swelling of the nerve leads to its incarceration at the spot where it fits so tightly in the foramen scleræ, and consequently extreme œdema develops in the strangulated papilla. Neuritis having this origin is hence not so much an inflammation proper as an inflammatory œdema, and is accordingly designated by the name of congestive neuritis, *choked disk* [or papillœdema]. It therefore constitutes a very important symptom of increase of the cerebral pressure. (*b*) *Direct transmission* of inflammation from the brain to the optic nerve must be assumed to exist chiefly in those cases in which an inflammation is present in the brain itself, and particularly at its base, as is, for example, generally the case in tuberculous meningitis or otitic processes. Here the inflammation is transmitted along the optic nerve and its sheaths to the papilla (*neuritis descendens*).

The part that increased *intra-cranial pressure* plays in causing choked disk is evident from the fact that often the swelling of the papilla diminishes directly after the pressure has been reduced by lumbar puncture.

Von Graefe was the first to distinguish the inflammations of the optic nerve *accompanying diseases of the brain* into congestive neuritis [choked disk] and descending neuritis. The differences between the two are mainly differences with regard to the swelling of the optic nerve and to the transmission of the inflammation to the adjacent retina. The degree of swelling of the nerve can be judged of from the way in which the vessels bend as they pass over the edge of the papilla to descend upon the retina; also from the parallactic displacement that the papilla shows with reference to the retina in examinations made with the inverted image. In the erect image the degree to which the papilla projects can be calculated from the difference in refraction between it and the retina (page 112). In choked disk (Fig. 275) the swelling is so great that the vessels appear kinked or actually interrupted at the border of the papilla. The engorgement, furthermore, if great, manifests itself in the often enormous distention of the retinal veins. On the one hand, the tissue changes are limited pretty sharply to the papilla itself. In neuritis descendens the swelling of the papilla is slight; there is no distinct bending of the vessels at the border of the papilla, and frequently the difference of level can be demonstrated only through the difference in refraction shown to be present by examination with the erect image. On the other hand, the exudation made patent by the cloudiness and discoloration of the disk becomes a prominent feature. Moreover, the exudation extends beyond the edge of the disk into the adjacent retina, so that the disk appears enlarged; frequently the picture of neuro-retinitis is produced. In spite of these distinctions the two conditions are not so widely separated as theory demands, since numerous transition forms occur between choked disk and descending neuritis.

The diseases of the brain which are complicated with optic neuritis are partly focal, partly diffuse affections. Among the former, it is above all the *tumors of the*

brain which result in neuritis, usually under the form of choked disk. Neuritis in this case is so frequent—it is said to be wanting in only 10 per cent, according to others in 20 to 30 per cent of cerebral tumors—that it forms one of their most important symptoms. This sympton is the more deserving of consideration inasmuch as a cerebral tumor may often run its course for a long time without producing any other positive symptoms—e. g., it may simply cause headache, or even this may be wanting. *Accordingly, in every case in which there is a suspicion of the existence of a cerebral affection, the fundus of the eye should be examined with the ophthalmoscope.* This is the more necessary, since choked disk sometimes fails to manifest itself by any disturbance of the vision. This is explained by assuming that in choked disk—in the beginning, at least—there is simply a state of œdema. The disturbance of vision is accordingly produced by compression of the nerve fibers due to the œdematous swelling. The degree of this compression, however, cannot by any means be determined from the ophthalmoscopic appearance, so that normal sight may be present along with a neuritis which with the ophthalmoscope appears very pronounced. In many of these cases the blindness does not come on till later, sometimes not till the advent of neuritic atrophy.

With tumors of the brain a special form of *neuro-retinitis* sometimes occurs, in which, besides the appearances in the optic papilla, minute splashes of silvery luster are visible in the macula lutea, so that a picture resembling that of retinitis albuminurica is produced [retinitis stellata, see page 579].

The *size* of the new growth is not of decisive importance in determining the development of a choked disk. Choked disk has been seen with tumors which scarcely reached the size of a walnut; at other times neuritis is absent, although the tumors are very large. Evidently other factors have to be considered and especially the *site* of the tumor. Tumors in the posterior fossa of the skull (e. g., tumors of the cerebellum) are the ones most often causing choked disk. In the posterior fossa of the skull the space is hemmed in by the tentorium which stretches over it and is but slightly yielding. Thus there is readily produced compression of the aqueduct of Sylvius or of the vena magna Galeni, and consequently a stasis of the cerebrospinal fluid in the anterior ventricles of the brain (dropsy of the ventricles). On the other hand, the fact that basal tumors, e. g., those of the hypophysis, rarely produce choked disk is accounted for on the ground that such tumors may block the orifice by which the intervaginal space of the optic nerve opens into the cranial cavity, so that the cerebrospinal fluid does not enter the space.

[In the majority of cases—though not with the regularity that Horsley contends for—the swelling of the disk due to brain tumor is greater in the eye on the same side as the tumor.—D.]

Moreover, with tumors of the brain there may occur not only congestive neuritis, but also *descending neuritis* and *simple atrophy* of the optic nerve. Descending neuritis takes place when the tumor excites, in its immediate neighborhood, an inflammation which is transmitted to the optic nerve. [Tumors of the frontal lobe give a characteristic picture (Kennedy). In the first stage (which may be lacking) there is bilateral papillœdema with good vision; in the second, subsidence of the papillœdema in the eye on the side of the tumor and the development in this eye of a central scotoma and subsequent optic atrophy, while papillœdema with good vision persists in the other eye. In the third stage the second eye also develops a central scotoma and atrophy. There is loss of the sense of smell on the side of the tumor.—D.] Simple atrophy may be produced because a tumor exerts a direct pressure upon the chiasm or the optic tracts, and thus causes their effacement. An example of this is furnished by the interesting cases of tumor of the hypophysis, which, by pressing upon the chiasm causes atrophy of the optic nerves with temporal hemiopia (see page 614). In other cases an accumulation of fluid takes place in the third ventricle in consequence of the tumor, so that

the greatly distended anterior inferior extremity of the ventricle presses upon the chiasm. In this way amaurosis develops with cerebral tumors, either without any ophthalmoscopic evidence at all or under the guise of a primary atrophy.

Among the *focal affections* of the brain which may, although but rarely, cause neuritis must be enumerated foci of softening, abscesses, thrombosis of the sinuses, aneurysms, apoplexies, and cysts (among these cysticercus and echinococcus cysts). Among the *diffuse affections*, disseminated sclerosis, acute and chronic meningitis, and hydrocephalus give rise to neuritis. The two affections last named, together with tubercles of the brain, are the most frequent causes of neuritis in children. Frequently such children are not brought to the oculist until later in life, when he finds a neuritic atrophy as the cause of the blindness, and can determine from the history of the case that a severe cerebral affection has preceded it. This form of blindness is incurable. Not to be confounded with it are those rare cases in which children become blind without known cause and without any ophthalmoscopic change in the fundus. This variety of blindness, the cause of which is at present unknown, sometimes disappears (Nettleship). Some cases of neuritis due to hydrocephalus are known in which a continual dropping of fluid (cerebrospinal fluid) takes place from the nose. Neuritis also occurs sometimes in malformations of the skull (particularly the kind known as [oxycephaly or] tower skull) and injuries of the skull (especially fractures on the base, with consequent meningitis). [Penetrating or non-penetrating injuries of the skull due to projectiles are frequently followed by a choked disk, which usually affects the vision but little and is not usually followed by atrophy. It yields readily to decompression and may disappear spontaneously (Jessop, Lister).—D.]

Neuritis has also been observed as a rare complication in *spinal diseases*, particularly in acute myelitis, in tetany and in multiple neuritis.

552. Other Causes of Neuritis.—2. *Syphilis.* This is a frequent cause of neuritis. The optic nerve may be attacked by the syphilitic affection directly. In other cases it becomes affected indirectly, owing to the development in the cranial cavity or in the orbit of inflammations or of tumors which result from syphilis, and which secondarily implicate the optic nerve.

3. Acute infectious *febrile diseases*, chronic *disturbances of nutrition* of various kinds, and *poisoning* especially by lead.

The optic nerve reacts in a most sensitive way to *disturbance of nutrition* of the general organism. Among such disturbances must be classed above all the infectious diseases; and neuritis occurs, although rarely, in the acute exanthemata (measles, smallpox, scarlet fever), and also in typhus, diphtheria, pneumonia, influenza, and whooping cough. Among chronic diseases that are to be placed in the same category are albuminuria, diabetes, [tuberculosis, chlorosis,] and anæmia. [Infection originating in the teeth, tonsils, and accessory sinuses of the nose may cause optic neuritis.—D.] In the female sex there is sometimes a connection between the neuritis and the genital system, in that neuritis sets in simultaneously with disturbances of menstruation, with pregnancy, or with lactation. These cases usually give a good prognosis, even when they get to the point of producing complete blindness for the time being. Externally similar to the conditions just spoken of, but etiologically quite different, are those cases in which an amenorrhœa and a papillitis develop as symptoms running parallel with one another, both being the result of a brain disease (tumor or serous meningitis).

Of *poisoning* in the narrower sense of the term, there should be mentioned besides lead-poisoning, that produced by alcohol and iodoform [also atoxyl and other arsenical preparations. The optic neuritis occurring in syphilitics after the use of salvarsan is not to be ascribed to the toxic action of the latter, but to the liberations of spirochætæ

or their toxins. Such a so-called neuro-recidive is avoided if mercury is given with or before the salvarsan.—D.] A few cases of neuritis have been known that have undoubtedly developed in consequence of great chilling of the body. Lastly, there should be mentioned in this connection the cases of neuritis and atrophy of the optic nerve that have been produced by a stroke of lightning or electric shock.

4. Acute anæmia after great *loss of blood*, the most frequent variety being that due to hæmorrhage from the stomach and to metrorrhagia. In these cases blindness, as a rule, does not set in until some days after the hæmorrhage, and is generally incurable.

5. *Heredity*. There are families members of which are attacked by neuritis without there being any special cause for it. Such a neuritis usually affects only the male members of the family, and these are generally attacked by it at the same age (about the twentieth year). [See page 624.]

6. *Orbital affections*, such as inflammations or new growths in the orbit or tumors developing upon the optic nerve itself. These are the only cases in which neuritis can with certainty be regarded as a purely local disease.

553. Morbid Anatomy.—The following changes occur in neuritis and choked disk.

Inflammation of the optic nerve starts from its connective-tissue portions—that is, from the sheaths and the connective-tissue septa. In the sheaths there is found, besides the dropsical condition already mentioned, actual inflammation with a formation of a cellular exudate (perineuritis). Within the trunk of the optic nerve the inflammation attacks the septa which show thickening with multiplication of their nuclei (interstitial neuritis). Owing to this, the bundles of nerve fibers, which are inclosed by them, are compressed and, undergoing atrophy, are destroyed. Accordingly, in neuritis the nerve fibers act mainly a passive part.

In *choked disk* the inflammatory symptoms are limited to the papilla, while the trunk of the optic nerve back of the lamina cribrosa is fairly normal. The main feature is that the papilla is found to be greatly swollen by an accumulation of œdematous liquid, so that it projects like a mushroom into the interior of the eye, and is thickened at its base so as to form an actual tumefaction (neuritic swelling, Fig. 276, *n*). The retina is pushed aside and thrown into folds by the enlarged optic nerve. Besides the œdema there are also found extravasations of blood, swelling of the nerve fibers, and the evidences of a scanty cellular infiltration, particularly along the blood-vessels (Fig. 276, *e*). Later on, the cellular exudation becomes more and more prominent and in the subsequent course of the disease leads to a new formation of connective tissue within the papilla, due to organization of the exudate. By the subsequent shrinking of the connective tissue the fibers of the optic nerve are rendered atrophic, and the picture of neuritic atrophy of the optic nerve is produced. We then find in place of the papilla a network of connective-tissue strands, and among them blood-vessels whose walls are thickened.

554. Treatment.—Treatment must be directed first of all against the lesion which forms the basis of the neuritis. Local treatment, in addition to a suitable regimen of the eyes, consists in the abstraction of blood at the mastoid process, in diaphoretic measures, and in the administration of absorbent remedies, such as iodide of potassium, mercury, etc [and in non-specific cases the salicyl preparations (Leber).—D.] In increase of brain

pressure decompressive operations like trephining and lumbar puncture are to be considered.

Trephining produces a permanent reduction of the intra-cranial pressure in brain tumors and in hydrocephalus, and thus subsidence of the neuritis and improvement of sight. It is particularly advisable in those cases in which the brain lesion itself is curable, and hence especially in meningitis serosa. In this disease by trephining promptly it may be possible to prevent a permanent blindness being left after the main disease has been cured. [Even in inoperable brain tumors, trephining is indicated as a palliative measure for relieving pain and saving the sight. Horsley lays down the rule that no case of optic neuritis, not due to general infectious disease or toxic causes, should be allowed to go on to blindness without an operation. In pretentorial growths the site of the trephining should be temporal, in subtentorial growths should be suboccipital (Frazier cited by De Schweinitz).—D.]

(*b*) *Retrobulbar Neuritis*

555.—Retrobulbar neuritis is located in the orbital division of the optic nerve. Hence, upon ophthalmoscopic examination, we find in the papilla either no changes at all or changes that are insignificant and not characteristic; but later, after the disease has subsided, the signs of atrophy frequently make their appearance there. This is the case when destruction of the fibers of the optic nerve has taken place within the region occupied by the focus of inflammation. The peripheral portion of the divided fibers then undergo an atrophy, which is slowly transmitted to the papilla, where it becomes visible with the ophthalmoscope (descending atrophy). Owing to the absence of distinct ophthalmoscopic changes in the recent cases, the diagnosis of retrobulbar neuritis must be made from the other symptoms, and mainly, in fact, from the way in which the vision is affected. In a few cases the disturbance of vision may increase to the point of complete blindness, but in most cases is confined to the central portions of the visual field which are supplied by the papillo-macular bundle. There is, therefore, a central scotoma in the field of vision. [A central scotoma due to optic-nerve disease is distinguished from one due to central chorioiditis or retinitis by the fact that in the former there is no change in the apparent size or shape of objects in or about the scotoma (retinal metamorphopsia; see page 125) and that the first colors to disappear are red and green. In primary disease of the macula retinal metamorphopsia is usually present, and a scotoma for blue develops before that for red or green (Mauthner).—D.]

Retrobulbar neuritis is either acute or chronic in its development. One portion of the toxic amblyopias (see page 630) is thought, by many, to be a chronic retrobulbar neuritis. In general, retrobulbar neuritis affords a good prognosis, since in cases which are not too far advanced the sight can usually be brought back again to the normal.

The *acute* form of retrobulbar neuritis is characterized by the suddenness with which the disturbance of vision develops. In the severe cases this failure of sight may attain such a degree within a few days that all perception of light is abolished. Ex-

ternally, the diseased eye looks normal; the pupil, however, is often a little dilated. The ophthalmoscope, too, shows scarcely anything besides some distention of the retinal vessels.[4] These symptoms are often accompanied by violent headache or by dull pain in the orbit, the latter being aggravated if the patient moves his eye or if the attempt is made to push it back in the orbit. Sometimes both eyes are attacked by this disease at the same time.

Acute retrobulbar neuritis is either the result of an *inflammation of the surrounding orbital tissue* or there is an idiopathic inflammation of the nerve. Cases of the former kind originate most frequently in the nose in cases in which a violent coryze has developed from the effect of cold, influenza, etc. The catarrh of the nasal cavity passes to the accessory sinuses of the nose and especially to the ethmoid cells, and thence the inflammation is transmitted directly to the orbital tissue and to the optic nerve (compare § 737). As a direct proof of the presence of an inflammation of the retrobulbar cellular tissue, we not infrequently find in such cases a slight amount of exophthalmus, and also paralysis of some of the eye muscles, especially those which lie close to the inner and upper walls of the orbit (internal rectus, superior oblique, levator palpebræ, and the superior rectus). When then as so frequently happens a cold is given as the cause of the trouble, the nose is always to be examined, since upon the result of this examination will depend the treatment; and if there is a demonstrable disease of the accessory sinuses these will require to be treated and, to effect this, will often even require to be opened. In other cases cold appears to cause acute retrobulbar neuritis, as it does other peripheral neuritides, directly, i. e., without implication of the nose. An *idiopathic inflammation* of the optic nerve must also be assumed to exist in disseminated sclerosis, in which the retrobulbar neuritis often occurs as a very early symptom; also in general affections such as acute infectious diseases, toxic conditions, and various disturbances of nutrition [including tuberculosis] in which an acute retrobulbar neuritis may be found in place of a papillitis. The hereditary form of neuritis also usually appears as an acute [bilateral] retrobulbar neuritis.

In benign cases (those due to cold or to nasal affections) the disease *subsides* very quickly; the sight gets better every day, so that in from two to four weeks it is once more pretty nearly normal. In other cases the cure takes a longer time and is then ordinarily incomplete, a central scotoma remaining [especially in the hereditary form]. But in rare cases there is left a total, permanent blindness, so that it is impossible at the outset of the disease to state the prognosis with certainty.

Recurrences of the disease are sometimes observed even after the lapse of years.

The *treatment* of the disease requires above all the consideration of the causal factor (the nose). In the way of symptomatic treatment energetic diaphoresis proves particularly effective in the acute stages; in addition the forms of treatment given for papillitis (page 622) are to be considered. [The salicylates or aspirin may be given, especially when pain is present.—D.]

III. Atrophy of the Optic Nerve

556. Atrophy of the optic nerve develops either as a primary affection or as secondary to an antecedent inflammation. We accordingly distinguish between simple and inflammatory atrophy.

(*a*) *Simple* (primary or genuine or non-inflammatory) atrophy is distinguished by the papilla becoming paler, and at length perfectly white or bluish-white and also becoming sharply defined and slightly excavated (atrophic excavation; see page 486); the gray dots of the lamina cribrosa

[4] Sometimes, on the contrary, ischæmia of the retina is present when the central vessels are subjected to compression in the inflamed portion of the optic nerve.

are visible more distinctly and over a larger area; the more minute blood-vessels of the papilla itself have disappeared, while the retinal vessels are not markedly altered (in contradistinction to inflammatory atrophy, in which the latter are narrowed, too). As the atrophy increases, the sight is reduced until there is complete blindness. The *causes* of simple atrophy of the optic nerve are: 1. Primary degeneration of the optic nerve. This is found above all in tabes, which is by far the most frequent cause of the simple form of optic-nerve atrophy. In the same way, though less often, optic-nerve atrophy is associated with a condition allied to tabes, [and like it a late manifestation of syphilis] namely, progressive paralysis of the insane. Optic-nerve atrophy usually develops in the initial stage of tabes at a time when the ataxic symptoms are slight or absent, and the diagnosis of tabes is not yet readily made. It is therefore a fact of great value to us that we know two other symptoms which likewise usually make their appearance very early. One of these symptoms regards the pupil, which no longer reacts to light (Argyll-Robertson pupil, page 386), and is generally also greatly contracted (spinal miosis, page 451). The other symptom, discovered by Westphal, is the absence of the patellar reflex. [Other important signs are a positive Wassermann reaction in the blood and especially in the spinal fluid and lymphocytosis of the latter with a positive globulin reaction.—D.] Spinal atrophy of the optic nerve always affects both eyes, although not necessarily both at the same time. It advances slowly but surely until there is complete blindness, and hence has rightly earned the name of progressive atrophy. Simple atrophy of the optic nerve is, moreover, a frequent complication of disseminated sclerosis. These cases are characterized by their irregular course—at times by transient improvement of sight—and they rarely lead to complete blindness. 2. A break in the line of conduction. This may be intra-cranial in site, a tumor or other focal affection compressing the nerve itself or the chiasm within the skull. In this case the atrophy is propagated gradually from the site of the break in the line of conduction down to the intra-ocular extremity (descending atrophy). The break in the line of conduction may also evidently be located near the periphery—i. e., in the orbit where the optic nerve may be thrown into a state of atrophy by inflammations, injuries, or as a result of compression by tumors. Atrophy by a break in the line of conduction may give the same ophthalmoscopic picture as tabetic atrophy. But while in the latter the pallor of the optic nerve can be made out at the very same time that the deterioration in sight begins, in the former it occurs subsequent to the deterioration in sight, since it takes some time—several weeks, at least, and usually longer—for the degeneration of the optic-nerve fibers to travel from the point where the break is situated down to the papilla.

Simple atrophy is found most frequently in middle life. In children it almost never occurs [except in the rare infantile or juvenile tabes, in which it is an almost con-

stant and sometimes almost the only symptom (Barkan)]; the atrophy of childhood is, as a rule, neuritic. Men are more frequently attacked by simple atrophy than women, a fact which is dependent upon the greater predisposition of the male sex to spinal affections and to syphilis. In old people a low degree of non-inflammatory atrophy of the optic nerve sometimes occurs, caused by atheromatous disease of the internal carotid or of the ophthalmic artery. In this case the vessels by the pressure they cause induce a partial atrophy of the optic nerve, which for a certain part of their course they directly adjoin (Bernheimer, Sachs, Otto).

The disturbance of sight in atrophy always affects not only direct vision, but also the *visual field*, which is found to be diminished. Sector-shaped defects or concentric contraction of the visual field are the most frequent forms under which this occurs. Color blindness also sets in early: first for red and green, and last of all for blue, which color, therefore, is recognized the longest. By this circumstance atrophy is distinguished from glaucoma simplex, which sometimes shows much resemblance to it, but which is not ordinarily associated with color blindness until late in its course.

In the simple atrophy of tabes the *anatomical changes* at the outset consist of discrete foci of disease, which appear gray upon cross section and which lie in the trunk of the optic nerve. In this case we are dealing with the same gray degeneration that exists in the posterior columns of the spinal cord in tabes. The nerve fibers lose their white medullary substance and are transformed into extremely minute fibrillæ, and hence the entire tissue acquires a gray and translucent appearance. Between the remains of the nerve fibers are found cells filled with granules of fat.

In the later stages secondary changes occur consisting of a moderate thickening and sclerosis of the connective-tissue septa and multiplication of the glia nuclei; but evidences of actual inflammation are wanting during the entire course of the process. In disseminated sclerosis, on the other hand, a proliferative inflammation of the connective tissue and of the glia, occurring under the form of insular foci, appears in the optic nerve at the very outset. Later on, the proliferated tissue shrinks and the nerve fibers lose their medulla. The axis-cylinder, however, remains intact for a long time and for this reason the sight is not as a general thing completely destroyed. Hence, in tabes and progressive paralysis the primary process is seated in the nervous elements (optic-nerve fibers, ganglion cells of the retina), and in sclerosis it is located in the supporting structure of the nerve.

The anatomical condition found in descending or ascending atrophy is similar to that occurring in gray degeneration of the optic nerve. The atrophy reaches its highest degree in those cases in which the eyeball has been completely destroyed, the optic nerve in this instance shrinking in the course of time to a thin strand consisting simply of connective tissue.

557. (*b*) *Inflammatory atrophy* of the nerve is the form which occurs as the final result of a neuritis or a retinitis (neuritic or retinitic atrophy). Inflammatory atrophy is distinct from the simple variety in its ophthalmoscopic features as well as its origin, because in it the papilla is traversed by connective tissue formed by an organization of the exudate. In *neuritic* atrophy the papilla is at first of grayish-white color, and its margins are slightly hazy; the veins are very distended and tortuous. Afterward the papilla becomes of a pure white or bluish-white, but we do not see the lamina cribrosa exposed to view as in simple atrophy. The papilla is now sharply defined, but is often smaller than normal and irregular, as though it had been shrunken; both arteries and veins are contracted and are frequently

inclosed within white streaks. There is often found about the papilla an irregular decolorization of the chorioid immediately adjoining it. In *retinitic* atrophy the papilla looks clouded and of a dirty grayish-red. Its outlines are faint, and the vessels are greatly thinned and often have entirely disappeared (Fig. 258).

558. The *prognosis* of atrophy of the optic nerve is in general unfavorable. Cases of simple atrophy for the most part lead to complete blindness. Inflammatory atrophy affords a somewhat better prognosis, since the amount of sight which the neuritis or retinitis has left is usually permanently preserved. *Treatment* consists primarily in the management of the causal disease. For the lesion of the optic nerve itself, in syphilitic cases salvarsan [especially by intra-spinal injection], mercury, potassium iodide are employed; and in these and also in non-syphilitic cases injections of strychnine, and the constant [or high frequency] current applied to the eye itself; unfortunately, however, generally with but slight success. [A beginning descending atrophy due to increased intra-cranial pressure may be helped by decompression and one due to hypophysis disease may be arrested by an operation to remove the diseased pituitary gland (Cushing).—D].

Treatment with [salvarsan and] mercury is indicated in those cases only of optic-nerve atrophy of which it is assumed that they are caused directly by syphilitic disease of the optic nerve or its vicinity. [If mercury is combined with salvarsan, the development of neuro-recidives (see page 622) is prevented.] But in optic-nerve atrophy due to tabes or paresis—disorders which are in the last analysis attributable to syphilis—we often see the decline of vision go on especially fast during a course of mercurial treatment, so that in such cases we prefer to avoid mercury.

IV. Toxic Amblyopia

559. Poisons may interfere with vision in various ways, by causing either paralysis of the eye muscles, of the sphincter pupillæ, and of accommodation, or by causing disease of the light-perceiving apparatus, i. e., the retina and optic nerve. We shall speak here only of affections of the latter class which are comprised under the name of toxic amblyopias. In acute conditions of poisoning, blindness sets in either suddenly, as after large doses of quinine, filix mas, or methyl alcohol, or at least within a few days, as occurs with atoxyl; in chronic poisoning, of which that caused by nicotine and alcohol should be mentioned here as being the most frequent form, the amblyopia develops very gradually, and is limited to a central scotoma. A third group is formed by lead poisoning.

560. Acute Poisoning by Quinine and Other Substances.—1. In acute *quinine poisoning* (after doses of [less than one to] three gm. or upwards) sudden blindness sets in with hardness of hearing or complete deafness. In most cases the blindness disappears again gradually but imcompletely, amblyopia and a markedly contracted visual field remaining permanently. Even at the beginning we find with the ophthalmoscope excessive attenuation of retinal vessels, with which is afterward associated a pallor of the disk.

Anatomical investigation in quinine poisoning produced experimentally in dogs shows during the very first days a destruction of the ganglion cells of the retina (Holden, Druault), these being primarily attacked by the poison, and as a result of the death of the ganglion cells a degeneration of the nerve fibers in the optic nerve very soon sets in.

Sudden blindness also occurs after poisoning by salicyl preparations [including oil of wintergreen], filix mas, pomegranate-root bark, optochin, atoxyl, arsacetin [osmic acid, potassium chlorate, and acetanilide] (all being substances which are taken as *medicines*), and by *methyl alcohol*, which is used as an intoxicating beverage. [In this country there have been many cases of blindness from methyl-alcohol poisoning. In a few cases this has occurred from inhalation of the vapor (as in shellac workers), but generally from drinking cheap liquor, Jamaica ginger, or other liquids which have been adulterated with deodorized wood alcohol ("Columbian spirits"). Blindness is almost always complete and permanent. Acute, complete or partial blindness is also caused by inhaling the fumes of exploded dynamite and of nitrobenzol (Stephenson).—D.]

561. Chronic Poisoning by Tobacco, Alcohol, and other Substances.—2. *Nicotine* and *alcohol amblyopia* are due to chronic poisoning by these substances. Nicotine gives rise to poisoning more often than alcohol, but very frequently the two poisons act together, the person affected being at the same time a great smoker and a great drinker. The symptoms in the two cases are the same. The disturbance of vision sets in so gradually that the patients are for the most part unable to tell exactly when it begins. At first medium-sized print can still be read, afterward the reading of ordinary print becomes impossible. The reduction in the visual acuity is almost always the same in both eyes—a fact which distinguishes this from other intraocular affections, such as cataract, chorioiditis, atrophy of the optic nerve, etc., in which the two eyes are usually affected to a different degree.

The symptom of *nyctalopia* is particularly characteristic. The patient declares that he sees much better in the evening than in the daytime; indeed, in recent cases he often imagines that in the evening he still sees as well as he used to do, and that it is only in the daytime that he has a troublesome cloud that dazzles his sight. Objective examination shows that really in most cases no observable improvement takes place when the illumination is reduced; but the annoying sense of dazzling is done away with, so that the patient believes that he sees better. In some cases, however, I have been able to make out a real improvement in the sight upon diminishing the illumination. One of these patients read finer print and read it better when he put on dark-gray goggles than he could do with the naked eye. Another patient, a coachman, could still recognize in the evening the numbers of the houses to which he had to go, while in the daytime he no longer was able to do it.

Many patients also declare that they cannot recognize *red* colors, particularly of small objects, as well as formerly. Their acquaintances, they find, look ill because their cheeks appear of a waxen yellow. Innkeepers, who are particularly apt to be affected with tobacco amblyopia, complain that they can no longer distinguish between copper and nickel coins, since they no longer appreciate the difference in color.

Objective examination shows but *slight ophthalmoscopic changes*. In recent cases the papilla is usually somewhat hyperæmic; in the older cases, on the contrary, it has grown paler in its temporal half. But these changes are often so little pronounced that one may say that the result of examination is negative.

Examination of the *vision* shows a moderate diminution of the visual acuity, which has its cause in a central scotoma (Fig. 278). This scotoma forms a horizontal

oval, extending from the macula lutea to the blind spot, and corresponding, therefore, to the maculo-papillary region of the retina (Fig. 268). At first there is simply a color scotoma. No gap is found in the field of vision if it is tested by means of a white object; but a red or green mark undergoes a change of color in the region of the scotoma. It appears less highly colored than in the other portions of the field of vision, and later on appears perfectly colorless. Later still, the mark disappears altogether from view in this portion of the visual field; the scotoma has now become absolute (see page 124), and the vision has become reduced to the lowest point that it can reach in this disease. The outer limits of the visual field always remain normal, and complete blindness is therefore not to be apprehended, but direct vision is destroyed, and with it the ability to carry on any fine work. Owing to the chronic course of the disease, it takes a series of months for the sight to be reduced as low as this; and, moreover, this extreme reduction does not occur in every case.

The *cause* of tobacco amblyopia is the excessive use of tobacco, whether by smoking or chewing. The disease is hence found almost exclusively in the male sex, and in males not generally until middle life. It would thus appear as if the resistance to nicotine diminishes with age. The quantity of tobacco which is sufficient to bring on a tobacco amblyopia varies according to the susceptibility of the individual, in many cases comparatively small amounts of tobacco sufficing for this purpose. The cheap varieties, which are usually richer in nicotine, and also moist tobacco, are more dangerous than the better, dry qualities. The abuse of spirituous beverages, which, to be sure, is very usual with great smokers, favors the development of tobacco amblyopia; but amblyopia also occurs in smokers who abstain altogether from alcoholic drinks.

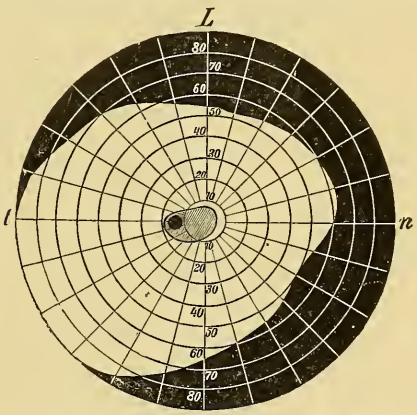


Fig. 278.—Field of Vision of the Left Eye of a Man suffering from Tobacco Amblyopia.

The visual field when tested with a white object (shown by the portion left white in the figure) is normal. When, however, the examination is made with a red object, a central scotoma is found having an extent represented by the shaded area which forms an irregular oval. The small black circle comprised in this area represents Mariotte's blind spot.

Treatment consists, above all, in abstinence from tobacco, and it is probable that in light cases alone is sufficient to effect a cure. To accelerate the cure we employ iodide of potassium internally or hypodermic injections of strychnine. [Stress has also been laid on the importance of preventing intestinal autotoxis (by suitable diet, purgation, free water-drinking, etc.). Furthermore, as stated in § 562, the urine ought to be examined in every case, and the treatment modified accordingly.—D.] In addition, we prescribe a suitable regimen for the eyes. Recent cases in which middle-sized print can still be read, and in which the scotoma has not yet become absolute, afford a good prognosis, since a perfect cure is usually obtained, although one or two months are required for it. In older cases, however, in which even quite large print can no longer be read and the scotoma is absolute, a complete cure is for the most part impossible.

Alcohol amblyopia also does not usually begin before middle life, and occurs as a rule only in spirit drinkers. It is frequently combined with alcoholic neuritis of the peripheral nerves. The treatment is the same as that of tobacco amblyopia, but the prognosis less favorable for the reason that abstinence from alcohol is much harder to secure than abstinence from tobacco.

Samelsohn was the first to discover the *anatomical changes* in nicotine and alcohol amblyopia. He showed that they were limited to the papillo-macular bundle, whose position and course within the optic nerve he was thus able to determine (page 606). In the course of this bundle it is found that the nerve fibers have disappeared and nothing but glia tissue is present, while the connective-tissue septa lying between the nerve fibers are thickened (Fig. 268). Samelsohn regarded this as the outcome of an interstitial inflammation of the optic nerve, the inflammation affecting the connective-tissue portion and especially the septa which convey the blood-vessels and which because of the inflammation become thickened. In consequence of this thickening, the optic-nerve fibers are compressed and made to atrophy. Others, however, think that what occurs is a primary lesion of the optic-nerve fibers by the poison and that if a thickening of the connective-tissue septa was found, this was a secondary change. The optic nerve would then behave in just the same way as has been shown to hold for the peripheral nerves in alcoholic neuritis. Lastly there are some who believe that even the destruction of the nerve fibers is not the primary affection, but that, in analogy with the conditions of acute poisoning (by quinine, etc.), this consists in a lesion of the ganglion cells in the retina and that an ascending atrophy develops in the nerve fibers as a secondary affair.

The same complex of symptoms as in nicotine and alcohol amblyopia occurs also in chronic poisoning due to various *other substances* which either are taken into the body for therapeutic purposes, like stramonium, chloral, iodoform, iodides, thiuret, thyreoidin, or which find their application in industries, like carbon bisulphide (in vulcanizing rubber) and aniline. [Iodoform may cause complete blindness.—D.]

3. *Lead* also causes disturbances of vision which develop in a chronic fashion. These, however, occur very exceptionally under the guise of amblyopia with central scotoma. As a rule, they appear under the form of a papillitis which not infrequently leads to complete blindness.

562. Autotoxæmic Amblyopia.—Disturbances of vision due to neuritides which are usually retrobulbar and run sometimes an acute, sometimes a chronic course, are also found in cases in which owing to anomalies of metabolism the opportunity is afforded for the production of toxic substances (*auto-intoxication*). The most frequent occurring example of such a disturbance of vision is that found in *diabetes*.

In this case we sometimes observe an amblyopia of the type of a nicotine amblyopia. This may be simply the result of the diabetes itself, but mostly it affects patients who smoke, so that the influence of nicotine cannot be surely excluded. It seems that the resistance of the optic nerve toward nicotine is diminished by the disturbance of metabolism due to the diabetes, for which reason a diabetic patient ought to be warned not to smoke much; and for a like reason the urine ought to be examined for sugar in every case of amblyopia with central scotoma, even when such amblyopia apparently comes from smoking or drinking.

Probably also referable to auto-intoxication are the cases of papillitis and retrobulbar neuritis which are found in anomalies of menstruation—during pregnancy and lactation—and in gout.

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563. Injuries of the Optic Nerve.—The optic nerve may be injured within the orbit [directly] by penetrating foreign bodies, stab wounds, shot wounds, etc. Because of this break in the line of conduction, blindness—partial or complete according to the severity of the lesion—is present immediately after the injury. With this there are, at

first no ophthalmoscopic changes demonstrable in the papilla. It is not until later, after weeks have elapsed, when the descending atrophy has traveled from the site of injury to the papilla, that the latter becomes paler and presents the picture of simple atrophy. It is only when the optic nerve is injured so far forward that the central vessels are also divided that characteristic ophthalmoscopic symptoms can be made out at once. In such cases immediately after the injury a picture develops analogous to that of embolism of the central artery. The arteries of the papilla and retina are bloodless, and the retina soon becomes clouded—a sign of its death.

Indirect injuries of the optic nerve occur not infrequently in consequence of injuries of the skull by the impact of a blunt object (a blow or fall upon the head, etc.). In such cases there is partial or complete blindness associated with the symptoms of a severe injury of the skull (the signs of concussion of the brain or of fracture of the base of the skull). Such blindness may be unilateral or bilateral. It has been demonstrated by the investigations of Holder and Berlin that in these cases we are dealing with indirect fractures of the walls, particularly of the upper wall, of the orbit, which fractures are continued into the optic canal, so that the optic nerve in the latter is crushed or lacerated. Several weeks or months afterward there is developed in the papilla the picture of simple atrophy. These cases of blindness are incurable.

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564. Tumors of the Optic Nerve.—The optic nerve may be affected with tumor formation, either primarily or secondarily. The latter most frequently happens through the growth of intra-ocular tumors, such as sarcoma of the chorioid and glioma of the retina, backward along the optic nerve. Primary tumors of the optic nerve are rare. They comprise fibromata, sarcomata with varieties (myxo-sarcoma, psammo-sarcoma, etc.) of the latter, and endotheliomata. These usually spring from the sheaths of the optic nerve, while the gliomata develop in the nerve-trunk itself. Pure neuromata, originating from the nerve fibers, have not up to the present time been observed with certainty in the optic nerve. Primary tumors of the optic nerve begin generally in youth, and grow very slowly. They cause an exophthalmus, distinguished from that occurring with other orbital tumors by the fact that the lateral displacement is either entirely absent or is at all events insignificant. The mobility of the eye remains good for a comparatively long time; on the other hand—and this is characteristic—blindness sets in very early. With the ophthalmoscope we find at first neuritis with the venous engorgement particularly marked; afterward atrophy. [In one case reported by Ellett the fundus appeared normal, although there was complete blindness. In structure the tumors vary greatly. Some may be classed as endotheliomata, others show an overgrowth of the fibrous connective-tissue elements of the nerve (fibromatosis). (Byers.)—D.] In many cases the patient shows other symptoms of neurofibromatosis (see page 499). Treatment consists in extirpating the tumor; in doing which we may, under certain circumstances, leave the eye in place. [See § 896.] Recurrences after operation are comparatively rare.

A few cases of tuberculous granulation-tumor of the optic nerve are also known.

[Hyaline bodies (so-called colloids) in the form of pearly or yellowish, rounded elevations, usually massed in mulberry-like aggregations, are sometimes found on the papilla and the adjacent retina. They were formerly confounded with druses (see page 457), from which, however, they differ in origin and structure (De Schweinitz, Leber). Amyloid bodies are also found in the same situation.—D.]

[565. Congenital Anomalies of the Optic Nerve.—They include coloboma (page 481), aplasia (page 599), pseudoneuritis (page 98), medullated nerve fibers (not strictly congen-

ital, see page 565), and the presence of shreds or larger masses of connective tissue enveloping the vessels and projecting from the disk like the masses in retinitis proliferans.—D.]

Disturbances of Vision without Apparent Lesion

566. Amblyopia and Amaurosis.—The expressions amblyopia[5] (weak sight) and amaurosis[6] (absolute blindness) are terms used to designate disturbances of vision. The former designation is now applied only to those cases in which the weakness of sight cannot be relieved by suitable glasses. For instance, a myope who sees badly with the naked eye, but possesses the full amount of visual acuity with the correcting concave glass, is not amblyopic but simply myopic. Under the name of amaurosis were formerly known those cases of blindness in which the eye had externally a normal appearance, so that this designation was equivalent to the expression "black cataract" ("schwarzer Staar"). The ophthalmoscope has thrown light upon these cases, which are for the most part referable to affections of the chorioid, the retina, and the optic nerve. At the present day the expressions cerebral amaurosis and spinal amaurosis are still used in the old sense; by these names being designated those cases in which blindness has set in as a result of diseases of the brain and spinal cord, while the external appearance of the eye is normal. But the word amaurosis is also employed at present in a wider sense as equivalent to total blindness, even when the eye shows external changes. Thus we say of an eye blinded by iridocyclitis that it is amaurotic.

Thanks to the refined methods of examination with glasses, but most of all thanks to the ophthalmoscope, it is at present possible in most cases to discover the cause of weak sight or of blindness. Nevertheless, there does remain a small number of cases in which we are unable even now to demonstrate any changes in the eye as a cause for the disturbance of vision. In some of these cases of disturbance of vision without apparent lesion, the changes are so minute that they are not discoverable by our present methods of examination. In other cases the cause of the disturbance of sight is not seated in the eye at all, but behind it, in the optic tract and even as far back as the cortex of the brain. In other cases still there are no anatomical lesions whatever, and what we have before us is simply the so-called functional affections—i. e., altered conditions of circulation and nutrition resulting in disturbance of function.

A. The Seat of the Disturbance of Sight is in the Eye Itself

567. (1) **Congenital Amblyopia.**—We assume this to exist in those cases in which, according to the account given by the patient, weak sight has existed for a long time, and in which all other causes for it can be excluded. We are justified in making this assumption whenever other congenital anomalies are also present in the amblyopic eye, such as an extreme degree of

[5] Properly blunt-sightedness from ἀμβλύς, blunt, and ὤψ, sight. [6] ἀμαυρός, dark.

hyperopia or astigmatism, coloboma of the iris or the deeper membranes, albinism, microphthalmus, etc. For experience shows that such eyes almost always display a reduction of the visual power, which cannot be brought to the normal even by the correction of the error of refraction present.

Congenital amblyopia is usually unilateral; the effected eye is then very apt to fall into a state of squint. If the amblyopia affects both eyes, nystagmus develops (see § 705).

[In view of the great frequency of retinal hæmorrhages in the new-born (see page 572), it is assumed that some of the cases of congenital amblyopia are due to a hæmorrhage of this sort which destroyed central vision and disappeared without leaving a trace of its presence.—D.]

568. (2) Amblyopia ex Anopsia.[7]—Amblyopia from non-use occurs when there has been present from earliest youth an obstacle to vision in the eye, which makes the formation of sharp images upon the retina impossible. In this category belong cases of opacities either of congenital origin or acquired early in life, situated in the cornea, lens, or in the region of the pupil (pupillary membrane). Amblyopia also develops in an eye which has squinted since childhood, because in this case the perception of the retinal images in this eye is suppressed, and the eye is thus purposely excluded from participation in the act of vision. In all these cases, the retina, owing to lack of exercise, fails to attain to that delicacy of function which belongs to normal eyes, or the functional capacity which has been already acquired is lost; but absolute blindness never occurs. The function of the retina never again becomes perfectly normal, even if the cause of the visual disturbance is done away with either through removal of the optical obstacle to sight or through correction of the squint by an operation. [Cases have been reported, some surely authentic, in which an amblyopic squinting eye has acquired good vision either through correction of the refraction or because loss of sight in the good eye has compelled the use of the amblyopic eye.—D.]

When—as in an adult—the development of the retina has once been completed, an obstacle to vision may last for many years without the retina's suffering any harm. Thus cataracts which have formed in adults have been operated upon with perfect success after lasting twenty years or more.

Treatment consists in the earliest possible removal of the obstacle to vision. This rule holds good particularly for the cataracts of childhood, the performance of an operation upon which was formerly as a matter of choice put off till the age of puberty, although we may operate upon cataract (by discission) in children even at the age of a few months with the best results. Exercising of the amblyopic eye is of service in bringing up the functional power of the retina. This is particularly employed in cases of strabismus, where by bandaging or atropinizing the sound eye we force the eye which squints to see (see § 693).

[7] From ἀ priv., and ὤψ, sight.

569. (3) **Hemeralopia**[8] **(Night Blindness).**—By hemeralopia, in the widest sense of the term we understand that condition in which one sees well by day, but by night or under feeble illumination from any cause sees poorly or not at all. [In the narrower sense it is used to denote a special disease (*idiopathic hemeralopia*).]

In this disease the patients state that they can see perfectly well in the daytime, but that in the poorly lighted streets in the evening they have to be led about. If tests of the vision are made in such a case, it is found that, in accordance with those statements of the patient, the visual acuity is normal when the illumination is good, but sinks with unusual rapidity when the illumination is diminished. If by letting down the window curtains we darken the room to such an extent that the examining physician can still read medium-sized print, the patient will perhaps no longer recognize the large letters, or he will even in walking through the room stumble over the chairs which stand in his way. A more accurate examination made by means of Förster's photometer (see page 127) shows a considerable reduction of the light sense. If the retina is set in action by sufficiently strong stimuli—i. e., by brilliant images—it performs its functions normally but as soon as the stimuli sink below a certain limit it no longer reacts toward them. This condition is called *torpor retinæ*.

Examination with the ophthalmoscope shows no changes whatever in the interior of the eye. But in most cases there is a xerosis of the bulbar conjunctiva (see page 229); i. e., we find in the latter upon the outer and inner side of the cornea a small rounded or triangular spot, over which the surface of the conjunctiva looks dry, and seems as if covered with a fine, whitish foam. Xerosis of the conjunctiva has no other connection with torpor retinæ than that both are symptoms of a reduced state of nutrition of the eyeball.

Idiopathic hemeralopia originates in a disturbance of nutrition of the retina, the nature and causes of which have not yet been fully investigated. The disease attacks mainly men, especially those of middle age; less often women. A predisposition to it is afforded by a reduction of the general nutrition. The disease accordingly is found in people who in general are insufficiently nourished, as the inmates of workhouses, penal establishments, or orphan asylums, soldiers (very frequently affected in the present war), and sailors (in these occurring simultaneously with scurvy). In Russia, the disease is found especially during and after the long fast at Easter, during which time the people eat no meat. Furthermore, hemeralopia is sometimes observed with jaundice, with intermittent fever, with chronic alcoholism, and also in pregnant women. Whether dazzling of the eyes by a bright light may give rise to hemeralopia is questionable. In persons who are thus predisposed to it the disease develops usually in spring. It often affects a number of persons at the same time.

The prognosis of idiopathic hemeralopia is favorable, as the disease usually gets well of itself after some weeks or months. It, however, leaves behind it a tendency to recurrences which usually make their appearance in the spring or summer of the following years.

As regards the treatment, the use of cooked liver and of cod-liver oil have for a long time enjoyed a great and deserved repute among the laity. In addition we take care to elevate the nutrition by strengthening diet and by corroborative remedies and to protect the eyes from light. In the lighter cases we make the patient wear dark glasses, in the severer cases we keep him for several days in a dark room. By this treatment an abbreviation of the disease is secured.

Hemeralopia in conjunction with xerosis of the conjunctiva is also found as a

[8] From ἡμερα, day, and ὤφ, sight.

precursor of keratomalacia, which likewise must be regarded as the consequence of a disturbance of the general nutrition (see page 280).

Hemeralopia in general is not a disease in itself, but simply a symptom which may belong to various diseases. These latter are divided into two groups—opacities in the media and diseases of the light-perceiving apparatus.

(a) *Opacities in the media* excite the symptom of hemeralopia when they occupy the periphery, and leave the center free, as in the case of peripheral opacities of the cornea and lens. Under brilliant illumination, when the pupil is contracted these no longer fall within the area of the latter, while, when the illumination is diminished and the pupil is dilated, they project into the pupil and interfere with sight. Again when there are faint diffuse opacities distributed uniformly over the entire cornea, the sight is often better when the pupil is contracted, because the dazzling due to diffused light is then less. In all these cases, therefore, the width of the pupil is what determines the visual power. On the other hand,—

(b) Hemeralopia in *diseases of the light-perceiving apparatus* has nothing to do with the width of the pupil. The diseases to be considered in this connection are idiopathic hemeralopia on the one hand, and, on the other hand, retinitis pigmentosa together with many other cases of atrophy of the retina, e. g., such as occur after retinitis, chorioiditis, glaucoma, etc. The nature of the visual disturbance, however, in these two categories is essentially different. In idiopathic hemeralopia there is a reduction of the light sense (page 126), probably as a result of insufficient regeneration of the visual substances. The fovea centralis even under physiological conditions has a feebler light sense than the periphery of the retina [see page 123] and hence in idiopathic hemeralopia stops functioning at the same time that the periphery of the retina does or even somewhat earlier, as the illumination is reduced—many patients stating that when the illumination is feeble they see a dark spot in the center of the visual field. In retinitis pigmentosa the reverse obtains. The periphery of the retina is diseased and, when the illumination is reduced, no longer functions, while central vision is still intact. Then the visual field becomes so small that it no longer suffices for the patient's orientation (page 114). In idiopathic hemeralopia the visual disturbance is transitory, in retinitis pigmentosa and the allied forms is permanent.

570. (4) **Nyctalopia.**[9]—This is the opposite of hemeralopia—i. e., is the condition in which the sight is better at night or in diminished illumination than in bright daylight.

It, too, occurs in two groups of diseases which have their seat either in the media or in the light-perceiving apparatus—only, in this case the site of the changes is just the reverse of that found in hemeralopia. The opacities of the media causing nyctalopia are centrally situated (in the cornea, pupil, or lens). Hence, when the pupil is contracted they occupy its entire area; on the contrary, when, owing to diminished illumination, the pupil dilates, its peripheral portions which are still transparent can be used for seeing. The affections of the light-perceiving apparatus causing nyctalopia are those in which the outlying portions of the field of vision are normal, while in the center there is a scotoma. In these cases, to be sure, the visual acuity is ordinarily no better with diminished illumination than it is in full daylight, but the feeling that central vision is blunted is less unpleasant, so that the patient imagines that he sees better in the evening. This symptom is most pronounced in tobacco amblyopia (see page 628). This variety of nyctalopia is entirely independent of the width of the pupil.

571. (5) **Color Blindness.**—Color blindness occurs both as a congenital

[9] From νύξ, night, and ὤψ, sight.

and an acquired affection. The former is not a disease but an imperfection of vision dependent upon unknown causes; the latter accompanies many diseases of the retina and optic nerve.

Congenital color blindness is known as daltonism, after the English physicist Dalton, who was himself color blind, and was the first to describe this defect accurately. It may be *total*, so that no color is recognized, and the external world looks to be gray on a gray ground like an engraving (achromatopsia), or it may be *partial*, only a certain group of colors being deficient (dyschromatopsia). The former variety is extremely rare; the latter, on the other hand, is pretty frequent. Very often it is not complete blindness for any special color that is present, but only a rather less marked ability to distinguish one from the other, so that colors are not recognized with the same certainty and at the same distance as is the case in the normal eye (*weakness of the color sense* occurring in what Nagel calls anomalous trichromates). There are thus all sorts of transition forms between normal color sense and total color blindness.

572. Young-Helmholtz Theory of Color Perception.—How we are to distribute the cases of partial color blindness into their different categories depends upon the theory of color perception that we adopt as the fundamental one. In the following discussion we shall first start with the Young-Helmholtz theory. This assumes the existence of three fundamental sensations, corresponding to the fundamental colors, red, green, and violet and supposes the other color sensations to originate through a varying mixture of the fundamental sensations. Partial color blindness would then consist in the deficiency of the sensation for one of the primary colors, so that the color sensations of the affected individual would be compounded only of the other two fundamental colors. According to the fundamental color that is deficient we make the distinction between red blindness [protanopia], green blindness [deuteranopia], and violet blindness [tritanopia.]

Now, in what way does a color-blind person—for instance, one affected with red blindness—comport himself? We are not to suppose that such a one does not perceive red objects at all, or that, if he sees them, they appear perfectly colorless. The fact is simply that the sensation which he has in looking at red objects is very similar to that which green objects excite in him, so that he confounds red and green with each other. To understand this, we must for the present adhere to the Young-Helmholtz theory. According to this, there are in the retina three species of fibers corresponding to the three primary colors. Each one of these species of fibers is set into action by all kinds of colored light, but to a different degree. Some fibers are set into action most strongly by red rays, less so by yellow, still less by green, and least of all by violet. They are hence designated simply as the fibers for the perception of red. The curve represented in Fig. 279 A illustrates the way in which these fibers act. The different colors of the spectrum are laid off upon the abscissa, while the ordinate shows the intensity of the excitation produced by each individual color. In analogous fashion the second set of fibers is set into action most strongly by the green rays (Fig. 279 B), the third group of fibers most strongly by the violet rays (Fig. 279 C).

In Fig. 279 D the curves of all three groups of fibers are erected upon the same

abscissa. The red ray, r', excites most strongly the fibers for the perception of red, more feebly those for the perception of green, and least of all those for the perception of violet. Under these circumstances we get a sensation of red, because the degree of excitation of the fibers for the perception of red exceeds that of the other fibers. In like fashion, a green ray, gr', stimulates the fibers for the perception of green more strongly than it does the other two kinds, and excites the sensation of green. An analogous statement holds good for the sensation of violet (v_1).

Now, a man afflicted with *red blindness* presents a condition differing from this normal one, in that the fibers for the perception of red are absent (Fig. 279 E). If he looks at the spectrum it appears to him shortened at its red end, since he sees only blackness at spots where others still perceive red. A red ray, r', which falls upon this man's retina sets in action only the fibers for the perception of green and those for the perception of violet—and of these the former more markedly, so that the resultant effect will be a green. If green light falls upon the retina, the fibers for the perception of green are again stimulated more strongly than those for the perception of violet, and again the sensation of green is produced. Where, then, we have two different sorts of sensations, viz., red and green, a person with red blindness has two that are similar—namely, both green. (The shade of green which appears to most persons with red blindness to have the same color as red is that hue of bluish-green which is complementary to red.) A person with red blindness, however, can distinguish these two sensations from each other, for, though similar indeed, they are not quite the same. They are distinguished from each other by their difference in luminosity. For let us assume that the red and green rays selected as an example are of the same luminosity to a normal eye. Such an eye can still distinguish them apart, owing to their difference in color. The case is otherwise with a red-blind man; in him the red ray, in spite of its luminous intensity, causes but slight stimulation of the fibers for the perception of green, simply because these fibers are in any case but slightly sensitive to red rays. The sensation produced by the red ray is hence a feeble one, and the color which is seen looks dark. The green ray, on the other hand, is perceived in its full luminosity, because the fibers for the perception of green are stimulated by it in the normal fashion. In this way it is generally possible for the man with red blindness to distinguish red from green, not indeed by

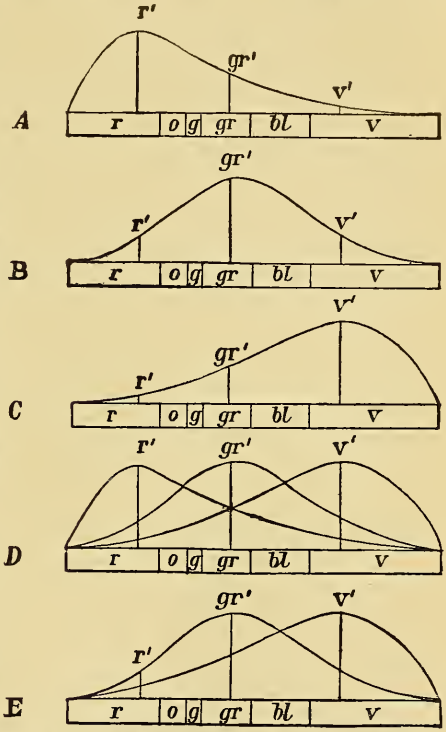


FIG. 279.—REPRESENTATION OF COLOR PERCEPTION, ACCORDING TO THE THEORY OF YOUNG AND HELMHOLTZ.

The abscissa represents the spectrum, the colors of which are red (r), orange (o), yellow (g), green (gr), blue (bl), violet (v): the curves which rise above the abscissa show graphically the sensitiveness of the three sorts of fibers in the retina toward rays of different wave lengths. The ordinates, r^1, gr^1, and v^1, indicate the intensity of the stimulation of the fibers produced by red, green, and violet rays, respectively. *A* gives the curve representing the sensitiveness of the fibers for the perception of red; *B*, that of the fibers for the perception of green; *C*, that of the fibers for the perception of violet. In *D*, all three curves are represented at the same time. *E* shows the curves of sensitiveness of a red-blind eye in which the fibers for the perception of red are supposed to be wanting.

the difference in color, but by their difference in luminosity. This difference, however, between the character of his own sensation and that of a man with normal vision usually remains unknown to the color-blind person. When growing up, he learns the expressions red and green from his associates, certain objects being pointed out to him as red and others as green. He is told that the leaves of the cherry tree are green and the cherries between them red. And, as he too notices a difference between the leaves and the cherries, although it is a difference of luminosity and not of color, he thinks that he sees just as other people do. On account of the sensitiveness to differences of luminosity, which color-blind persons usually possess, they can generally tell correctly the color of objects even when they have never seen them before. Thus it happens that, in the case of many color-blind persons, neither do they themselves know anything of their defect nor are their associates aware of it. Thus a physician once came to me who was charged with the task of testing the employees of a railroad for color blindness. He wished to inform himself under my tuition in regard to the methods of investigating the color sense. When I came to show him the different tests, it soon turned out that he himself was red-blind. Up to that time he had known nothing of this fact, and indeed was quite offended at the imputation of being color-blind. And it even happens that the color-blind carry on occupations which in a peculiar degree demand an excellent sense of color; thus there are color-blind painters.

While with many of the color-blind the defect remains undiscovered during their whole life, in others it is disclosed by their committing some gross mistake in the choice of colors, as, for instance, in the case of the tailor who wished to mend a black coat with a patch of red cloth. How does a color-blind man commit such mistakes? We have seen above that a man with red blindness distinguishes red and green of equal luminosity by the fact that the former looks darker to him than the latter. If now we gradually diminish the luminosity of the green, we must finally reach a point at which this color looks no lighter to the man with red blindness than does the red which has not been altered in luminosity. At this moment he is deprived of the means of discriminating between the two colors, afforded by their difference in luminosity, and is now unable in any way to distinguish the two colors apart. Colors chosen upon this principle are hence known as *confusion colors*. On account of the great sensitiveness of color-blind people to differences in luminosity, the preparation of these confusion colors require great care, and is best performed by painters who are themselves color-blind, and who keep toning down two different colors until they seem to them to be alike. Confusion colors prepared in this way are very well adapted for the detection of color blindness (Stilling, Nagel).

What has been said in regard to those affected with red blindness is also true of the other two classes of the color-blind, those affected with *green blindness* and those affected with *violet blindness*. The circumstance that is common to all people affected with partial color blindness is that one of the three fundamental sensations is deficient. It is not necessary that one of the three species of fibers should be completely absent, as, for the sake of simplicity, has been assumed in the example above adduced. On the contrary, it is probable from various reasons that the excitability of one kind of fibers has simply become altered so that its curve is to be imagined as something different from that which the plan outlined above presents; the curve of the fibers for the perception of red, for example, approximating to that of the fibers for the perception of green.

573. Other Theories.—Many authors place *Hering's theory of color perception* at the basis of their classification of color blindness.

This theory starts from an analysis of the sensations which we have in looking at a color. Most colors excite in us a mixed sensation. Thus, in orange we see, besides

yellow, a certain amount of red; another sort of yellow, again, has a tinge of green, etc. Still, among all the shades of yellow, there is one in which we can perceive no other color besides yellow; this is the pure or primary yellow. Of such pure colors which excite in us a simple, unmixed sensation there are besides yellow only three—namely, pure red, pure green and pure blue. These four primary colors form two pairs—namely, red and green and yellow and blue. The two colors of each pair are called contrary colors, because they have this peculiarity that they never can be perceived at the same time in the same color. We can conceive of a blue which affords simultaneously the impression of some green or some red, but we cannot conceive of one which would also excite the sensation of yellow. The contrary colors, therefore, are mutually exclusive, so far as sensation is concerned.

Every color may occur in different degrees of concentration and of luminosity. This depends upon the fact that every color produces in us besides the sensation of color also that of white. Colors along with their color "value" have also a white "value" and upon the mutual relations of these two values depend the concentration and brilliancy of the color. The primary colors have along with the white "value" only one color value, but the mixed colors have two. Thus, there belong to violet a blue, a red, and a white value. Now, the way in which the action of light upon the terminations of the nerves in our retina takes place is that bodies ("visual substances") are present in the latter which suffer chemical changes due to the light. Such changes may be of two different and indeed opposite kinds, the visual substances being either decomposed ("disassimilated") or regenerated ("assimilated") by the light. The white value which all colors possess depends upon the disassimilative action which they exert upon the visual substance for the perception of black and white. In the absence of light, assimilation of this substance takes place, so that we have the sensation blackness. Besides the black and white visual substance there are two others—namely, a red-green and a blue-yellow substance, as we will call them for short. These are not altered by every kind of light, but only by that kind which has the corresponding color value. Pure red, for instance, would disassimilate only the red-green substance, pure green would cause its assimilation, while violet acts both on the red-green and on the blue-yellow substance. If pure red and pure green light fall at the same time upon the same spot of the retina, it depends upon the proportion between the two whether disassimilation prevails over assimilation or the contrary. The resulting sensation consequently is either red or green, but never both together. If the two contrary colors are so chosen with respect to their quantity that they are in equilibrium in their action upon the visual substance, their color values are abrogated; there only remains the action of the two kinds of light upon the black-white substance, so that there is a sensation of white of a certain degree of luminosity. For these reasons, therefore, the contrary colors mutually exclude each other in sensation, and when mixed in certain proportions afford a sensation of absence of color (i. e., they are complementary colors).

According to Hering's theory of colors, the cause of color blindness must be conceived to consist in the absence of one or of both of the visual substances for colors. In the latter case, in which nothing but the black-white visual substance is left, total color blindness exists; all colors now act simply by reason of their black-white values, and hence are perceived as white of different degrees of luminosity (i. e., gray). Absence of the red-green visual substance causes red-green blindness, absence of the blue-yellow substance, blue-yellow blindness. The former comprises the great majority of cases—namely, those which according to the theory of Helmholtz are called red-blind and green-blind. A man with red-green blindness sees in the spectrum only two colors, yellow and blue. These are separated by a gray space (the "neutral" space) which corresponds to the pure green. Pure red and pure green act upon the eye affected

with red-green blindness only with their white values and hence both appear gray, on which account they are by such an eye confounded with one another. Mixed colors undergo an alteration of their tone, inasmuch as of their two color values but one comes into play.

[A number of *other theories* of color perception have been advanced but none has been accepted as explaining all the facts in normal and pathological cases.—D.]

574. Vision of the Color-Blind.—If we disregard the theories for explaining color blindness and confine ourselves to the simple facts, we must admit at the outset that we cannot at all know what sensations the separate colors excite in a man with congenital color blindness. The only sure thing is that the man with total color blindness sees all colors of the spectrum of the same hue and differing only in luminosity, and that the man with partial color blindness recognizes only two colors in the spectrum, while for the man with normal sight at least three fundamental sensations must be assumed to exist, in order to be able to obtain all color sensations from them by mixture. Hence, we call the former dichromates, the latter trichromates.

In the most frequent form of color blindness the two colors that are perceived in the spectrum correspond in position to yellow and blue, between which lies the neutral area, which looks gray; and the two colors confounded are red and green, both looking like a certain shade of gray. Of such color blindness two types can be distinguished. The first (the red-blind of Helmholtz) sees the spectrum shortened at the red end, and shades of red hence look comparatively dark. The other type (the green-blind of Helmholtz) sees the spectrum unshortened. Hering classes both types as red-green blind. Since the first type confounds with gray a red that shades into yellow, i. e., it is undersensitive to yellow—he calls it the relatively blue-seeing type of red-green blindness. The second type sees a somewhat bluish-red as a gray, and hence is called in Hering's nomenclature the relatively yellow-seeing type of red-green blindness.

Violet blindness (Helmholtz), or blue-yellow blindness (Hering), like total color blindness is rare.

Congenital color blindness occurs, as observations upon a great number of men have shown in from 3 to 4 per cent of the male population. In women color blindness is much more rare, perhaps because their color sense undergoes a sort of education through their having such frequent occasion to be busy with colored objects (in dress-making, etc.).

575. Tests for Color Blindness.—Color blindness entails no disadvantage upon those who are troubled with it beyond rendering them less fit for the performance of certain callings. Among these are all those occupations which require precise discrimination of colors, e. g., that of the painter, the dyer, etc. Recently particular attention has been called to the fact that the railroad and nautical services also require an accurate sense of color. The signals used on railroads or ships are most frequently red or green, which are just the colors that are confounded by most color-blind persons; in this

way accidents might be [and are] caused. For this reason the employees upon railroads and sea-going vessels are at present in most countries tested with reference to their color sense, and their entry into the service is made conditional upon the proof that their color sense is perfect.

The *demonstration of the existence of color blindness* requires accurate and careful testing. Many of the color-blind who are aware of their defect try to conceal it from the examiner, especially if some material advantage, such as, for instance, a business position, depends upon the result of the testing. Accordingly, in the case of such persons we must be on the lookout for all sorts of artifices, and particularly on the lookout for previous practice in the ordinary methods of testing the color sense. On the other hand, people with a good color sense might be considered to be color-blind if from want of imagination or practice they call the colors that are set before them by incorrect names. We should not, therefore, undertake to test the color sense by setting colored objects before the person and asking him the name of the color. If we do so, the man who is color-blind will in many cases by using a little attention give the right answer, while on the other hand, an uneducated man will not infrequently call the colors wrong. The test is better performed by placing before the person under examination those colors which according to experience color-blind persons readily confound with each other, and then seeing whether mistakes are actually committed. For this purpose a large assortment of colored *worsteds* is used (Seebeck, Holmgren). One of the worsteds is set before the person to be tested, and he is asked to place with it all the worsteds that look like it. If, then, samples of different and indeed quite dissimilar colors are placed together—for instance, gray and red with green—these give the special confusion colors of the person under examination, and make it possible to determine the kind of color blindness that he has. [The worsted test is unsatisfactory because not a few color-blind persons are able to pass it successfully. Much better are the tests of Stilling and Nagel. *Stilling's pseudo-isochromatic diagrams* are composed of patterns (usually numbers) set on a differently colored background, the tints used being] selected by the aid of a color-blind painter so as to correspond to the confusion colors of a man who is color-blind. The latter since he cannot recognize the differences in color [cannot distinguish the number from its background].

[In *Nagel's test*, which may also be used as a quantitative test (see below), there is a set of cards, each bearing a series of little color disks arranged in a ring. In some rings the disks are all of one color, but of slightly different shades; in others the disks are of two or three different colors (confusion colors). By making the patient state which rings are monochromatic and then making him pick out in the dichromatic or trichromatic rings all the disks that are of one special color, we can readily ascertain whether he is color-blind and what sort of color blindness he has. This and Stilling's test, supplemented by the lantern test (see below) afford the simplest and most satisfactory means of determining the presence of color blindness.—D.]

For the accurate determination of confusion colors *Nagel's anomaloscope* is used. [In this the patient, looking through a tube, sees two semicircles, one containing a sodium yellow, the other either a lithum red, a thallium green, or a mixture of red and green. The intensities of the colors can be varied at will by a measured amount. The intensities of colors used to make a match between the two semicircles indicates the state of the patient's color sense.—D.]

For the scientific examination of the color-blind, the *spectroscope* is indispensable. By its aid we determine whether the color-blind man sees the spectrum shortened at one of its two ends, and what colors he can distinguish in it. Furthermore, by means of the apparatus we show him isolated portions of the spectrum and make him tell

both by naming the colors and by comparing them with specimens of other colors, how the different parts of the spectrum look to him.

For a *quantitative* determination of the color sense, the method of Donders, Weber, Wolffberg, and others is adapted. Here small disks of colored paper upon a background of black satin serve as test objects. When the color sense is normal, disks of a definite size must be recognized at a definite distance, which to be sure, is different for the different colors. The weaker the color sense of the person under examination, the nearer must he get to be able to tell the color correctly, even supposing that he recognizes it at all. The distance at which the color begins to be recognized gives the intensity of the color sense for the color in question. Instead of colored paper we may use colored glasses which are lighted from behind. These last tests (*lantern tests*) approximate nearest to the conditions which are present in the railroad service. [Excellent lantern tests, particularly adapted for testing railroad employees are those of Edridge Green, Thomson, and Williams.—D.]

It is impossible to cure congenital color blindness.

576. Acquired Color Blindness.—This is a frequent symptom of affections of the light-perceiving apparatus—that is, of the retina, of the optic nerve, and even of the central terminations of the optic tracts. Affections of the optic nerve, particularly its atrophy, are, however, by far the most frequent cause of disturbances of the color sense.

Such disturbances are never absent when once the visual acuity has become considerably reduced as a consequence of the affection of the optic nerve. In these cases the color blindness does not set in suddenly, nor for all the colors at once; but first and very gradually the perception of green and red is extinguished, then that of yellow, and last of all that of blue. Acquired color blindness may therefore be utilized for purposes of diagnosis. If the sight is impaired simply by dioptric obstacles (e. g., by opacities in the cornea and in the lens), the perception of color [although less keen] remains [fairly] intact, even when the general features of objects can no longer be recognized; but as soon as the color sense proves to be defective, an affection of the light-perceiving apparatus must be assumed to exist. (For the color sense in the periphery of the visual field, cf. page 124 and Fig. 37.)

[All the tests used for congenital color blindness can be used for the acquired form. They are particularly important in the tobacco and alcohol amblyopias so often occurring in railroad and marine employees, in whom, therefore, frequent re-examination of the color sense, especially by the Nagel, Stilling, and lantern tests, should be made.—D.]

B. The Seat of the Disturbance of Sight is Central

577. (1) **Amblyopia and Amaurosis in Brain Diseases.**—Disturbance of vision may be set up by disease of the brain without their being any ophthalmoscopically perceptible changes in the eye, such as neuritis or atrophy of the optic nerve. Disturbances of vision of this sort may be only transient, even when they amount to absolute blindness. Uræmic amaurosis (see page 579), which is produced by retention of the urinary constituents, affords a good example of this. But in those cases in which gross lesions of the brain, such as inflammatory processes or new growths, give rise to the disturbance of vision, the latter is permanent, and ophthalmoscopic changes,

usually under the form of a descending atrophy of the optic nerve, are often associated with it later. Disturbance of vision dependent upon a central cause makes its appearance not infrequently under the guise of *hemiopia* (homonymous or temporal).

578. (2) **Scintillating scotoma** (scotoma scintillans, amaurosis partialis fugax, or teichopsia[10]).—The patient who suffers from this notices besides a feeling of vertigo a sparkling light that appears before his eyes, and rapidly increases until finally he can scarcely see at all. Persons who are more accurate observers of their sensations usually aver that the sparkling originates from a small spot situated not far from the point of fixation, and that within the area represented by this spot external objects are invisible (hence the name scintillating scotoma). The scintillation and with it the gap in the visual field spread rapidly, the boundaries of the scintillating area being often formed by lines zigzagging in and out, so as to form projecting and re-entrant angles. After a quarter or half an hour, the attack abates, the visual field beginning to clear up at the point first affected. Scintillating scotoma is usually accompanied by headache and sometimes also by nausea, and frequently a regular attack of migraine is joined with it.

The central origin of scintillating scotoma is apparent not only from the accompanying and following headache, but also from the fact that it [almost] always affects both eyes in the same way, and frequently occurs under the form of hemiopia—i. e., it occupies but one half (and that too, the homonyous half) of the field of vision in each eye. The symptoms, on account of their short duration can be caused only by disturbances of circulation, whose transient character indicates that it is not anatomical changes but disorders of innervation in the vessels that lie at the root of them. With extension of the angioneurotic disturbance to the other areas, other disorders of central origin, such as formication in the lips and arm, weakness or paralysis of one of the extremities, aphasia, etc., set in. These come and go with the scintillating scotoma. But scintillating scotoma is most intimately associated with the most frequent angioneurosis of the brain, i. e., migraine, so that it has also been called by the name of migraine oculaire. The circulatory disturbance sets up an irritation of the optical elements—an irritation which according to the laws of projection is referred to the external world, and appears under the form of a colored scintillation, while at the same time the perception of peripheral impressions is abolished. So also at the beginning of a fainting attack, which in fact is likewise due to circulatory disturbances in the brain, symptoms make their appearance which are perhaps identical with scintillating scotoma; the patients averring that everything looks green and blue, or scintillates, or grows dark before the eyes.

Scintillating scotoma is an uncommonly widespread affection. If it occurs infrequently—at intervals of several years—no significance is attached to it by the patient, as it disappears again rapidly, and without leaving any bad results. It is only when the symptom is repeated frequently—and it may even recur several times a day—that those who are troubled with it come to the physician. Such patients allege as the cause of their scintillating scotoma excessive physical or mental exertion, straining of the eyes, dazzling light, or a great sense of hunger; often, however, no definite cause can be made out. The treatment must be confined to opposing the cause

[10]From τεῖχος, wall, and ὄψις, vision, on account of the zigzag lines, resembling fortification walls, often seen on the edge of the scintillating spots.

of the scotoma. Such treatment consists in increasing the general strength and the avoidance of excessive exertion. If the attacks are frequently repeated we order quinine in small doses to be taken for some time. Ordinary cases of scintillating scotoma are associated with no evil consequences of any kind, and it is the exception for scintillating scotoma to be the precursor of a serious affection of the brain (brain tumor, progressive paresis, apoplexy, etc.).

579. (3) **Disturbances of Vision in Hysteria and Neurasthenia.**—These find their expressions under the forms of amblyopia and asthenopia.

Hysterical amblyopia consists in a diminution of the visual acuity, a contraction of the field of vision, and a diminution in the color sense and light sense. The contraction of the visual field is concentric; and in many cases the field gets smaller and smaller the longer the patient is tested with the perimeter (reaction of exhaustion, Förster). This depends upon the rapid exhaustion of the nervous system that is peculiar to patients of this sort.

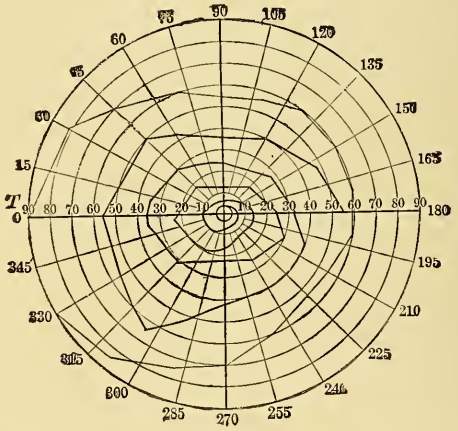


Fig. 280.—Spiral Fatigue Field of Von Reuss. (After Weeks.)

[The exhaustion field is characteristic rather of neurasthenia than of hysteria. If we test each meridian of the field in succession and repeat the examination on reaching the meridian first tested, the progressive shrinking of the limits in each successive meridian may make the field assume a spiral form (Fig. 280). Other peculiar modifications of the field found in hysteria, beside the contracted field mentioned above (see Fig. 281) and the reversed color field described below (see also Fig. 281), are: (*a*) The tubular field. The field is greatly contracted and remains of the same absolute size, no matter how far the patient is removed from the point of fixation. (*b*) The oscillating field, in which there are a series of scotomata in each meridian (see Fig. 282). This seems very rare.—D.] Typical hemiopia does not occur as a purely hysterical affection. Hysterical amblyopia is found to the most marked degree in those cases of hysteria that are associated with disturbances of sensibility (hemianæsthesia, etc.). It is generally present in both eyes, although for the most part to a greater degree on the side upon which the general sensibility is affected.

The diagnosis of hysterical amblyopia is based principally upon two points. The first of these is the absence of any demonstrable changes in the eye which might explain

the enfeeblement of sight. The second is the failure of the separate symptoms constituting the disturbance of vision to show that agreement with each other that they ordinarily present. Thus, the acuity of vision and the extent of the visual field change frequently (usually doing so as the other hysterical symptoms grow better or worse); the relations of the color limits within the visual field are not in accordance with the rule (the limits for red, instead of being narrower than those for blue—see page 125—, are usually wider [or cross them in places—reversal and interlacing of color fields]); these relations are not properly proportioned to the total extent of the visual field; persons whose visual field is unusually contracted still move with perfect security and without stumbling in a space which is not well known to them; in fact, even in those who are absolutely blind we sometimes find the same thing occur if they think that they are not observed. Furthermore, the pupillary reflex to light is retained even when the blindness is complete. It can be seen from these statements that it is often difficult to draw the line between simulation and a hysterical blindness—

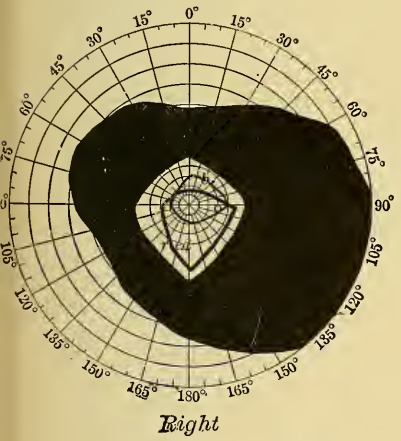


Fig. 281.

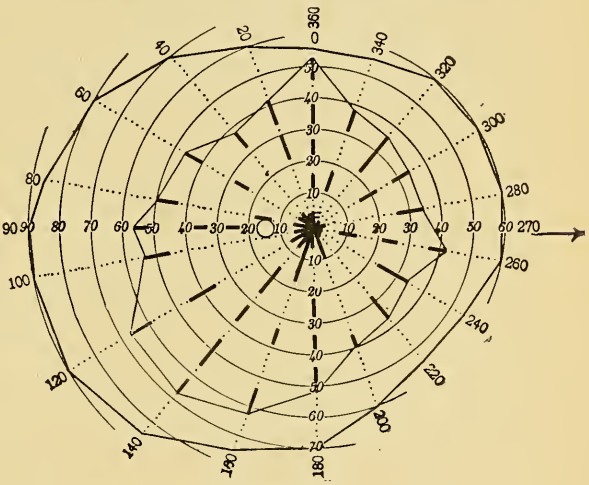


Fig. 282.

[The Visual Field in Hysteria. (After De Schweinitz in Posey and Spiller.)
Fig. 281.—Contracted field (in this case a tubular field) with partial reversal of color fields. *b*, field of blue. *r*, field for red.
Fig. 282.—Contracted and oscillating field (Wilbrand). The dark lines denote scotomata.—D.]

i. e., one having an actual existence in the imagination. In the latter case there may be other evidences of hysteria associated with the symptoms of the hysterical amblyopia which will render the diagnosis more certain.

[Von Reuss has called attention to the fact that in hysteria the involvement of the field is apt to remain constant, while in neurasthenia it is variable (Peters).—D.]

Hysterical amblyopia chiefly attacks young people, particularly of the female sex. It is sometimes produced by injuries, even when they do not affect the eye itself (traumatic neurosis). Hysterical amblyopia affords a good prognosis, as ordinarily a complete cure takes place. The disease, however, usually lasts for a long time, often for years. Treatment consists in the management of the causal lesion, re-enforced locally by hypodermic injections of strychnine and the application of the constant current. The brilliant results sometimes obtained by the two last-named remedies are, however, mainly ascribable to their psychic influence upon the patient when the latter has confidence in the treatment and anticipates a cure from it.

[A temporary amblyopia or perversion of vision, apparently nervous and the result of shock, since it is unaccompanied by ophthalmoscopic changes and disappears in two or three weeks, is produced by shell explosions. (Jessop, Lister.)—D.]

Nervous asthenopia,[11] a condition which occurs both in hysterical subjects and in neurasthenics, consists in an incapacity of the eye for any continuous exertion, in spite of there being good visual power.

Some complain that after reading or working for even a short time everything becomes covered with a cloud so that the work has to be laid aside. Others, again, allege that after pursuing their occupation for a little while, indeed even after reading a few lines, they have violent pains in the lids, eyeballs, or head, which render the continuance of the work impossible. When no strain is put on the eyes, there is generally no trouble; in other cases, however, the pains never entirely disappear, or a great sensitiveness to light is constantly present.

In making the diagnosis, proof must first of all be forthcoming that there is no error of the refraction or of the muscular equilibrium to cause the trouble. [These cases are frequently associated with convergence-insufficiency and with insufficiency of accommodation, both of which may require treatment (see §§ 684 and 796). Other cases are due to reflex disturbances and remote infections (especially infection or irritation in the nose and its accessory cavities, in the teeth, and in the tonsils.—D.] Nervous asthenopia, like hysterical amblyopia, with which it frequently is associated, is often extremely obstinate, and sometimes for years prevents the patient affected by it from engaging in any serious occupation. In it, too, the psychical factor plays a great part in the treatment. I have found electricity the most efficient means.

[11] [See also page 354.]

CHAPTER XII

DISEASES OF THE LIDS

Anatomy and Physiology

580. External Anatomy.—The lids (palpebræ[1]) are, in origin, folds of the external skin, which push their way over the eyeball to cover and protect it. The boundaries of the upper lid are formed by the eyebrow (supercilium), but the lower lid passes without any sharp line of demarcation into the cheek. The lids bound the palpebral fissure, at the two extremities of which (the angles of the eye) they unite. The external angle of the eye (canthus externus) runs out to a sharp point; when the lids are drawn apart there is put upon the stretch a delicate reduplication of skin (the external commissure), connecting the upper and lower lids in this situation. The inner angle of the eye, on the contrary, presents a horse-shoe shaped notch, at the bottom of which lies the caruncle (Fig. 60, *C*). The mean width of aperture of the palpebral fissure varies with the individual. On an average, the fissure opens so far that, with the ordinary way of looking, the upper lid covers the uppermost part of the cornea, while the lower lid leaves the lower margin of the cornea free. The shape and width of the palpebral fissure are of the greatest influence upon the expression of the eye. Eyes which have the reputation of being large and beautiful are generally not really large eyeballs, but eyes with a wide-open palpebral fissure. So, too, the expression of the laity that "the eye is smaller" usually has reference not to an actual diminution in the size of the eyeball, but to a lesser degree of patency of the palpebral fissure.

The skin covering the lids is about the thinnest in the human body. As, moreover, it is but very loosely attached to its bed through the medium of a lax and non-fatty connective tissue, it can very readily be made to shift its position. For the same reason it can readily wrinkle up and stretch out again, as the lids open and shut. In old people it is thrown into numerous wrinkles. Because of the ease with which it is displaced, it is readily distorted by scars in its vicinity, so that ectropion cicatriciale is produced. So, too, on account of the laxity of its attachment, it is very apt to be affected by extensive ecchymoses and œdema. It is only in the neighborhood of the free border of the lid that the skin is closely united to the subjacent tarsus by rigid connective tissue. This free border of the lid forms a narrow surface which looks downward in the upper lid, upward in the lower lid (*r*, *r*, Fig. 48 A). When the lids are closed, these two surfaces are adjusted to each other with perfect accuracy, so that, with the aid of the lubri-

[1] From *palpare*, to stroke.

cation afforded by the secretion of the Meibomian glands, they are able to keep the lachrymal fluid in. When we open the lids forcibly in people affected with lachrymation and blepharospasm, we not infrequently see spurt from the eye a stream of tears which have been kept in by the closed lids—a proof that the closure of the lids is water-tight.

The lines along which the free border of the lids is reflected on to the anterior and posterior surface, respectively, of the lid are called the anterior and posterior margins of the lids (Fig. 283, *v* and *h*); the narrow surface lying between them is the intermarginal strip. The anterior margin of the lid is rounded off, and has jutting from it the eyelashes (cilia), which are arranged in several rows one behind the other. The cilia upon the upper lid are larger and more numerous than upon the lower. The posterior margin of the lid, where the free border of the lid passes into the conjunctival surface of the latter, is sharp. Directly in front of it lies a single row of small puncta, the orifices of the Meibomian glands (Figs. 283 and 284, *m*). Between these and the cilia runs a fine gray line (*i*, Fig. 284) which divides the intermarginal strip into two parts, anterior and posterior. The free border of the lid has the above character as far inward as the spot where the punctum lacrimale is situated, a spot which corresponds to the inner extremity of the tarsus (*p*, Fig. 284 and Fig. 285).

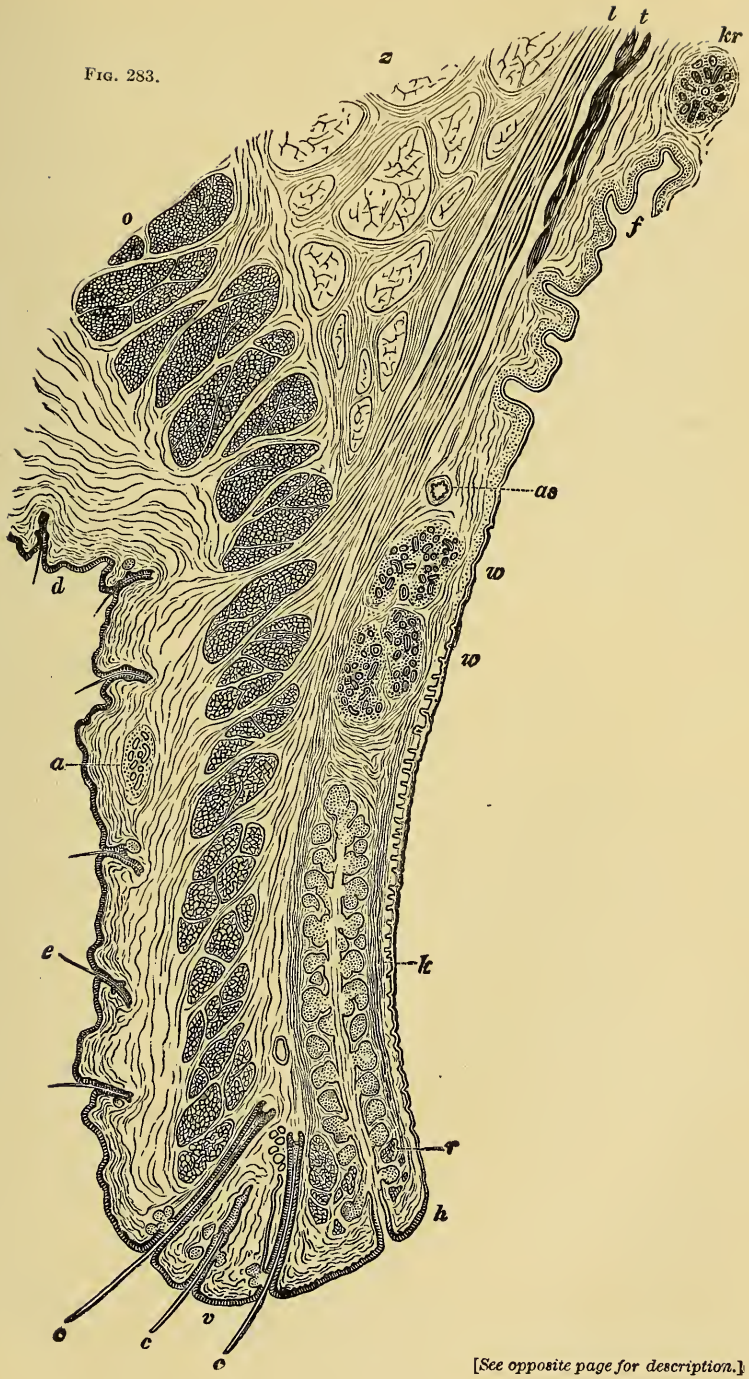
On everting the lids we get a view of their posterior surface, which is covered with the conjunctiva. This is intimately adherent to the tarsus, and, particularly in the upper lid, allows the Meibomian glands situated in the tarsus to show through clearly.

That part of the cornea and of the scleral conjunctiva which ordinarily is not covered by the lids is called the *interpalpebral zone*. Since in this situation the eyeball is deprived of the protection of the lids, it is particularly exposed to many sorts of disorders, and it is therefore important to know its situation. This situation changes according to circumstances, and in the following way: 1. In the ordinary way of looking the interpalpebral zone comprises the entire cornea, with the exception of its extreme upper part, and comprises also a corresponding large triangular area of the conjunctiva on both sides of the cornea. 2. When the eyes are a little screwed together—e. g., when we are walking in the face of the wind or rain or in the midst of smoke—the interpalpebral zone diminishes in size, and at the same time is depressed so as to occupy

Explanation of Fig. 283.—Perpendicular Section through the Upper Lid. Magnified 6 × 1.

The skin of the lid presents in the upper part, above a sulcus, the covering fold, *d;* below, it covers the anterior edge of the lid, *v*. In the skin are found fine hairs, *e e*, sweat glands, *a*, and on the anterior edge of the lid, cilia, *c*, *c*, *c*. In the neighborhood of the latter are situated the sebaceous glands (Zeiss's glands), and in front of the hair-papilla of the hindmost cilium is seen the transversely divided tubule of a modified sweat gland (gland of Moll), the excretory duct of which runs down alongside of the cilium and empties into its hair-follicle. Beneath the skin lie the transversely divided bundles of fibers of the orbicularis, *o o*, of which those placed most posteriorly, *r*, form the musculus ciliaris Riolani. The posterior surface of the lid is covered by conjunctiva which is intimately adherent to the subjacent tarsus, *k*, and over the latter shows papillæ, especially in the area between *k* and *w* corresponding to the convex (upper) border of the tarsus. Still higher up in the neighborhood of the retrotarsal fold, *f*, it acquires an adenoid character. The Meibomian glands have their orifices in front of the posterior edge, *h*, of the lid; above them lie the branched, tubular glands, *w*, *w*, and still higher Krause's glands, *kr*, and in front of these Müller's musculus tarsalis superior, *t*, and the tendon of the levator palpebræ superioris, *l*. From the latter leashes of fibers pass between the bundles of the orbicularis to the skin of the lids. *z*, lax cellular tissue. *as*, arcus tarseus superior. Above the roots of the cilia is the cross section of the arcus tarseus inferior.

Fig. 283.



[See opposite page for description.]

the lower half of the cornea. The lower lid is raised a little and covers the extreme lower part of the cornea, and the upper lid drops a good deal so that its border lies only a little above the center of the cornea. Then the interpalpebral zone forms upon the cornea a zone from 4 to 6 mm. in breadth which occupies the lower half of the cornea, with the exception of its extreme lower part, and with which there is connected on either side a very small triangle of scleral conjunctiva. The interpalpebral zone as thus defined is the part which more than any other is constantly exposed to external injuries. Hence, in many men we find this portion of the scleral conjunctiva somewhat injected all the time, and later on in life we find it occupied by the pinguecula. In this spot are developed pterygium, zonular opacity of the cornea, and xerosis of the conjunctiva and cornea. In inflammations of the conjunctiva this division of the latter is frequently distinguished by being somewhat more swollen than the rest, or it may even protrude into the palpebral fissure under the form of a transversely placed, very œdematous swelling. 3. When the eye is turned upward in sleep the interpalpebral zone, in case the lids are not completely closed, is displaced, so as to occupy mainly the scleral conjunctiva beneath the cornea, and at most the extreme lower portion of the latter. Affections within the confines of the interpalpebral zone as thus defined are found when the palpebral fissure is kept open during sleep, and hence mainly in lagophthalmus, in which the conjunctiva beneath the cornea is found injected or œdematous, and in which, when the affection is of greater extent, the lowermost division of the cornea also suffers damage. The same turning upward of the eyeball that occurs in sleep takes place also when one winks because of the approach of anything endangering the eye, for which reason injuries by burns and caustic substances affect principally the extreme lower portion of the cornea.

581 (8) **Lid Muscles.**—In the lids are found two voluntary muscles, the orbicularis (sive sphincter) palpebrarum and the levator palpebræ superioris. The *orbicularis* lies directly beneath the skin of the lid to which it belongs; it is nothing but a flat expanded cutaneous muscle which surrounds the palpebral fissure in the form of a circle. We can distinguish in it two portions, a central and a peripheral. The central portion lies in the lids themselves, and is hence called the palpebral portion (portio palpebralis). Its fibers originate from the internal palpebral ligament, the *ligamentum canthi mediale* (sive *canthi internum*). This is a firm, fibrous ligament (Fig. 284,*l*) which is attached to the frontal process of the superior maxilla (*F*), and lies directly beneath the skin of the internal angle of the eye (Fig. 285). Hence, it is visible even in the living, especially in lean persons with thin skins, if the eyelids are drawn outward, a manœuvre that causes the internal palpebral ligament to project and bulge the skin forward. From the internal palpebral ligament the fibers of the palpebral portion of the orbicularis run in arches over the anterior surface of the two lids, covering them from the free border of the lids to the margin of the orbit, and finally meeting at the outer side of the palpebral fissure. Here they unite by means of a tendinous inscription (the *rhaphe palpebralis lateralis*). Beneath this lies the external palpebral [or canthal] ligament (*ligamentum palpebrale laterale*) (ligamentum canthi externum) which connects the temporal extremities of both tarsal cartilages with the temporal border of the orbit (Fig. 284, *le*;

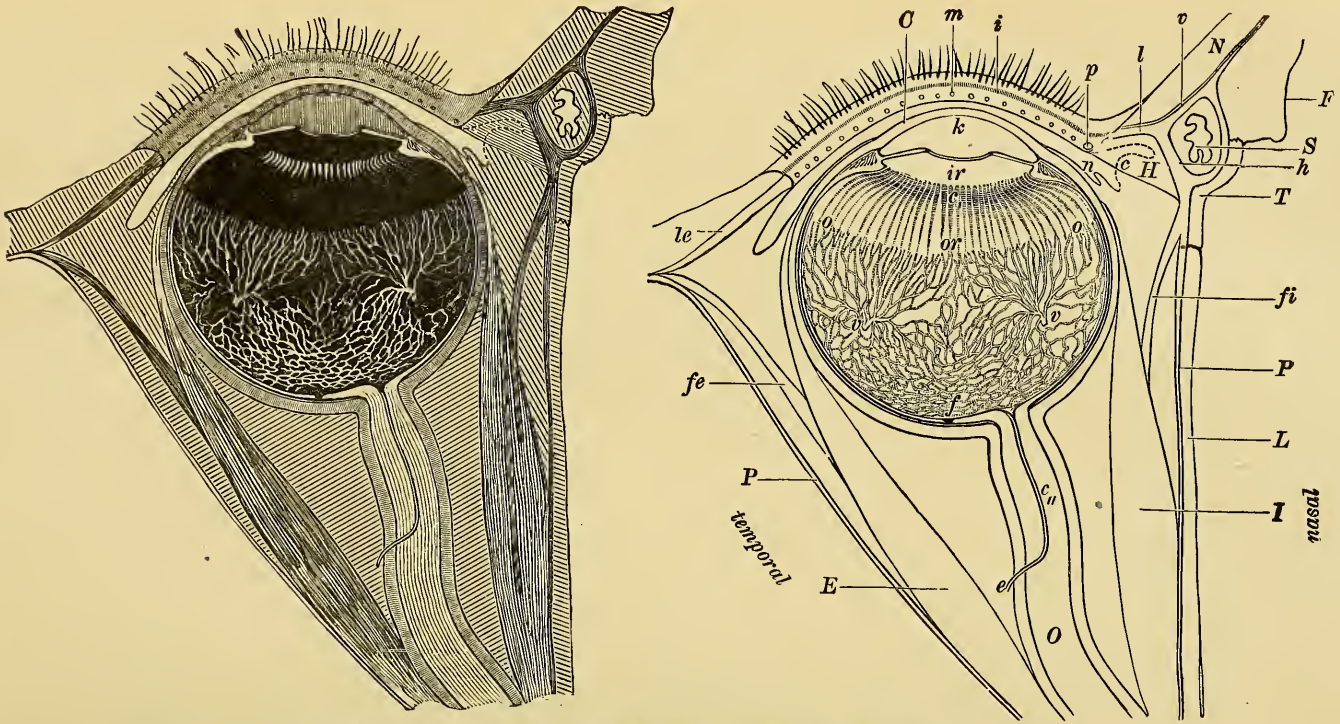


FIG. 284.—HORIZONTAL SECTION OF THE ORBIT. (SCHEMATIC.) Magnified 2×1.

FIG. 284.—The nasal wall of the orbit is formed by the lamina papyracea (os planum) of the ethmoid (*L*), the lachrymal bone (*T*), and the frontal process (*F*), of the superior maxilla. The last two bones bound the fossa sacci lacrimalis, in which lies the lachrymal sac, *S*. The bony walls of the orbit are coated by a periorbita (periosteum), *P*, from which the palpebral ligaments take their origin. The internal palpebral ligament, *l*, divides into an anterior limb, *v*, and a posterior limb, *h*, which together inclose the lachrymal sac. From the posterior limb arise the fibers of Horner's muscle, *H*. *le*, external palpebral ligament; *fi* and *fe*, the slips of fascia [check ligaments], likewise originating from the periosteum, going to the internal rectus muscle, *I*, and the external rectus, *E*. The skin, *N*, of the dorsum of the nose passes into the lower lid, at whose free border are seen the cilia and the orifices of the Meibomian glands, *m;* between the two extends a gray line, *i*. At the inner extremity of the lids lies the inferior punctum lacrimale, *p*, and farther along in the conjunctival sac the caruncle, *c*, and the plica semilunaris, *n*. From the eyeball, the lower half of which is exhibited, the lens and along with it the vitreous humor have been taken out. The anterior chamber, *k*, the iris, *ir*, and the ciliary body, consisting of the corona ciliaris, c_I, and the orbiculus ciliaris, *or*, are visible. Back of the ora serrata, *o*, is the chorioid with its veins which are aggregated into vortices, *v*. *f*, fovea centralis retinæ; c_{II}, central vessels of the optic nerve, *O*, entering it at *e*.

Fig. 285). The external portion of the orbicularis is the orbital portion (portio orbitalis). It lies outside of the palpebral portion, upon the margin of the orbit and the parts surrounding it.

The *levator palpebræ superioris* arises at the bottom of the orbit from the circumference of the optic canal, and from this point runs forward, lying as it does so upon the superior rectus [with which it is intimately connected by bands of fascia.—D.]. Spreading out in the form of a fan, its tendon runs down upon the anterior surface of the tarsus of the upper lid (*l*, Fig. 283). Besides this striated levator muscle of the lid, there is also an organic muscle discovered by Heinrich Müller, and called the *musculus tarsalis superior*. The smooth fibers of this arise from between the striated fibers of the levator, along the under surface of which they too run to the upper margin of the tarsus (*t*, Fig. 283). An analogous bundle of smooth muscular fibers is also present in the lower lid, where it lies to the lower side of the inferior rectus, and is attached to the tarsus of the lower lid (*musculus tarsalis inferior* of Müller).

The orbicularis is innervated by the facial nerve, the levator by the oculo-motor, and the two tarsal muscles of Müller by the sympathetic.

The *ligamentum palpebrale mediale* arises from the frontal process of the superior maxilla (*F*, Fig. 284) and first passes straight outward, skirting the anterior wall of the lachrymal sac (*S*). Then it turns round the anterior and external walls of the lachrymal sac, and runs backward to the crista lacrimalis posterior of the lachrymal bone (*T*). We accordingly distinguish in the internal palpebral ligament two branches, which meet at the point where it begins to turn. The anterior branch (*v*) is situated directly beneath the skin, and hence is visible in the living subject; at its point of reflection it gives off a process to the upper and lower tarsus respectively (Fig. 285). The posterior branch (*h*), which starts from the point of reflection and extends to the crista lacrimalis, can be brought to view only by dissection. The two branches together with the lachrymal bone (*T*) bound a space, triangular on cross section, in which lies the lachrymal sac, the walls of the latter being united by loose connective tissue with the inner surface of the ligament. Into the external surface of the ligament are inserted the fibers of the palpebral portion of the orbicularis. One portion of the fibers of the latter springs from the anterior, another portion from the posterior branch of the ligament. The latter fibers, whose insertion is in part continued out beyond the posterior extremity of the ligament upon the inner wall of the orbit, are called the *pars lacrimalis musculi orbicularis*,[2] or, from their discoverer, Horner's muscle (*H*). The insertion of the fibers of the orbicularis into the internal palpebral ligament is of significance for the conduction of tears. When these fibers contract, they draw up the ligament, and hence also indirectly the wall of the tear sac so far as it adjoins the ligament. The sac is thus dilated, and enabled to suck up the tears (see § 625).

The fibers of the orbicularis fuse with the anterior surface of the tarsus. In the neighborhood of the free borders of the lid there are some bundles which lie near the inner margin of the lid, partly in front of, partly behind the excretory ducts of the Meibomian glands (*musculus ciliaris Riolani* sive *subtarsalis; r*, Fig. 283).

582. Tarsus; Glands of Lids.—The *tarsus* (*k*, Fig. 283) forms, so to speak, the skeleton of the lid, giving it rigidity of form and affording it firm

[2] [The tensor tarsi of some anatomists.—D.]

support. The tarsus of the upper lid is broader (higher) than that of the lower (Fig. 285). In the tarsus are distinguished a free and an attached (convex) border, and also an anterior and a posterior surface. The fibers of the orbicularis (*o*, Fig. 283) lie upon the anterior surface, while the posterior surface is covered by the conjunctiva. The two extremities of the tarsus are continuous with the external and internal palpebral ligaments. To the convex border of the tarsus is attached a fascia which runs from it to the margin of the orbit, and upon either side is connected with the

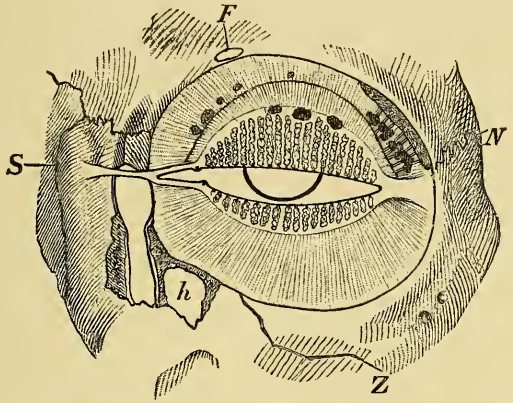


Fig. 285.—Septum Orbitale and Lachrymal Sac. Natural size.

The skin and the muscular fibers of the orbicularis have been removed from the lids and the parts surrounding them, so that the septum orbitale lies exposed to view within the bony circumference of the orbital cavity. The septum orbitale consists of the tarsus, which is broader in the upper lid, narrower in the lower, and of the fascia tarso-orbitalis. The external extremities of the tarsi are attached by the broad, but not very dense rhaphe palpebralis lateralis to the malar bone, somewhat below the suture, *N*, between this bone and the zygomatic process of the frontal bone. The internal palpebral ligament is narrower, but stout; its anterior limb, which is the only one visible in the drawing, runs from the frontal process of the superior maxilla, *S*, outward and divides so as to be inserted into the inner extremities of both the upper and lower tarsal cartilages. (At the point of insertion is seen the somewhat projecting papilla lacrimalis.) The fascia tarso-orbitalis, represented in the drawing by the radial lines of shading, runs from the convex border of both tarsal cartilages, and from the palpebral ligaments to the margin of the orbit, and together with these parts closes in the orbit in front. The tarsal cartilages and the fascia are here supposed to be transparent. Hence, in the former there can be seen the Meibomian glands, which, in consonance with the varying breadth of the tarsus, diminish in height from the center of the latter to its two ends. Moreover, in the upper lid three acinous glands (cf. Fig. 283, *w*) are visible along the upper border of the tarsus. Still higher up a curved line shows the situation of the fornix conjunctivæ. Upon the fornix, especially in its nasal half, lie the acinous glands of Krause (Fig. 283, *kr*), while in the temporal half of the fornix are found lobules similar in character, but more densely packed, representing the inferior lachrymal gland. This adjoins the excretory ducts of the superior lachrymal gland, whose anterior border comes into sight just below the upper margin of the orbit. At the inner and lower margin of the orbit the bone has been chiseled away to show the lachrymal passages. The lachrymal sac lies behind the internal palpebral ligament, its apex rising a little above the latter. The line that in the drawing runs straight upward from the apex of the lachrymal sac to the horizontal suture is the suture between the frontal process of the superior maxilla and the lachrymal bone, upon which two bones the lachrymal sac rests (cf. Fig. 284, *F* and *T*). The lachrymal sac, after undergoing a slight constriction, passes into the nasal duct. To the outside of this is the antrum of Highmore, *h*, which has been opened up and is accessible to view. *Z*, suture between the superior maxilla and the malar bone. *F*, supraorbital foramen.

palpebral ligaments (fascia tarso-orbitalis). Hence, when the lids are shut the orbit is closed in all over anteriorly by fibrous structures which together form the *orbital septum*—namely, the two tarsi in conjunction with the fascia tarso-orbitalis and the two palpebral ligaments (Fig. 285).

The tarsus is called the cartilage of the lids. It is not, however, cartilage, but a very dense fibrillary connective tissue, in which are imbedded the *Meibomian glands*. These are elongated acinous glands which, lying parallel with each other, traverse the tarsus from its attached to its free

border. They are longest in the middle of the tarsus, where the latter attains its greatest height, and grow progressively shorter toward the edges of the tarsus (Fig. 285). In their essential character the Meibomian glands are nothing but large sebaceous glands. Like the latter they secrete sebum, which lubricates the edges of the lids. By this the overflow of tears over the free border of the lids is hindered; the closure of the palpebral fissure is rendered water-tight; and, lastly, the skin of the border of the lid is protected from maceration by the tears. Acinous mucous glands are frequently found near the convex border of the tarsus (Fig. 283, *w*, and Fig. 285).

At the free border of the lids there are found, in the vicinity of the cilia, hair follicles and the sebaceous *glands* (here called Zeiss's glands) connected with them. Moreover, close to the free border of the lids, sweat glands occur, the structure of which varies somewhat from that of the ordinary sweat glands, for which reason they are known as modified sweat glands or Moll's glands. They empty into the hair follicles of the cilia.

In accordance with its anatomical structure, the lid can be readily divided into *two parts*. The anterior or cutaneous portion contains the skin, together with the cilia and also the fibers of the orbicularis. The posterior or conjunctival portion consists of the tarsus with the Meibomian glands and of the conjunctiva. The two portions are joined simply by loose connective tissue, and so can be very readily split apart. For this purpose we need only make a stab in the gray line that runs between cilia on the one hand and orifices of the Meibomian glands on the other (Fig. 284, *i*). The splitting of the lid into its two layers forms an important part of many trichiasis operations.

583. Vessels of the Lids.—The *blood-vessels* of the upper lid arise from two arterial arches, the arcus tarseus superior and inferior (*as* and *ai*, Fig. 42), which run along the upper and lower margins of the tarsus. From them fine twigs are given off to all parts of the lid. The most vascular portions are the free border of the lid and the conjunctiva.

The veins of the lids are still more numerous and of wider caliber than the arteries. They form, beneath the upper and lower retrotarsal folds, a dense plexus, which even in the living subject can be seen in this situation shining through the conjunctiva of the fornix when the lid is everted. The veins of the lids in part empty into the veins of the forehead, in part into the branches supplying the ophthalmic vein. The latter set, in order to reach to the veins of the orbit, must pass between the fibers of the orbicularis. Hence, permanent contraction of the orbicularis, such as occurs in blepharospasm, may lead to engorgement of the veins, and consequently to œdema of the lids, a result which, in fact, we very frequently observe, especially in children with conjunctivitis eczematosa and coincident blepharospasm.

The *lymphatic vessels* of the lids are abundant, especially in the conjunctiva. Furthermore, lymph spaces of larger size (periacinous spaces) are found about the acini of the Meibomian glands. The lymph vessels of the lids run to the lymph gland in front of the ear, which, consequently, is often found to be swollen in the severer forms of inflammation of the lids and sometimes also in affections of the conjunctiva (e. g., conjunctivitis gonorrhoica).

584 Movements of the Lids.—These are performed in the following way: In *opening the eye* the upper lid is raised by the levator palpebræ superioris, while the lower lid sinks by its own weight, although it does so but very little [see also page 656]. Owing to the fact that fibers from the tendon of the levator run to it, the skin of the lids above the convex border of the tarsus is drawn quite far in between the eyeball and the upper margin of the orbit, at the same time that the upper lid is raised. In this way there is formed a furrow, over which the lax skin of the lid hangs down under the form of a fold (covering fold, *d*, Figs. 41 and 283). In many cases this is so large as to reach the free border of the lid, and cause disfigurement (ptosis adiposa; page 665).

With regard to the shutting of the eyes, we must distinguish between winking and tight closure of the lids. *Winking* consists in a quick contraction of the palpebral fissure, in which the lids do not come into perfect contact. It can be performed voluntarily, but usually results through reflex action, which is excited by the sense of dryness in the eye or by the presence of foreign bodies—dust, smoke, etc. It is effected by means of the trigemimus, which is the sensory nerve of the eye and its vicinity, and is hence rightly called the sentinel of the eye. The purpose of winking is to cover the surface of the eyeball with a uniform layer of the lachrymal fluid and thus both prevent the drying of the eye and also wash the dust off of it.

In *firm closure* of the lids, which usually is done voluntarily, the edges of the lid are brought into complete contact. This may be done gently as in sleep or forcibly as in the act of squeezing the lids together. In the latter case the skin about the lids is drawn toward the palpebral fissure and at the same time is thrown into numerous wrinkles. When the lids become closed in sleep the eyeball also performs a movement, rolling upward (Bell's phenomenon). Any one, when he is fighting against sleep and his lids are shutting together, can feel that the eyes are being drawn up as by an invisible force. In persons with thin lids (women and children) we can recognize the convex cornea through the upper lid, and determine that it is directed upward beneath the closed lids. It is still easier to do this in cases of staphyloma of the cornea. This behavior on the part of the eyeball is important, inasmuch as the protection of the cornea by the upper lid is thus provided for, even when the palpebral fissure is not completely closed in sleep. It is not till lagophthalmus reaches quite a high degree that a portion of the cornea remains constantly visible in the palpebral fissure; and this portion is, in fact, always the lowest part of the cornea, which consequently is most exposed to the danger of undergoing desiccation (keratitis e lagophthalmo; see page 277).

Winking is effected by the peripheral portion of the orbicularis, while in firm closure of the lids, the orbital portion of this muscle also comes into play.

[The upper lid is raised not only by the levator, but also by the musculus tarsalis superior (see page 652) and by the superior rectus. For the tendon of the latter is so solidly united with that of the levator by fascial bands that it may be said to have a common insertion with the levator in the tarsus of the upper lid. Hence, when the superior rectus contracts and raises the eye, it assists in lifting the upper lid and the retrotarsal fold at the same time. The same thing happens if the superior rectus retracts from any cause, e. g., as the result of a complete tenotomy producing a traumatic paralysis. The upper lid is then pulled up, and the eye consequently is wider open than its fellow. On the contrary, in an advancement, in which the tendon of the superior rectus and with it the fascial bands are brought forward, the upper lid is carried forward too, and the eye is 1 or 2 mm. less open than its fellow. The same thing happens in a complete paralysis of the superior rectus, not due to division of its tendon; the relaxed muscle allows the lid to droop somewhat, especially when the eyes are carried up.—D.]

[The lower lid is carried down not only by its weight, but also by the action of the musculus tarsalis inferior (supplied by the sympathetic) and by a slip which runs from the tendon of the inferior rectus directly to the tarsus. Hence, when the inferior rectus pulls the eye down it depresses the lower lid at the same time, and hence also, as in the case of the superior rectus, a complete tenotomy of the inferior rectus makes the palpebral fissure wider and an advancement of this muscle makes it narrower.—D.]

[By expansions from their tendons the external and internal recti also act upon the lids; so that when the eye is abducted the outer canthus is pulled outward, and when the eye is adducted the inner canthus is pulled back and in (Dwight).—D.]

I. Inflammation of the Skin of the Lids

585. In the skin of the lids we find almost all those diseases which appertain to the skin in general. With regard to them, therefore, reference must be made to the text-books on skin diseases. In this place only such affections of the skin of the lids will be considered as are of comparatively frequent occurrence in the lids, or which, in consequence of the peculiar anatomical structure of the latter, present some special features in their course and their result.

1. *Exanthemata*

586. Erysipelas.—Among the *acute* exanthemata erysipelas requires special mention. If this attacks the skin of the face, the lids participate very markedly in the inflammation, so that they are very greatly swollen, and the patient for several days together cannot open his eyes. When the swelling and infiltration are specially marked, the skin of the lids becomes gradually discolored and blackish, and at length to a large extent gangrenous (erysipelas gangrænosum). Not infrequently the erysipelatous process penetrates under the guise of a phlegmonous inflammation into the deeper parts, so that abscesses are produced in the lids or even in the orbit itself. In the latter case, implication of the optic nerve may occur, and, by transmission of the suppuration to the cranial cavity, meningitis may take place and lead to a fatal issue.

587. Herpes Febrilis.—Herpes febrilis forms on the lids small limpid vesicles which are usually situated in groups placed on a common, slightly

reddened base. After a few days they dry up without leaving any scar. Herpes febrilis is usually unilateral and develops in consequence of febrile infectious diseases, most often those of the respiratory tract. It is often associated with an eruption of vesicles on the cornea (page 285). Sprinkling the vesicles with a desiccant dusting powder suffices for the treatment.

588. Herpes Zoster.—Herpes zoster is an affection of the skin which consists in the formation of vesicles along the terminal expansion of a nerve. Among the cranial nerves the trigeminus is the one in whose area of distribution this affection occurs. The efflorescences are then found in the vicinity of the eye, for which reason herpes of the trigeminus is known as herpes zoster ophthalmicus or zona ophthalmica.

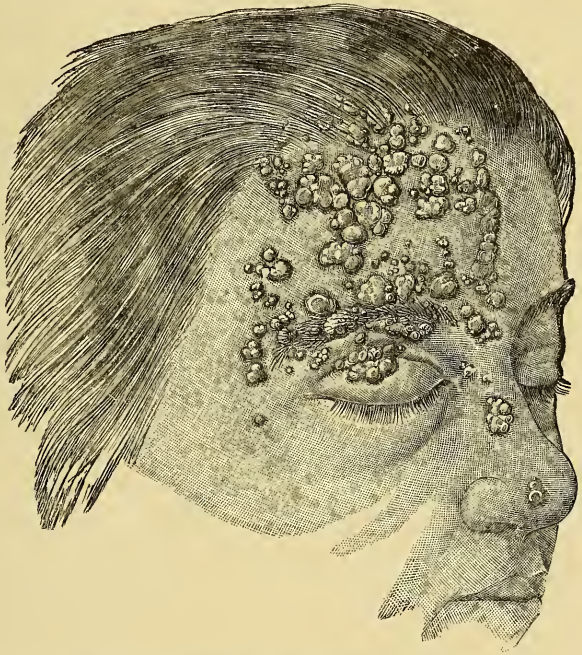


Fig. 286.—Herpes Zoster Ophthalmicus.

In a girl twenty years of age. Beginning of the disease five days previous. The vesicles occupy the region of distribution of the first branch of the trigeminus. This is the case even with those on the nose which extend down to the tip of the latter (corresponding to the distribution of the ramus naso-ciliaris of the first branch).

Violent neuralgic pains in the course of the trigeminus usually precede for some days the outbreak of herpes. Then the exanthem makes its appearance, with accompanying febrile symptoms—vesicles, which for the most part are arranged in groups, starting up upon the reddened skin. The vesicles most frequently occupy the region of distribution of the first branch of the nerve, so that they are found upon the upper lid, upon the forehead as far as the scalp, and also upon the nose (Fig. 286). When the district supplied by the second branch of the trigeminus is affected, the vesicles are situ-

ated upon the lower lid, over the superior maxillary region as far down as the upper lip, and over the region of the malar bone. Sometimes the terminal expansions of both branches are affected simultaneously, while it is extremely rare for the region of the third branch to be involved. It is a characteristic feature of the exanthem, which is almost always confined to one side, that the affection of the skin is sharply delimited at the middle line.

At first the vesicles contain a limpid fluid, which soon becomes cloudy and purulent, and finally dries up into a crust. If this is removed, an ulcer is found beneath it, a proof that the suppuration has penetrated into the corium. After the ulcer heals, cicatrices remain which are visible during the whole life, and by their characteristic arrangement render it possible to diagnosticate the previous existence of a herpes zoster even years afterward. By this formation of cicatrices the vesicles of herpes zoster are distinguished from those of herpes febrilis, in which the epidermis alone is detached by the fluid, so that they heal without leaving any trace of their existence behind.

The affection of the skin is very frequently complicated with an affection of the eye, consisting either of a keratitis or an irido-cyclitis. By the presence of such a complication the prognosis of herpes zoster is rendered essentially worse [see below].

An inflammatory affection of the trigeminus, which is located either in the trunk of the nerve itself or in the Gasserian or ciliary ganglion, lies at the bottom of herpes zoster ophthalmicus. By what means the inflammation of these structures is produced remains in most cases unknown. In some cases herpes has been seen to develop as a result of chilling of the body, head injuries, the use of arsenic, and poisoning by carbon monoxide gas.

The treatment of herpes zoster is purely symptomatic. We avoid opening the vesicles, as by doing so the raw surface in the skin would be exposed and pain would be excited. To prevent this we sprinkle the affected spots with dusting powder (rice starch) which causes the vesicles to dry up into crusts, beneath which the ulcers can heal undisturbed. The affection of the cornea or of the iris is to be treated according to the ordinary rules.

After the subsidence of *herpes zoster*, anomalies in the function of the trigeminus often remain; anæsthesia or neuralgia, both combined, persisting for a long time in the area supplied by the affected branches. The cornea, which even while the inflammation is still present is less sensitive than normal, usually retains this condition of diminished sensibility for a long time. The two following phenomena likewise must be referred to alterations in the nervous influence: The first consists in the abnormally low tension that the eyeball frequently shows when it participates in the inflammation; the second is the striking elevation of temperature of the skin upon the affected side, which not only is present while the inflammation is recent, but often lasts for quite a long time afterward.

It is said that the eye is implicated only in those cases of herpes zoster ophthalmicus, in which the naso-ciliary branch is involved (Hutchinson). The disease in the eye affects either the cornea or the iris.

The cornea may be implicated in various ways. In the first place, it may be im-

plicated through the breaking out upon it of herpes vesicles from which quite large ulcers may develop [see page 285]. In other cases deep infiltrates may form which do not undergo purulent disintegration, but are very slow in disappearing (keratitis profunda; see page 299). The cornea may also be affected indirectly, in that a paralysis of the trigeminus remains, and as a consequence of this a keratitis neuroparalytica is set up. Then I have seen two cases in which herpes was complicated with facial paralysis, and as a result of the latter, a keratitis e lagophthalmo developed. Iritis is often associated with these various diseases of the cornea, and iritis and irido-cyclitis may also develop as a result of herpes independently—i. e., without there being any simultaneous affection of the cornea at all [page 426]. Mydriasis and paralysis of the oculo-motor or abducens nerves also occur in consequence of herpes zoster.

589. Eczema.—Eczema of the lids occurs both in an acute and a chronic form. *Acute* eczema is often artificial, i.e., caused by the application of substances which the skin does not tolerate, such as tincture of arnica, blue ointment, adhesive plaster, etc.

At the outset, as long as the skin is simply red and swollen, the disease looks like erysipelas, but is distinguished from it by the fact that in erysipelas the skin is infiltrated throughout its entire thickness, and hence feels much thicker and firmer than in eczema.

Chronic eczema either develops from an acute eczema or is chronic from the start. It may be simply one of the evidences of an eczema that is distributed widely over the body, or it may be present in the lids alone. In the latter case it usually has a local cause, which is most often a wetting of the skin of the lids with the tears in chronic conjunctivitis, ectropion, affections of the lachrymal sac, etc. In these cases often only the lower lid is attacked.

Acute eczema is treated with compresses of aluminum acetate, and washing with water is avoided. Afterward when the swelling of the skin has gone down ointments are indicated. Of these we select either diachylon ointment (Hebra) or ointments made with zinc oxide or white precipitate (1 or 2 per cent) or with ichthyol (5 or 10 per cent). The ointments are spread thickly on pledgets of linen, which are laid on the closed lids and retained there by a bandage. In extensive eczema the entire face is covered with a linen mask smeared with ointment on the inside.

In chronic eczema the application of ointments or pastes is likewise indicated, and the skin at the same time is protected by them from the tears. In squamous eczema we use tar ointments and for washing the lids salicylic spirit.

Eczema of the lids is especially common in children, particularly under the form of moist eczema, which is called by the name of crusta lactea (milk crust, tetter). It forms the most frequent accompaniment of conjunctivitis eczematosa. The connection between eczema and conjunctivitis is either this, that both owe their origin to the same casual disease, scrofula, or else the eczema is the result of the conjunctivitis. For, as the latter is associated with profuse lachrymation, the lids are constantly moistened with the overflowing tears, and hence become eczematous. Besides, children are in the

habit of rubbing their eyes with their hands, so that all the parts about the eye are wet with the lachrymal fluid. The eczema requires treatment both on its own account and also because of any eczematous conjunctivitis that may chance to be present. The latter is cured much more quickly if at the same time the eczema of the skin is got out of the way—contrary to the popular belief which is disposed to take the opposite view. (We often hear the complaint, "The eruption which the physician has driven from the skin has broken out in the eye.") The treatment in this case too is performed by means of the ointments, above given. Another effective method of treatment consists in the application of a 5- to 10-per-cent solution of silver nitrate (see page 201).

Eczema at the border of the lid, being modified by the peculiar anatomical structure of the region, appears under a special form and will receive separate description later under the head of blepharitis ciliaris.

2. *Phlegmonous Inflammations of the Lids*

590. Under this head belong: 1. *Abscesses of the lids.* These originate most frequently after injuries. In other cases the affection starts from the bones; periostitis and caries of the margin of the orbit lying at the root of it. This is especially apt to be the case in scrofulous children, in whom, moreover, the carious diseases of the margin of the orbit are frequently referable to injury. Lastly, erysipelas not infrequently gives rise to abscesses of the lids if the inflammation penetrates from the skin into the deeper parts. 2. *Furuncles,* which, mainly occur in the region of the eyebrows. 3. *Anthrax pustule* (malignant pustule). This arises through a transfer, by a process of inoculation, of the poison of anthrax (the bacillus anthracis) from animals affected with anthrax to man. It is hence most frequently found in those persons who have to do with animals or the products obtained from them—e. g., in hostlers, shepherds, graziers, butchers, tanners, and furriers. In the Vienna clinics most of these patients come from Hungary. The disease sometimes terminates fatally.

The symptoms of the phlegmonous processes in the lids are marked inflammatory œdema and indurated infiltration in the skin of the lid or beneath it. Associated with this are swelling of the lymphatic glands in front of the ear and near the lower jaw, and fever and prostration. In the after-course of the disease disintegration of the infiltrated portion of the skin takes place, or, if the case is one of abscess, softening of the infiltrate sets in, with escape of the pus externally by its breaking through the skin. Not infrequently extensive gangrene of the skin of the lids occurs. The result of this is cicatricial shrinking of the lid in the course of healing and its consequent contraction, so that lagophthalmus or ectropion is produced. In both erysipelas and malignant pustule it is not uncommon for both lids to

be affected by the destructive process. This latter presents the peculiarity that even when it is of great extent it leaves exempt the free borders of the lids together with the cilia that they bear. This exemption is perhaps to be ascribed to the fact that of all portions of the lids the free border is most abundantly supplied with blood-vessels, and hence less readily falls a prey to necrosis. The preservation of the border of the lid is a very favorable circumstance for those cases in which a plastic operation upon the lids afterward becomes necessary, since the border of the lid can be used to skirt the edge of the implanted flap.

In cases of tuberculous disease of the orbital margin, the suppuration often occurs without inflammatory symptoms of any account and under the guise of a cold abscess.

Treatment follows the general rules of surgery. In abscesses of the lids an incision should be made as early as possible (that is, as soon as we are able to make the diagnosis), in order to prevent the extension of the suppuration into the deeper parts (orbit and meninges). However, in cold abscesses we may first make the attempt to cure the abscess together with the bone lesion by aspirating the pus with a syringe and then injecting some iodoform emulsion into the abscess cavity. When the skin of the lids is destroyed by inflammation, it is our business to strive to prevent as far as possible the subsequent contraction of the lids due to cicatrization. In large losses of substance in the lids it is best to refresh the edges of the two lids in isolated spots and unite them by sutures. As long as the palpebral fissure is kept closed in this way, lagophthalmus cannot develop, and the cicatrix that forms is broader. It is also advisable in such cases to graft bits of skin upon the granulating surface of the injured lids. In order not to endanger the result by a secondary shrinking of the newly formed cicatrix, the lids that have been thus artificially united are not separated again until some months after the cicatrization has been completed. If in spite of these measures, such a considerable contraction takes place, that lagophthalmus or ectropion is caused by it, the skin which has been destroyed must be replaced by blepharoplasty.

3. *Ulcers of the Skin of the Lids*

591. Ulcers are produced partly as the result of injuries (burns, the action of caustic substances, and contusion), partly spontaneously. Of the latter kind are the tuberculous (including as special forms scrofulous and lupous) ulcers and syphilitic ulcers. In children scrofulous ulcers are found not infrequently in conjunction with caries of the adjacent bone. Lupus is likewise of frequent occurrence in the lids, usually migrating to them from the neighboring regions (nose or cheek). From the lids it may pass over to the conjunctiva and even to the eyeball; and so, when lupus of

the face has lasted for a long time, considerable changes in the lids and eyeball are often found, which may even produce complete blindness.

The syphilitic ulcers of the lids are either examples of initial sclerosis or disintegrating gummata. Soft chancre also occurs on the lids.

[Chancre of the lid is usually found either in the intermarginal space or at the canthi or on the tarsal conjunctiva. It is distinguished from a hordeolum by its chronicity, the attendant enlargement of the preauricular or submaxillary glands, the presence of the spirochætæ, and the positive Wassermann reaction (Alter). Occasionally the cornea may be seriously involved. Very rarely double or even triple chancres are found (Finlay). Gummata form indolent tumors which may break down into indurated, painless ulcers or may be absorbed spontaneously (see page 676). Very similar tumors may be caused by excessive doses of the iodides (Stephenson). In the diagnosis of gummata the luetin test is particularly valuable, as it may be positive when the Wassermann test is not (Alter).—D.]

Among the ulcers of the skin of the lids must also be mentioned *vaccine ulcers*. The way in which they develop is that by some carelessness secretion is carried from children's vaccine pustules to the lids. They are found most frequently in women whose children have been vaccinated a short time before. They form pretty large, very coated ulcers, situated on the edges of the lids, and accompanied by considerable œdema of lids and even conjunctiva. To these symptoms are added swelling of the lymph gland in front of the ear, and sometimes even fever (cf. pages 210 and 275).

[**592. Blastomycetic Dermatitis.**—This is a disease which so far has been observed only in America, and mainly in the Mississippi valley. It begins as a papule, changing speedily to a pustule and then to a spreading, tumor-like mass, composed of irregular elevations and containing miliary abscesses, from which pus oozes spontaneously or under pressure. The disease has a serpiginous growth and may produce extensive destruction of the skin, but rarely, if ever, attacks the conjunctiva. It looks very like epithelioma, but is due to a peculiar yeast-like, budding organism, which can be obtained from the abscesses. Potassium iodide up to 10 gm. three times a day is almost a specific (Bevan) and the application of the X-ray is also beneficial (Fagin).—D.]

4. *Œdema of the Lids*

593. Œdema of the lids is of course not a disease but only a symptom yet as such it is so frequent and at the same time so conspicuous that it deserves quite a detailed description. Its development is favored to an unusual degree by the anatomical structure of the lid (see page 647): hence it is found not only in connection with every violent inflammation of the lids themselves or of the neighboring parts, but also in consequence of simple venous congestion or an altered state of the blood. In the former case we are dealing with *inflammatory œdema* (œdema calidum), in the second case with a *non-inflammatory œdema* (œdema frigidum). As long as the œdema is on the increase, the skin of the lid is found to be smooth and tense; but as soon as the œdema begins to decrease, this is at once made manifest by the formation of minute wrinkles in the skin of the lid—a phenomenon which is therefore of value as affording evidence that the process has already passed its acme.

Œdema of the lids often causes the patient more alarm than does the lesion which lies at the bottom of it, because he cannot open the swollen eye, and hence cannot see with it. And for the less experienced physician marked œdema presents difficulties,

inasmuch as it hinders the accurate inspection of the eyeball. If, in consequence, the physician gets but a transient view of the eye, or no view at all, he may easily make a false diagnosis, and may cause the patient great anxiety for what is perhaps an insignificant affection. For the benefit of the general practitioner, therefore, those affections which are associated with œdema of the lids will be enumerated in the following lines, and at the same time the symptoms will be given from which the diagnosis can be made.

The first thing to be done is to open the lids sufficiently in spite of the œdema, for which purpose we may with advantage use Desmarre's elevator [see Fig 2], especially in the presence of marked swelling or violent blepharospasm. We then see whether the conjunctiva is free from redness, and the eyeball itself is normal, not protruding, and freely movable; or whether, on the contrary, morbid changes can be made out to exist in these parts.

(a) Upon Separating the Lids the Deeper Parts Appear Normal

594.—It is necessary first to determine whether what we have before us is an inflammatory or a non-inflammatory œdema. The former is distinguished from the latter by the redness, the increased temperature, and not infrequently also by the sensitiveness to touch. Let us assume that we are dealing with an *inflammatory œdema.* In order to find out what affection lies at the bottom of it, we now try to ascertain whether in palpating the swollen part we do not come upon some one spot which is distinguished by greater induration and special painfulness.

1. If such a spot is found close to the free border of the lid, we are usually dealing with a *hordeolum.* In the very inception of this affection nothing besides the above-mentioned symptoms are noticeable. But, in the days immediately following, a yellowish point of discoloration is discovered either between the cilia, or, if we are dealing with a Meibomian stye, upon the inner surface of the lid.

2. If the indurated and sensitive spot occupies the internal angle of the eye, our first thought must be of an acute *dacryocystitis.* This diagnosis is confirmed, if, upon pressure in the region of the lachrymal sac, pus is evacuated from the puncta, or if the patient says that epiphora has for a long time preceded the inflammation. To be sure, a furuncle or a periostitis may also develop in the region of the lachrymal sac, but these cases, in comparison with the frequently occurring dacryocystitis, are extremely rare.

3. In œdema of the lids due to a *furuncle* or a *malignant pustule* there is felt, contrary to what takes place in erysipelas, a circumscribed, indurated, and painful nodule of considerable extent in the skin of the lid itself; while, if the infiltration lies deep in the tissues, we are dealing with a commencing *abscess* of the lid. In *periostitis* of the margin of the orbit the latter can be felt through the œdematous lid, and it is then found to be not sharp, but thickened and enlarged, and tender to the touch.

4. In *erysipelas* the redness and swelling of the lid are uniform. The skin itself when grasped between the fingers feels thicker and harder; while, on the other hand, circumscribed infiltration is absent. The swelling, as a rule, occupies both lids and also extends to the neighboring parts; and when we have had the case under observation for some time, we can see that the swelling migrates. If in the course of the inflammation an induration develops which can be felt to be deeply seated, it is a proof that the process has penetrated into the deeper tissues, and that an abscess of the lid is forming.

Cases of erysipelas sometimes occur which are very slight in intensity and extent, and present correspondingly insignificant inflammatory symptoms. Then only the lids themselves, and perhaps the dorsum of the nose, too, are swollen; these parts are not tense but of doughy consistence, and are scarcely reddened; and fever and pain are absent. The swelling disappears within a few days, and the skin then peels off, but it may also happen that the doughy swelling lasts for weeks or even months—a condition

of things that the dermatologists call by the name of erysipelas perstans. Such light cases of erysipelas usually recur, and thus acquire a resemblance to the cases of—

5. *Recurrent neurotic œdema* of the lids. In this a very marked œdematous swelling of the lids—a swelling, however, which is usually free from redness—suddenly makes its appearance, and then very rapidly—often within a few hours—disappears again. Frequently there are associated with it similar œdematous swellings in other parts of the body—e. g., on the lips, the trunk, or the extremities, more rarely in the larynx or pharynx. These transient attacks of œdema are referred to temporary disturbances in the innervation of the vessels (angioneuroses), and are allied to urticaria. They occur most frequently in women, and especially at the time of the menses.

6. Acute *eczema* of the lids is often associated with marked œdema of the latter, and in the first few days of its development may be hard to distinguish from an erysipelas.

Chronic eczema squamosum often also causes a moderate œdema of the lids in old people the skin of whose lids is lax, and it is then readily overlooked because the skin affection often manifests itself only by an insignificant roughness of the surface when stroked with the finger—the roughness being due to the slight desquamation.

7. Œdema of the lid due to *traumatism* is almost always accompanied by extensive hæmorrhagic suffusion of the lid, and from this fact can readily be recognized. If œdema of the lid develops in consequence of the sting of an insect, it is easy to make the diagnosis when we are able to discover the site of the sting.

Non-inflammatory œdema is met with as one of the symptoms of general œdema, as, for example, in heart disease, in hydræmia, and in nephritis. Not infrequently the lids are the very first part of the body in which these varieties of œdema show themselves, and thus give warning of the causal disease. In such cases the œdema of the lids sometimes appears under the guise of flying œdema (œdema fugax)—i. e., it comes suddenly and disappears again within a few days or even a few hours, only to return after a short interval of time.

A variety of œdema holding an intermediate position between the inflammatory and the non-inflammatory kinds, is that which is observed in connection *with blepharospasm* that has lasted a long time (especially in children with conjunctivitis eczematosa). This chiefly affects the upper lid, and is mainly referable to the compression of the palpebral veins, by the contracted orbicularis (see page 197).

(*b*) *Upon Separating the Lids Changes are Found in the Conjunctiva or Eyeball*

595. 1. Among affections of the *conjunctiva*, those associated with œdema of the lids are acute blennorrhœa and diphtheria, less frequently a violent catarrh, or, as above stated, a conjunctivitis eczematosa. The diagnosis is easily made, from the appearance of the conjunctiva and from the character of the secretion.

2. Violent *inflammations in the interior of the eyeball* lead to œdema of the lids; severe irido-cyclitis and acute glaucoma doing so to a less extent, *panophthalmitis* to a more considerable degree. In the latter disease, as in acute blennorrhœa, chemosis is also present. A confusion between the two diseases can, however, be readily avoided; since in panophthalmitis the purulent secretion in the conjunctiva is wanting, while a purulent exudate is visible in the interior of the eye (in the anterior chamber or in the vitreous). An important differential sign is the protrusion of the eyeball and the consequent diminution in its mobility in panophthalmitis, symptoms which are never present in acute blennorrhœa.

3. *Tenonitis, orbital cellulitis,* and *thrombosis of the cavernous sinus* share with panophthalmitis the symptoms of œdema of the lids, chemosis, and protrusion and immobility of the eyeball. These affections might hence be confounded with each

other and with panophthalmitis. From the latter, however, they are at once distinguished by the fact that in all three the eyeball itself, except for the œdema of the conjunctiva, looks normal in its anterior portion, while in panophthalmitis the suppuration in the interior of the eye is visible. For the differential diagnosis between the three affections first named, see § § 719 and 720.

A *tumor* developing in the depth of the orbit may also, along with the protrusion of the eyeball, cause œdema of the lid due to congestion. In this case, however, inflammatory concomitants are either slight or are absent altogether.

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596. Mortification of the Skin of the Lids.—This occurs under two forms, dry mortification (*necrosis*), and moist mortification associated with putrefaction (*gangrene*). Mortification develops from (1) intense inflammation of the skin of the lid itself. This is most frequently the case in erysipelas and also in anthrax; and, very rarely indeed, mortification occurs in variola. (2) Severe inflammation of the structures situated beneath the skin of the lids, such as the subcutaneous cellular tissue (in phlegmons) and even the conjunctiva itself (in gonorrhœal conjunctivitis and diphtheria). Cases of the latter kind are extremely rare, but I have actually seen all four lids become gangrenous in a child with ophthalmia neonatorum. The child not only escaped with its life, but even partly regained its sight. (3) Occlusion of the vessels of the lid by embolism (in pyæmia and sepsis) or thrombosis (in exhausting diseases). (4) Infection of peculiar character (hospital gangrene, noma). (5) Traumatisms, such as crushing, burning, corrosion and freezing (from too prolonged application of iced compresses).

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597. Dystrophy of the Skin of the Lids.—In old age the skin of the lids loses its elasticity and becomes wrinkled and lax. To a less extent this loss of firmness also affects the fascia tarso-orbitalis that lies beneath the skin. In that case this fascia and the skin with it are pushed forward by the orbital fat, especially in fat persons. Thus develops the well-known baggy bulging of the lower eyelids, which is commonly regarded by its possessor as an inflammatory swelling and is called by the name of "tear bags." In the upper lid it is especially the skin over the inner angle of the eye that protrudes in this hernia-like fashion.

A relaxed condition of the skin, which occurs in young persons as well as old is the *ptosis adiposa* of Sichel, which consists in the fact that the covering fold of the upper lid is of unusual size, so as to hang down over the free border of the lid in the region of the palpebral fissure. It was formerly assumed that this enlargement was caused by an excessive accumulation of fat in the covering fold, for which reason the name of ptosis adiposa was given to it. Its true cause, however, is that the bands of fascia connecting the skin with the tendons of the levator and with the upper margin of the orbit are not rigid enough; consequently the skin is not properly drawn up when the lid is raised, but hangs down in the form of a flabby pouch (Hotz).

A condition differing from ptosis adiposa is *blepharochalasis* (χάλασις, relaxation). In this the skin of the upper lid becomes so thin that it lies in countless little wrinkles, and looks like rumpled tissue paper. Moreover, owing to the dilatation of numerous small superficial veins, it acquires a red appearance. In consequence of its relaxation and the looseness of its attachment it hangs down like a pouch; while the border of the lid itself is scarcely lower than usual. This change occurs in cases in which there have been frequent antecedent œdematous swellings of the lid (e. g., it occurs after recurrent neurotic œdema of the lid, see page 664), as a result of which the skin is stretched and loses its elasticity.

Except for the disfigurement they cause, neither ptosis adiposa nor blepharochalasis entails any disagreeable symptoms. They can be removed by ablating the

excess of skin, and attaching the skin to the upper border of the tarsus by Hotz's operation, so as to prevent its drooping (see § 831).

598. Other Cutaneous Affections.—*Elephantiasis* affects the lids under the form of a monstrous thickening, especially pronounced in the upper lid which hangs down over the lower and upon the cheek, and which, on account of its weight, cannot be raised, and thus renders vision with the eye so covered impossible. Elephantiasis usually develops as a result of repeated inflammations of the lids. The treatment consists in excision of the skin to a sufficient extent for the lid to regain approximately its normal dimensions.

Under the name of *chromidrosis*[3] is denoted that rare affection in which the sweat from the skin of the lids is colored. As a result of it blue spots come out upon the lids which can easily be wiped away with a cloth that has been dipped in oil, although in a short time they make their appearance again. This disease is said to occur especially in women. A large number of the known cases must probably be referred to simulation—i. e., to the intentional application of some blue coloring matter to the lids.

II. Inflammation of the Border of the Lids

599. Varieties.—The free border of the lid is simply a part of the skin of the lid, which, however, is distinguished by many anatomical peculiarities such as its cilia with their hair follicles and glands, its particularly abundant vascular supply, etc., so that its diseases bear a special stamp. Affections of the borders of the lids are among the most frequent of all diseases.

Hyperæmia of the border of the lid manifests itself by the reddening of it, so that the eyes look as if rimmed with red. It occurs in many people in consequence of insignificant injurious influences, such as prolonged weeping, great straining of the eyes, staying in vitiated air, a wakeful night, etc. This is especially true of persons with a delicate skin, who at the same time have a light complexion and blonde or reddish hair. In many of these persons the hyperæmia of the lids is present all the time, and sometimes lasts for their whole life. With respect to the troubles that it causes, and also with respect to its treatment, the same statements hold good that will be made in speaking of blèpharitis.

Inflammation of the border of the lids (blepharitis ciliaris or blepharoadenitis[4]) appears under the two following principal forms:

1. *Blepharitis squamosa.* In this the skin between the cilia and in their vicinity is covered with small, white or gray scales like the dandruff upon the scalp, or, as some have said, the border of the lids looks as though strewn with bran. If the scales are removed by washing, the skin beneath them is found to be hyperæmic but not ulcerated. Upon removing the scales, some cilia usually fall out—a proof that they are less firmly attached than usual; but, as their follicles are not injured, they grow again afterward.

A sub-variety of blepharitis squamosa, which is of less frequent occurrence, appears under the following form: The border of the lids is covered with yellow crusts, which are sometimes rigid, sometimes flexible and fatty

[3] From χρῶμα, color, ἵδρωσις, sweating.
[4] From βλεφαρον, lid and ἄδην, gland; i. e., inflammation of the glands of the lid.

(like wax or honey). When they are removed, no ulcers are found beneath them, but simply reddening of the skin of the lid. The yellow crusts are therefore not inspissated pus, but simply the excessively abundant secretion of the sebaceous glands, which has solidified in the air into yellow crusts.

2. *Blepharitis ulcerosa.* In this form also the border of the lid is covered with yellow crusts; but, after washing them off, we find not merely a hyperæmia of the skin but ulcerative processes as well. Thus we see here and there in spots small yellow elevations, from the center of which rises a cilium. These are little abscesses, which have originated from suppuration of a hair follicle and of the sebaceous gland belonging to it. With these we find little excavations—that is, ulcers which have been formed out of small abscesses that have opened. Again, in other spots we notice small scars, the remains of similar ulcers. At the site of the scars the cilia are permanently deficient, because their hair follicles have been destroyed by suppuration. Since new hair follicles are constantly being transformed, one after another, into abscesses, the row of cilia, when the process has kept up for a long time, becomes more and more thinned out; the cilia that are still present are arranged in separate groups, which for the most part are glued together into tufts by the dried secretion. Blepharitis ulcerosa, accordingly, is distinguished from blepharitis squamosa by its deeper situation and the purulent character of the inflammation. It is hence to be regarded as the more serious of the two forms, the one in which both the inflammatory symptoms are more pronounced, and permanent sequelæ, particularly destruction of the cilia, remain.

For the correct *diagnosis* of blepharitis the crusts covering the border of the lid must be removed, in order to determine the state of the skin beneath them. If the skin beneath the crusts is normal, we are not dealing with blepharitis at all, but with a disease of the conjunctiva, the dried secretion from which forms the yellow crusts. In blepharitis the skin is at least found to be reddened (in blepharitis squamosa), or it is covered with ulcers (in blepharitis ulcerosa). In the vicinity of the ulcers the skin of the palpebral border not infrequently is thickened by hypertrophy of the papillæ so that warty excrescences are formed, which are painful and bleed readily, and which must be removed.

600. Symptoms and Course.—The *annoyance* suffered by the patient is slight in the lightest cases of blepharitis, so that many patients visit the physician more on account of the disfigurement due to the reddened border of the lids than on account of any distress they experience. But in most cases the patients are annoyed by the increased sensitiveness of the eyes which water readily, especially during work and in the evening, are sensitive to light, heat, and dust, and tire quickly. In the morning the lids are stuck together.

Blepharitis is distinguished by its eminently chronic *course*, which often extends over a series of years. In young patients the disease often disappears of itself, when they grow up; in others it continues during the whole life. Proper treatment always produces considerable improvement, or even

effects a cure, which latter, however, is in most cases not lasting, as after the discontinuance of the treatment the disease usually returns; a permanent cure is obtained in only a few cases.

After lasting some time blepharitis entails a series of *sequelæ*, which to a certain extent react in their turn upon the blepharitis and render it worse. These are—

1. *Chronic conjunctival catarrh.* This is the constant concomitant of blepharitis, the annoyance produced by which is in no small part dependent upon it.

2. Blepharitis ulcerosa leads to permanent *destruction of the cilia.* This may go on till nearly all the cilia are lost. In that case there are found upon the border of the lid a few scattered, minute, and abortive hairs. This condition, called *madarosis*,[5] produces marked disfigurement. As soon as all the cilia have been destroyed, the blepharitis ceases of itself, since there are no longer any hair follicles to undergo suppuration.

3. By the traction produced by scars which remain after suppuration of the hair follicles, neighboring cilia may be given a false direction, so as to turn backward toward the cornea (*trichiasis*).

4. *Hypertrophy* of the border of the lid may develop in consequence of its being constantly congested and swollen by inflammation. The lid is then found to be thicker and more misshapen at its free border, and drooping in consequence of its weight (*tylosis*[6]). This change affects mainly the upper lid.

5. The lower lid very often undergoes, as a result of blepharitis, a change of position under the form of *ectropion.* This develops in the following way: Owing to the formation of the scars, the conjunctiva is drawn a little forward over the border of the lid. The border of the lid then looks as if it had a rim of red conjunctiva about it, and the posterior margin of the lid, which before was sharp, is now rounded off, and can no longer be distinctly made out. In consequence of this change of form, the borders of the two lids no longer fit exactly to each other when the latter are shut together. Furthermore, on account of the absence of its sharp posterior margin, the lid is no longer perfectly applied to the eyeball, and a shallow groove remains between the eyeball and the border of the lid (*eversion of the border of the lid*). In common with the border of the lids, the puncta are also turned forward so as no longer to dip into the lacus lacrimalis (*eversion of the puncta*). Thus the conduction of tears into the lachrymal sac is interfered with, so that epiphora develops. A portion of the tears runs down over the lower border of the lid upon the skin of the latter, which because of this continual wetting becomes reddened, excoriated, and even eczematous; consequently it loses its pliability and becomes gradually contracted. In this way the lower lid is drawn farther and farther away from the eyeball, so that an ectropion of the entire lid is gradually developed from the eversion of its border. At the same time the lachrymation also constantly increases, and

[5] From μαδᾶν, to melt away, to fall off. [6] From τύλος, a callous spot.

this in turn reacts injuriously upon the blepharitis, the border of the lid being irritated to the point of inflammation by the tears that are constantly flowing over it.

601. Etiology.—The causes of blepharitis are either of a general or a local nature.

The *general causes* lie partly in the constitution of the patient, partly in external injurious influences. In the first category are to be mentioned anæmia, scrofula, and tuberculosis, which particularly in children and young people furnish a frequent cause of blepharitis. When with increasing age the constitution improves, the blepharitis also disappears. In many families blepharitis is hereditary, being a kind of family disease. Among external injurious influences are to be considered all those which are known to be also causes of chronic conjunctival catarrh (see page 148). Among these belong vitiated air, smoke, dust, heat (for example in the case of stokers), staying up late at night, etc. Blepharitis produced by general causes is always bilateral.

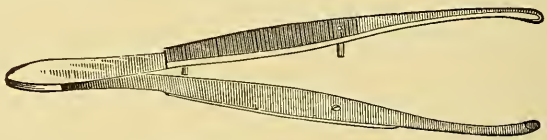
Among the *local causes* of blepharitis the most frequent are chronic inflammations of the conjunctiva (chronic catarrh, conjunctivitis eczematosa, and trachoma) and epiphora. The latter excites inflammation of the border of the lid by keeping it continually wet. The epiphora may be caused either by increased secretion of tears or by interference with their discharge into the nose. The former is the case, for example, in conjunctivitis eczematosa, which is characterized by profuse lachrymation. Since in this case the inflammation of the conjunctiva and the scrofulous diathesis of the patient act both at the same time to favor the development of blepharitis, it is easy to understand why conjunctivitis eczematosa should so exceedingly often be found combined with blepharitis.

Epiphora may, however, also be produced by interference with the conduction of tears, as a result, for instance, of an affection of the lachrymal sac; in this case the blepharitis is found only in that eye in which the affection of the lachrymal sac exists. Hence the rule is in unilateral blepharitis to examine the lachrymal sac at once, just as, on the contrary, in bilateral blepharitis we shall first have to look for a general condition as the cause of it. Another cause of interference with the conduction of tears, and hence also of blepharitis, is imperfect closure of the lids due to ectropion, to paralysis of the facial nerve, to congenital and acquired contraction of lids, etc.

Since the border of the lids is simply a modified portion of the external skin, it would be proper to consider the affections of it from the *dermatological* standpoint—that is, to compare them with analogous affections of the skin. From this point of view blepharitis squamosa might be regarded as seborrhœa. That form which is associated with furfuraceous scales would correspond to seborrhœa squamosa or seborrhœa sicca of the skin, which we most frequently observe, under the guise of increased formation of scales, upon the scalp. The form of blepharitis squamosa which is characterized by yellow, fatty crusts, is probably identical with the seborrhœa oleosa, which is

likewise found upon the scalp, particularly in children, and in this situation is known under the names of scurf or scall. In blepharitis ulcerosa it is a case of eczema, which, on account of the hairy character of the border of the lid, is associated with suppuration of the hair follicles, such as is also the case in other hairy portions of the skin. For the inflammation creeps from the epidermis into the hair follicle, and the staphylococci which enter the gland from its orifice, are what cause the suppuration. If the hair follicle alone suppurates, the small pustules, such as are ordinarily found, develop on the margin of the lid. But if the inflammation makes its way from the interior of the hair follicle to the surrounding connective tissue, so that the latter also suppurates, a somewhat larger circumfollicular abscess is formed. This corresponds to the acne pustule of the skin and when occurring on the lid border is called hordeolum. A genuine sycosis of the lid border (trichophytia) also occurs, although very rarely (Herzog). (For sportrichosis, see page 210.)

602. Treatment.—The treatment of blepharitis must take account both of the causal indication and also of the local changes. Regard to the causal indication requires the improvement of the patient's constitution and of the hygienic conditions under which he lives. In most cases, it is true, the object aimed at cannot be attained, owing to external circumstances. Local causes of blepharitis, such as lesions of the conjunctiva and the lachrymal sac, lagophthalmus, etc., are to be removed as far as possible. In the treatment of the diseased border of the lids themselves ointments play the



[Fig. 287.—Cilium Forceps.—D.]

chief part. Their action is to be principally attributed to the fat they contain. This softens the scales and crusts and thus facilitates their removal, and also prevents the occlusion of the orifices of the palpebral glands; it renders the skin more pliable, and protects it from being wet by the overflowing tears. We must, therefore, select as the basis of the ointment a soft pliable fat, which also should be somewhat hydrous so that by the evaporation of its water the ointment may produce a refrigerant action. The unguentum emolliens [cold cream] and lanolin fulfill these requirements; the latter should be mixed with an equal amount of vaselin to make it more pliable. Of medicaments added to the ointments the mildest are boric acid (2 per cent), noviform (2 per cent), white precipitate (1 or 2 per cent), and ichthyol (2 to 5 per cent); rather more irritant in action are salicylic acid, resorcin, and yellow precipitate (all in a strength of from 1 to 2 per cent). The way to apply the lid ointment is for the patient to rub it with the finger on the closed interpalpebral fissure before going to bed. Before he does this the crusts and scales that stick to the edge of the lids should be removed as carefully as possible by washing with lukewarm water or with milk so that the ointment shall come in contact with the skin itself. This cleansing is to

be done again in the morning after washing off the remnants of the ointment. The physician must insist very particularly upon this point, since the act of cleansing the lids is often painful, and hence, especially in children, is frequently not performed with sufficient care. In blepharitis squamosa the use of a tar soap or salicylic tincture is serviceable for cleansing the lids each morning. When by means of this treatment we have finally succeeded in bringing the border of the lids back to their normal state, we continue the use of the salve for some time longer, as otherwise the blepharitis will very soon recur. [A serviceable procedure is the following. Close the eyes gently. Make a good lather with any good soap or shaving cream and fill the eyelashes with it as if to shave them, grasp the eyelashes with a damp cloth held between thumb and finger, and with this stroke them out so as to remove all lather, scales, and loose lashes. When the lashes are clean, dry them, then rub a very little of the ointment (preferably a 1-per-cent ointment of yellow oxide of mercury) gently into the roots of the lashes and edges of the lids. Wipe off all excess of ointment. Do this twice a day (cf. also remarks in fine print below.—D.]

In blepharitis ulcerosa, in addition to the employment of the ointment, the abscesses which form must be opened every day, and the cilia that project from them must be epilated. For this purpose we make use of the cilium forceps—that is, of a forceps with broad rounded ends. The healing of the ulcers can be accelerated by touching them lightly with a pointed stick of nitrate of silver.

[The *removal of crusts* from the lids and from the cilia is important not only for diagnosis but for treatment. Crusts and scales may be loosened and teased off by stroking the cilia with a cotton swab wet with hydrogen peroxide.—D.]

In the treatment of blepharitis the mistake is very frequently committed of prescribing highly *irritant ointments*, by which the inflammation is simply aggravated. If we select as the basis of the ointment the unguentum emolliens, we must take care that the ointment is prepared fresh from time to time, as otherwise the fat would become rancid and cause irritation. In severe cases of blepharitis ulcerosa it is advisable to smear the ointment thickly upon a pledget of linen and apply it over night to the eyes by means of a bandage. In this way the ointment acts in a much more penetrating way than if it were merely smeared upon the borders of the lids. This procedure is particularly indicated when the blepharitis is caused by congenital shortness of the lids, because here the closure of the palpebral fissure during sleep is at the same time insured (see page 687).

In many cases of blepharitis ulcerosa, particularly if *tylosis* is present at the same time, we will fail to accomplish our object until we have epilated all the cilia. We do this in several sittings, and subsequently also remove the cilia that grow in afterward, keeping on in this way until the border of the lid once more looks perfectly normal. We need not fear that the cilia, even if we have epilated them ever so often will ultimately fail to grow in again. For the tylosis itself, massage of the lid, with the aid of the white-precipitate ointment, does good service. This acts partly by exciting resorption, partly because it helps to remove mechanically the contents of the palpebral glands, and thus prevents their occlusion.

The hair-follicle mite, acarus or demodex folliculorum, is found very often in the hair follicles of the cilia, and, it is said, may very frequently give rise to blepharitis (Raehlmann).

Not to be confounded with blepharitis is the condition called *phthiriasis palpebrarum*—i. e., the presence of crab lice (phthirius inguinalis or pediculus pubis) upon the eyelashes. Here the borders of the lids look strikingly dark. Upon more careful inspection we discover as the cause of the discoloration the black nits of the crab lice sticking fast to the cilia; sometimes, too, a grown specimen is found lodged between the cilia. The disease, which is rare and which is found almost exclusively in children causes trouble only from the itching it produces. It is easily cured by means of blue ointment, which, when rubbed into the borders of the lids, kills the parasites.

III. Diseases of the Palpebral Glands

603. The glands which here come under consideration are the glands of the hair follicles of the cilia (Zeiss's glands) and the Meibomian glands. The affections of the former have already been treated of in part under the head of blepharitis, a disease which presents a diffuse inflammation extending over the whole border of the lid, and in which the hair follicles are implicated. To be distinguished from this is the isolated inflammation which is limited to one or a few of these glands, and forms an independent affection. If such an inflammation develops as an acute affection, it is known as hordeolum; if as a chronic affection, it is known as chalazion. From these, again, are to be distinguished the cases of simple occlusion of the glands with inspissation of their contents but without inflammation—cases which form the starting point of the infarcts in the Meibomian glands.

1. *Hordeolum*[7] (*Sty*).

604. There are a hordeolum externum and a hordeolum internum.

Hordeolum externum [or hordeolum zeissianum] is produced by suppuration of one of Zeiss's glands. There is first noticed an inflammatory œdema of the affected lid, which in severe cases may even extend to the conjunctiva bulbi. Upon careful palpation there is discovered in the swollen lid a spot which is distinguished by greater resistance and by special sensitiveness to touch. It lies near the border of the lid, and corresponds to the inflamed gland. In the next day or two the swelling at this spot increases, and the skin over it grows red, afterward shows a yellowish discoloration, and finally perforates near the border of the lids with a discharge of pus. After the evacuation of the pus the inflammatory symptoms rapidly abate, the small abscess cavity soon closes, and the entire process comes to an end. In spite of the fact that the duration of the disease is but a few days, the affection itself is still very burdensome to the patient on account of the pain, which is often considerable, in the tense and greatly swollen lids. In addition to this many persons have a number of repeated attacks.

Hordeolum internum is much rarer than hordeolum externum. It consists in a suppuration of one of the Meibomian glands, and is hence also called hordeolum meibomianum. The course of the disease is, on the whole, the same as that of the hordeolum externum; but as the Meibomian glands

[7] From *hordeum*, barley. [The proper classical form is *hordeolus*.—D.]

are larger than those of Zeiss, and are enveloped in the firm connective tissue of the tarsus, the inflammatory symptoms are more violent, and it takes a longer time for the pus to be evacuated. The pus at first, as long as it is shut in in the affected glands, appears upon eversion of the lid as a yellowish spot shining through the conjunctiva. Afterward it breaks through the conjunctiva or is discharged through the orifice of the gland. Perforation through the skin occurs only as an exception, in contradistinction to hordeolum externum, in which this is the rule.

Hordeolum externum and internum are essentially the same process—i. e., they are both an acute suppuration of a sebaceous gland, for the Meibomian glands are nothing but modified sebaceous glands. A hordeolum is analogous to acne of the external skin. The violent inflammatory symptoms, and particularly the marked œdema, which distinguish a hordeolum from ordinary acne pustules of the skin, are caused by the peculiar anatomical structure of the lids, which especially predisposes the latter to inflammatory swelling.

Hordeolum is found principally in young people, particularly if they are of anæmic or scrofulous constitution and at the same time suffer from blepharitis. The latter, by causing accumulation of scales and crusts on the border of the lids, favors multiplication of the bacteria (staphylococci) that are always present there, which can thence readily penetrate into the orifices of the glands. [Repeated crops of styes often indicate systemic disturbance, especially one due to digestive or menstrual disorders.—D.]

The *treatment* of hordeolum in the beginning of the disease consists in the use of moist warm compresses, which are applied to the lids in order to convert the hard infiltrate more rapidly into pus. When the yellow color of the pus is visible beneath the skin or the conjunctiva, the abscess may be opened by a small incision, and thus the duration of the inflammation may be shortened by several days. The prime means for avoiding a recurrence of the hordeolum is the treatment of any blepharitis that may be present. [Yellow oxide ointment is, hence, a useful application for this purpose. Sometimes calx sulphurata given every three hours in doses of gm. 0.015 seems to abort a forming hordeolum. If there is a tormenting succession of hordeola, treatment with an autogenous vaccine prepared from the pus of the hordeola themselves may be tried.—D.]

2. *Chalazion*[8]

605. Chalazion is a *chronic* affection of the Meibomian glands. It forms a hard swelling which develops very gradually in the lid. In many cases this occurs without any inflammatory symptoms whatever, so that the swelling is not noticed by the patient until it has become quite large.

[8] From χάλαζα, hail.

In other cases, however, there are moderate inflammatory concomitants, which, nevertheless, are insignificant in comparison with those which accompany a hordeolum. The tumor keeps constantly enlarging for months until it becomes as big as a pea or bigger; it then bulges the skin far enough forward to produce a perceptible disfigurement of the lid. Upon palpating the tumor we can make out that it is pretty resistant, and that it is intimately connected with the tarsus, while the skin lying over it can be displaced from side to side. Upon everting the lid we find the conjunctiva over the tumor reddened, thickened, and somewhat protruding. Later on, the tumor assumes a grayish look as seen through the conjunctiva, and ultimately the latter is perforated; then a viscid, rather turbid fluid flows out, which represents the central softened portions of the tumor. But the main portion of the latter, consisting of spongy granulations, remains behind, for which reason the tumor after it has opened does not at once disappear completely. On the contrary, it diminishes very gradually in size, and meanwhile it is not uncommon for the granulation masses to project like a fungoid growth through the perforation in the conjunctiva. It requires months more for the tumor to disappear completely.

Chalazion shares with the hordeolum internum its situation in the Meibomian glands, but is distinguished by the character of the process. Hordeolum is an acute inflammation, which goes on to suppuration and is over in a few days. Chalazion is a chronic disease, which does not lead to suppuration but to the formation of granulation tissue, and lasts for months or even years.

Chalazion affects adults more frequently than children. Not infrequently several chalazia are found at once in the same patient. A chalazion annoys the person who has it by the disfigurement it produces, and also by the condition of irritation which it keeps up in the eye. This condition of irritation is partly the result of the chronic inflammation of the lids, partly the result of the mechanical injury done to the eyeball by the uneven and bulging conjunctiva covering the inner surface of the tumor.

The older physicians considered a *chalazion* as a hordeolum which had become hardened—i. e., had not gone on to suppuration—a view which is still at present widely diffused among the laity. Others supposed that the chalazion was a simple retention cyst of the Meibomian glands, and analogous to the atheromata of the sebaceous glands. Such retention cysts do occur, but they are rare, and are essentially different from chalazia. In a chalazion there is a peculiar chronic inflammation, which produces not pus but granulation tissue, and which is probably caused by a micro-organism differing from the ordinary pus cocci. Microscopic examination of a chalazion shows that first the epithelium of the acinus proliferates and an inflammatory infiltration is produced in the surrounding tissue of the tarsus. The latter process soon predominates, so that both gland acini and tarsus tissue ultimately are lost in the overgrowth of small cells (Fig. 288). This cell growth forms a soft tissue of the nature of granulation tissue, and, like it, containing giant cells. In the granulation tumor are found amorphous flakes representing remains of the inspissated contents of the acini; and it is inclosed without

by a capsule of connective tissue. The way in which the latter develops is that the tissue surrounding the growing tumor is continually compressed and condensed by it. Finally, the central portions of the granulation tumor, being very poor in vessels, break down by a sort of mucilaginous softening, so that a cavity filled with a turbid liquid forms in the center of the growth.

Horner has called attention to the analogy between chalazion and acne rosacea of the skin. In the latter subaceous glands play the same part that the Meibomian glands do in chalazion.

In old chalazia, where perforation has not occurred, the entire contents sometimes are liquefied. The chalazia are then transformed into a sort of cyst with thick envelope and turbid, mucilaginous contents. Chalazia which develop on the excretory duct of a Meibomian gland assume a special appearance. They are situated near the free border of the lid, from which they project like a nipple, while on their posterior side they are flattened out by the counter-pressure of the eyeball. If they injure the eye mechanically, they are to be removed by albation.

Sometimes a chalazion perforates the tarsus not behind but in front. Then the granulation tissue grows out into the subcutaneous cellular tissue and forms a soft flat tumor situated near the lid border and extending alongside of it. To this tumor (chalazion externum) the skin, which shows a reddish or bluish discoloration, is adherent. The tumor remains unchanged for months. To remove it we must incise the skin and curette out the soft tissue beneath.

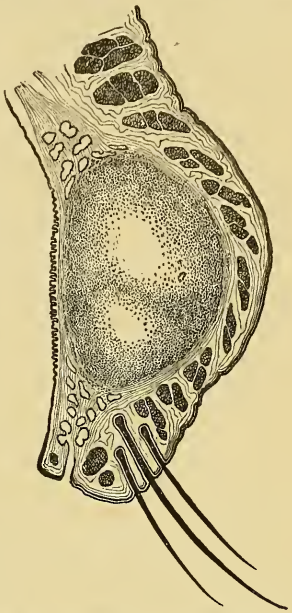


FIG. 288.—CHALAZION. VERTICAL SECTION THROUGH THE UPPER LID. Magnified 4 × 1.

The chalazion has developed in about the middle of the highest point of the tarsus, and, as it projects farther forward than backward, it produces a crooking forward of the lid. The swelling consists of granulation tissues with giant cells, one of which is so large as to be actually recognizable with the naked eye in the section. It lies near the anterior wall of the larger one of the two cavities that have been produced by mucilaginous softening of the granulation tissue. The growth is surrounded by a thin capsule. Upon its anterior aspect it is covered by the skin of the lids, which is thinned, as can be seen from the way in which the cross section of the muscular bundles of the orbicularis are narrowed. The conjunctiva over the swelling, owing to papillary proliferation, is thicker and uneven. Above and below the swelling are unchanged acini of Meibomian glands.

It not infrequently happens that persons who have never suffered from chalazia begin all of a sudden to have one after another. New chalazia keep developing at intervals of one or more weeks, manifesting their presence each time by renewal of the slight inflammatory symptoms. Finally, one or more chalazia are found in each of the four lids. In particularly bad cases actual degeneration of the lids, especially the upper, takes place. The lids are thickened, so that they can hardly be everted. In one case which I observed the lid had become a centimetre thick. The skin of the lids forms nodular projections, but can be displaced on its bed and is not essentially altered. The conjunctival surface of the lids, on the contrary, appears uneven, nodular, and reddened, velvety in some spots, in others gray and translucent or perforated by sprouting granulations. In extreme cases of this sort we might at first be disposed to suspect tarsitis or a neoplasm. In operating upon such cases we see that the entire tarsus has disappeared in a spongy and partially softened granulation tissue.

Treatment.—Quite small chalazia are best left alone. Larger chalazia are removed by an operation, in order to do away with the disfigurement and also with the irritation of the eye. [See § 825.]

3. *Infarcts in the Meibomian Glands*

606. In elderly people we frequently see, upon everting the lids, small bright-yellow spots beneath the conjunctiva. These are the inspissated contents of the Meibomian glands, which accumulate in their excretory ducts or their acini and distend them. These infarcts usually cause no disturbance. But sometimes they are transformed by the deposition of lime salts into hard, stony masses (lithiasis) conjunctivæ[9]. These bulge the conjunctiva forward and even perforate it with their sharp edges, which then cause mechanical injury to the eye. In this case they must be removed from their bed, after an incision has been made in the conjunctiva, and thus be got rid of.

The *infarcts* of the Meibomian glands should not be confounded with the much more frequently occurring concretions that develop within new-formed glands in the conjunctiva of the tarsus. These also appear under the guise of yellow spots, but are more superficial (see page 134).

607. Affections of the Tarsus.—The tarsus is implicated not only in the diseases of the Meibomian glands, but also in those of the conjunctiva. This is particularly true of trachoma and of amyloid degeneration of the conjunctiva. In the former we can often feel, when we evert the upper lid, that the tarsus has become thicker and more unshapely. This depends upon its inflammatory infiltration, which subsequently leads to atrophy and distortion through cicatricial contraction, and which must therefore be looked upon as the chief cause of trichiasis. In tarsal cartilages that have undergone this change, the Meibomian glands are also in great part found to be destroyed (see page 169). In amyloid degeneration of the conjunctiva the tarsus itself falls a victim to the same degeneration, so that it is transformed into a large, unshapely, friable structure (see page 207).

There is a primary affection of the tarsus (*tarsitis syphilitica*) which occurs as a consequence of syphilis. This develops very gradually, and as a rule without any notable pain. When it has reached its acme we find one or both lids of the same eye greatly enlarged and the skin of the lid tense and reddened. Upon palpation we can convince ourselves that the cause of the enlargement lies in the tarsus, which can be felt through the skin as a thick structure of cartilaginous hardness and ungainly form. The swelling of the tarsus is usually so great that the lid can no longer be everted. The enlarged tarsus consists, as is evident when it is cut into, of a lardaceous, bloodless tissue. The cilia upon the affected lid fall out, and the lymphatic gland in front of the ear upon the same side swells up. After the swelling has been for weeks maintained at the same height, it disappears very slowly again until the tarsus has reached its former volume, or has even, in consequence of atrophy, fallen somewhat below it. It takes several months for the disease to run all through its course. Tarsitis makes its appearance in the third stage of syphilis, and is accordingly to be looked upon as a gummatous infiltration of the tarsus [cf. page 662].

IV. Anomalies of Position and Connection of the Lids

1. *Trichiasis and Distichiasis*

608. *Trichiasis*[10] consists in a distortion of the cilia, which, instead of looking forward, are directed more or less backward so as to come into contact with the cornea. This anomaly of position either affects all the cilia

[9] From λιθος, a stone. [10] From θριξ, hair.

or only those which jut out furthest back; it may also extend over the whole length of the border of the lid, or be present over a part of it only (total and partial trichiasis). The inverted cilia are seldom normal, being for the most part stunted, and consisting of short stumps or of minute, pale, and scarcely visible hairs.

Trichiasis causes a continual irritation of the eyeball, due to the action of the cilia; there are photophobia, lachrymation, and the feeling as if a foreign body was in the eye. The cornea itself suffers more considerable injury. Superficial opacities are produced in it, since the epithelium, in consequence of the constant irritation, undergoes a sort of callous thickening, and thus in a way protects the cornea against the effect of the external injury. In other cases deposits like pannus, or ulcers of the cornea are formed. It not infrequently happens that persons are tormented by frequent recurrences of corneal ulcers, until at length the physician discovers one minute cilium which is directed against the cornea, and which has been the cause of the formation of the ulcers.

The most frequent cause of trichiasis is trachoma (page 175). The conjunctiva, which in the regressive stage of trachoma undergoes cicatricial shrinking and contracts, tends to draw the skin of the lid backward over the free border of the latter, and thus puts the cilia more and more in a false direction. At first the most posterior cilia, afterward the anterior rows, too, are turned backward. The distortion of the tarsus acts to produce the same effect. Owing to this distortion, that portion of the tarsus which adjoins the free border of the lid bends off at an angle from the rest of the cartilage and is turned backward (t_1, Fig. 48 B), and in so doing it draws with it the covering of the free border of the lid which is firmly attached to it.

Partial trichiasis, in which only some of the cilia are turned backward, develops in consequence of scars which have been left upon the free border of the lid or in the conjunctiva by blepharitis, hordeolum, diphtheria, burns, operations, etc.

Under the name of *distichiasis*[11] is designated the condition in which, the lids being otherwise normally formed, there are two rows of cilia, one of which looks forward, and the other, which is usually less perfectly developed, is situated directly at the posterior edge of the lid and is directed backward. This condition occurs as a rare congenital anomaly, and is sometimes present in all four lids.

The term *distichiasis* is employed by most authors not only for the condition in which there are congenitally two rows of cilia, but also the acquired anomaly of position due to trachoma, when simply the posterior rows of cilia are set backward, while the anterior are still directed forward. But this condition is identical in its nature with trichiasis proper, and differs from it only in degree. At the commencement of

[11] From δίς, double, and στίχος, a row.

the cicatricial contraction the rows of cilia are first drawn apart; then the posterior, and last of all the anterior row are turned backward. Accordingly, distichiasis is developed first and trichiasis afterward, and a sharp line of distinction cannot be drawn between the two. I hence prefer to use the expression trichiasis for this condition in all its phases, and to confine the term distichiasis to the congenital cases in which two regular series of cilia are present. Then trichiasis and distichiasis really denote two perfectly distinct conditions.

609. Treatment.—When only a few of the cilia have an improper position they can be removed by epilation. Inasmuch as they grow again, epilation must be repeated at intervals of a few weeks—a thing which can very often be attended to by the patient himself or his relatives. It is still better to employ a method in which, with the performance of epilation, the follicle of the cilium is at the same time destroyed, so that the cilium does not grow again. The best procedure for this purpose is electrolysis (see § 828). When quite a large number of the cilia or all of them are directed toward the eyeball, epilation is not suitable; in that case these methods are indicated by means of which the cilia are brought to their proper position by a shifting of the place in which the hair bulbs are implanted (see § § 827-834).

2. *Entropion*

610. Entropion[12] is a rolling inward of the lid. Trichiasis affects simply the lid border, of which both the shape and the position are faulty. In entropion the shape of the lid border may be normal, but the lid border as a whole is revolved back so as not to be visible at all when the open eye is seen from in front. Hence, to get a view of the border of the lid we have to roll the lid out again by pulling it toward the margin of the orbit. However, the dividing line between trichiasis and entropion is not a sharp one, and the evil consequences of the two are the same.

According to their etiology we distinguish the two following varieties of entropion:

(*a*) *Entropion Spasticum* is that form which is produced by the contraction of the orbicularis. The fibers of the palpebral portion of the orbicularis (see page 650) describe arcs having a curvature in two different directions. One sort of curvature is due to the fact that the muscular fibers encircle the palpebral fissure; the concavity of these arcs accordingly looks toward the palpebral fissure and is directed downward in the upper lid, upward in the lower. The second variety of curvature is caused by the fact that the muscular fibers in conjunction with the lids are molded to the anterior convex surface of the eyeball; the concavity of these arcs looks backward in both lids. When the fibers of the orbicularis contract, they tend to shorten from the form of an arc to that of its chord. In so doing they exert a double action: by the flattening out of the first set of curves they narrow the palpebral fissure; by the flattening out of the second set they press the

[12] From ἐν, in, and τρέπειν, to turn.

lids against the surface of the eyeball. Either component may lead to inversion of the tarsus, if the character of the subjacent structures is such as to give rise to this condition. By the action of the orbicularis in the sense of the first component, the lids are forcibly opposed to each other by their narrow edges when the palpebral fissure is being closed. We may conceive of the two tarsi as represented by two visiting cards, standing one over the other in the same vertical plane, so that the lower edge of the upper rests upon the upper edge of the lower, and the edges of the two are forcibly opposed. Then slight pressure with the finger upon the line of contact of the two edges suffices to produce inversion of the cards in a sense opposite to that in which the pressure is made. In like fashion the opposed tarsi bend forward or backward, according as the subjacent structures either tend to press them forward or, on the other hand, fail to afford them a sufficiently firm support. Much more important, however, for the production of a change of position in the lids is the second component, in accordance with which the fibers of the orbicularis press the lid against the eyeball; for the lids are in perfect contact with the eyeball only so long as the latter furnishes them a uniform bed to lie upon. When then, owing to lack of uniformity in the bed, either the free or the attached border of the tarsus receives insufficient support posteriorly, a bending of the tarsus results in the sense either of an entropion or an ectropion.

The stronger the contraction of the fibers of the orbicularis in general, the more readily will the mechanical causes mentioned lead to an alteration in the position of the lids. And from what has been said it is easy to understand that there is a spastic ectropion as well as entropion; it depending upon the mechanical conditions above described and also upon other circumstances (especially upon the character of the skin of the lids) whether blepharospasm causes the lids to bend forward or backward.

For an entropion to develop, two conditions are necessary: Deficient support of the free border of the lid, and an abundant amount of extensible skin upon the lid. The former condition obtains when the eyeball is wanting; entropion spasticum, therefore, develops above all when the orbit is empty. For entropion to develop, however, it is not necessary for the eyeball to be entirely absent; it is sufficient that it should be diminished in size, or should simply be situated more deeply in the orbit, as it is in the old and lean. Again, when the border of the lid is rolled inward, the skin of the lid is drawn after it. If this is prevented an entropion cannot develop. If the entropionized lid is put back in place, and then the skin of the lid is drawn a little up toward the margin of the orbit and fixed there by pressure, the rolling in again of the lid is rendered impossible. Entropion spasticum, therefore, does not usually develop in persons having firm and elastic skin upon the lids, but requires the presence of a large amount of wrinkly, readily displaceable skin, such as we meet with in old persons.

It is clear that the rolling in of the lids is favored when the fibers of the orbicularis are very forcibly contracted, as in blepharospasm, and also when the pressure with which the muscular bundles of the orbicularis are squeezed against the border of the lid is aggravated by an external pressure—i. e., by a bandage. Similarly blepharophimosis acts to favor the development of entropion, since it draws the skin toward the free border of the lid.

If we summarize what has just been said, it follows that entropion spasticum develops mainly in elderly people with flabby lids, and that its production is favored by deep placing, diminution in size, or absence of the eyeball, by the existence of a blepharospasm or a blepharophimosis, and by the wearing of a bandage. For the last-named reason entropion is a frequent and unpleasant complication during the after-treatment of cataract operations where the patients are elderly people whose eyes have to be kept bandaged for quite a long time.

Entropion spasticum is almost invariably found to be restricted to the lower lid.

(*b*) *Entropion Cicatriceum* is caused by the cicatricial contraction of the conjunctiva by means of which the free border of the lid is drawn inward. It may be said to form one step further on the way upon which trichiasis has started. Like the latter, this condition is observed after trachoma, diphtheria, pemphigus, burns of the conjunctiva, etc.

The *treatment* of entropion is either with or without operation. In entropion spasticum which has formed beneath a bandage, the discontinuance of the latter is often all that is required. If we are compelled by important reasons to continue the bandage, we place upon the lower lid in the neighborhood of the margin of the orbit a roll of adhesive plaster, which is kept pressed against the lid by the bandage. This procedure depends upon the observation that the entropionized lower lid takes a correct position spontaneously when we press back with our fingers that portion of it which lies next to the margin of the orbit. Another method of exerting a constant pressure of this sort consists in fastening a narrow strip of a good sticky adhesive plaster to the lid throughout its entire length; the strip being placed about in line with the lower margin of the orbit and being drawn quite tight. [We may also evert the lid and try to hold it in place with collodion.—D.]

If entropion is due to absence of the eyeball, we enjoin the wearing of an artificial eye.

If we cannot attain our object by bloodless measures, we must then proceed to the performance of an operation (see § 837).

3. *Ectropion*

611. Ectropion consists in the revolution of the lid outward, so that its conjunctival surface looks forward. It is, therefore, the opposite of

entropion. There are different degrees of ectropion. The lowest degree is that in which the internal margin of the lid stands off a little from the eyeball (*eversion* of the border of the lid). Even this very slight degree, however, bears within itself the conditions for its own increase. With the eversion of the border of the lid there is also associated eversion of the puncta, in consequence of which epiphora develops, and by this a contraction of the skin of the lower lid and hence an increase of the ectropion are produced. Ectropion may present all degrees up to complete eversion of the entire lid. The consequences of ectropion are epiphora and also redness and thickening of the conjunctiva wherever it is exposed to the air. The hypertrophy of the conjunctiva may reach such a high degree, especially if this membrane has already been considerably altered (by gonorrhœal conjunctivitis or by trachoma), that the conjunctiva looks like exuberant "proud" flesh (wound granulations), whence the old designations ectropion luxurians (or sarcomatosum). In marked ectropion the cornea is incompletely covered by the lids, so that keratitis e lagophthalmo is set up.

According to differences in their etiology the following kinds of ectropion are distinguished:

(*a*) *Ectropion Spasticum.* It was shown, in speaking of entropion, that the lids can be bent by blepharospasm, and that the direction in which they are bent depends upon the mechanical relations of the individual parts. In ectropion these conditions are the direct opposite of those which we have found to be the causes of entropion. They consist partly in the displacement forward of the tarsal portion of the lid, and partly in a firm, elastic character of the skin of the lid, by virtue of which the palpebral border is drawn toward the margin of the orbit. We often have the opportunity to observe the effect of a traction of this sort when we try to open the palpebral fissure in a child with swollen lids and with blepharospasm. As soon as we draw the lids apart they become spontaneously everted, and in such cases if we should not carefully put the lids back in place we might readily set up a permanent spastic ectropion; for the peripheral bundles of the palpebral portion of the muscle contract spasmodically behind the everted tarsal portions of the lid, and maintain them in their faulty position. Then the everted lids swell up, because they become congested and this swelling renders their reposition the more difficult the longer the condition lasts. Inasmuch as a certain degree of tension of the skin of the lids is requisite for the development of spastic ectropion, this condition is found principally in children and young persons.

The second condition mentioned above for the development of ectropion is the forcing of the edge of the lid away from the eyeball so that eversion of the tarsus is facilitated. It occurs mostly as a result of thickening of the conjunctiva, particularly when due to gonorrhœal conjunctivitis and trachoma. However, the eyeball itself may force the lids so far forward that they become ectropionized, as is the case in enlargement or protrusion of the eye.

The two predisposing causes above mentioned will be the more likely to induce eversion of the lid, the greater the blepharospasm present.

From what has been said, it follows that ectropion spasticum is particularly apt to occur in young persons who at the same time suffer from inflammation of the conjunctiva with swelling of the latter and with coincident blepharospasm. Ectropion spasticum frequently affects the upper and lower lids simultaneously.

(*b*) *Ectropion Paralyticum* arises as a result of paralysis of the orbicularis. The lids are then no longer kept pressed against the eyeball by the contraction of the fibers of the orbicularis, and thus the lower lid sinks down of its own weight. For this reason ectropion paralyticum is found only in the lower lid; the upper lid, because of its weight, remaining applied to the eyeball even when there is no action of the muscle. In conjunction with drooping of the lower lid there is inability to lift it. Consequently the palpebral fissure cannot be perfectly shut (lagophthalmus).

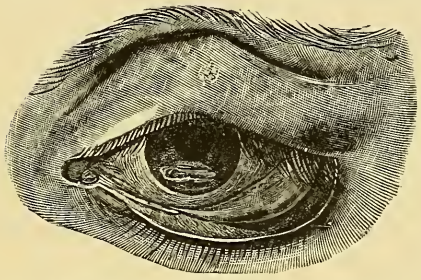


FIG. 289.—ECTROPION WITH LAGOPHTHALMUS.

This developed as a result of a cured caries of the upper and lower margin of the orbit. Beneath the middle of the eyebrow the skin of the upper lid is drawn by a scar 1 cm. long toward the upper margin of the orbit and attached to the latter. The free border of the lid is hence drawn up in its middle portion; but, what is of most consequence, the lid is prevented from going down when the eye shuts. There is a second scar at the outer end of the lower margin of the orbit. This scar is concealed by the border of the lid, but through the latter one can feel that here there is an irregular notching of the otherwise sharp and smooth margin of the orbit. The border of the lid at this spot is attached to the bone, and consequently the whole lid is drawn outward and downward. This is especially the case at its outer half, so that here the conjunctiva of the tarsus, being exposed to the air, is particularly hypertrophied and reddened, and hence appears dark in the drawing. The lower lid, being fastened down, cannot be raised when the eye is shut. Hence between the two lids the lower part of the cornea remains uncovered even during sleep. This uncovered portion presents an oblong ulcer with yellowish-white border and a depressed and therefore somewhat darker center. Above and below, an areolate gray cloudiness adjoins the yellow border of the ulcer.

(*c*) *Ectropion Senile* also is found only in the lower lid (Fig. 291). The way it develops is that in old people the lower lid is relaxed in all its parts and is pressed with insufficient force against the eyeball by the enfeebled fibers of the orbicularis. Another factor that here comes into play is the chronic catarrh of the conjunctiva (catarrhus senilis), which is so frequent in old people.

Likewise due to enfeeblement of the action of the orbicularis is the ectropion of the lower lid, that occurs when the lid has been divided in a vertical direction at any spot, or when the external commissure has been destroyed, so that continuity of the orbicularis is interrupted somewhere.

(*d*) *Ectropion Cicatriceum* develops when some part of the skin of the lid has been destroyed and has been replaced by scars so that the lid is contracted. Injuries (particularly burns), ulcers, gangrene, excision of the skin in operations, etc., may give rise to it. Ectropion frequently develops as a sequel to caries of the orbit in scrofulous children (Fig. 289). Here, in addition to the contraction of the skin of the lid, its fixation to the osseous

cicatrix upon the margin of the orbit is of moment, and with this there is associated marked retraction of the skin. So also ectropion is set up by the contraction of the skin and the loss of its elasticity that result from the eczema which occurs in connection with the continual wetting of the skin of the lids by the tears or from various other causes. Ectropion is therefore frequently found in conjunction with a blepharitis of long standing and with disorders of the conjunctiva and the lachrymal sac.

Non-operative *treatment* is successful mainly in ectropion spasticum. It consists in putting the lid back in place and keeping it pressed against the eyeball by a well-fitting bandage. In ectropion paralyticum we must employ, besides the bandage, the remedies indicated for the cure of facial paralysis, particularly electricity. Ectropion senile is curable without an operation only as long as it has not reached any very high degree. For quite a long space of time the eye must be bandaged at night, and the patient must be told, when wiping away the tears that flow down over his cheek, to apply his handkerchief from below upward, and not, as is ordinarily done, from above downward, a procedure by which the lid is drawn down only so much the more. It is, furthermore, advisable to slit up the inferior canaliculus in order to diminish the epiphora due to the eversion of the punctum. The higher degrees of ectropion, and particularly ectropion cicatriceum, require treatment by operations of one kind or another, for which see § § 838-841.

4. *Ankyloblepharon*

612. Ankyloblepharon[13] consists in an adhesion of the upper to the lower lid along the palpebral margin (Fig. 290). It is either partial or total, and is very often combined with an adhesion between the lid and the eyeball, or symblepharon. It also has a common etiology with the latter; it originates, that is, when, as a result of burns, ulcers, etc., the borders of the two lids are converted into raw surfaces at opposed points and so become adherent.

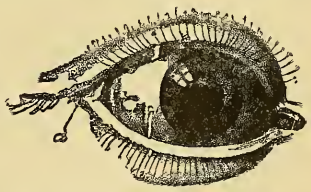


Fig. 290.—Ankyloblepharon. This occurred as the result of an injury from a splinter of glass. After the injury, the patient without calling in any medical aid kept the eye bandaged for some time. At *a* the margin of the upper lid is adherent to the margin of the lower, and over an area corresponding to this adhesion the cilia are absent. To the temporal side of the site of adhesion, a small portion of the palpebral fissure has been preserved as far as the external angle of the eye. Hence in this case simple division of the bridge would suffice to remove the ankyloblepharon.

Through ankyloblepharon the palpebral fissure is diminished in size, and the movements of the lids are hindered; in total ankyloblepharon there is complete occlusion of the palpebral fissure. The treatment, when we are dealing with simple ankyloblepharon without coincident symblepharon, consists in separating the adherent lids by an operation. If the adhesion extends as far as the angle of the lid, the

[13] From ἀγκύλη, a stiff limb [or a thong, and βλέφαρον, eyelid].

latter must be supplied with a lining of conjunctiva, as otherwise the adhesion would form again, starting from the angle. In the cases in which symblepharon is present along with the ankyloblepharon, it depends mainly upon the extent of the former whether an operation is practicable at all or not.

5. *Symblepharon* (see page 217)

6. *Blepharophimosis*

613. In blepharophimosis[14] the palpebral fissure appears to be contracted at the external angle of the eye (Fig. 291). Upon drawing the lids apart we see that the contraction is produced by a fold of skin which extends in a vertical direction at the external angle of the eye and juts out in front of it like a sliding screen. If we draw the fold of skin outward, we disclose behind it the normally formed external palpebral angle with the delicate ligament uniting the borders of the two lids. The distinction between ankyloblepharon and blepharophimosis, two conditions which are commonly confounded, is therefore as follows: In ankyloblepharon the borders of the lids are adherent to each other, but in blepharophimosis they are normal, and the contraction of the palpebral fissure is only apparent, being caused by the drawing of a fold of skin over its outer extremity.

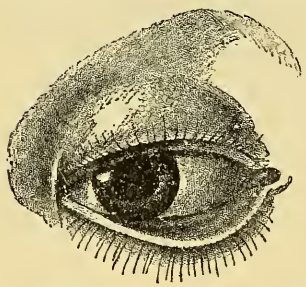


Fig. 291.—Blepharophimosis and Ectropion Senile.

The ectropion as is usually the case, is more marked in the nasal than in the temporal portion of the lid, because in the former the contact of the lower lid with the upper is interrupted by the horseshoe-shaped notch at the inner angle of the eye. Moreover in the present case the blepharophimosis counteracts the drooping of the lid at the outer angle.

Blepharophimosis is most frequently found in persons who suffer from epiphora and blepharospasm of long standing—that is, it is especially met with in chronic inflammations of the conjunctiva. It originates in a contraction of the skin of the lids due to their being frequently wet with the tears or with secretion. If this contraction is particularly marked in a horizontal direction, the skin from the adjacent part is drawn up so as to project over the palpebral fissure on the temporal side like a sliding screen. This effect is re-enforced by the action of the fibers of the orbicularis, which in blepharospasm draws the skin on the outer side of the face in toward the external angle of the eye. We can artificially imitate blepharophimosis by pushing the skin from the temple over the palpebral fissure with our fingers; and conversely can make an existing blepharophimosis disappear by drawing the skin out toward the temple. Blepharophimosis is ordinarily not found at the inner angle of the eye, because the adjoining skin of the bridge of the nose is not so readily displaced, although in old

[14] From βλέφαρον, lid, and φίμωσις, contraction, from φιμός, a muzzle.

persons with thin skins a projecting fold of skin is sometimes formed here too.

Blepharophimosis accordingly, like that form of ectropion which occurs in connection with chronic catarrh, epiphora, etc., owes its origin to a contraction of the skin of the lid. The difference between the two lies in the fact that, in the first case, the contraction makes itself apparent chiefly in the horizontal direction; in the second case, in the vertical direction. Blepharophimosis and ectropion, therefore, as originating from the same cause, may be both present at the same time (Fig. 291). That this in general is but rarely the case is due to the upward traction which the vertical fold of the skin forming the blepharophimosis exerts upon the lower lid and which opposes the eversion of the latter. For this reason, indeed, blepharophimosis really favors the development of an entropion, which in such cases can often be cured simply by the abolition of the blepharophimosis. Another consequence of blepharophimosis is the contraction—to be sure, an apparent one only—of the palpebral fissure, which consequently cannot be opened as wide as usual.

Blepharophimosis, at least in young persons with elastic skin, may gradually disappear of itself, provided its causes (epiphora, blepharospasm) have ceased to act. If it does not appear spontaneously and if it causes any trouble, it may be removed by widening the palpebral fissure by means of canthoplasty (see § 835).

7. *Lagophthalmus*

614. By lagophthalmus[15] is meant an incomplete closure of the palpebral fissure when the attempt is made to shut the lids together. In the lesser degrees of lagophthalmus complete closure of the palpebral fissure is still possible by squeezing the lids tight; but since during sleep there is no such squeezing of the lids, but only a gentle closure of them, such patients sleep with their eyes open, and from this the disease derives its name. In the higher degrees of lagophthalmus it is no longer possible for the patients to bring the lids into contact even by forcible squeezing.

The evil consequences of lagophthalmus depend upon the harm which the eyeball suffers from being insufficiently covered. What part of the eyeball is it that remains uncovered by the lids in lagophthalmus? If we tell a patient with a slight lagophthalmus to shut his lids tightly together, we see that the borders of the lids remain separated some millimetres from each other, and that between them there lies the part of the sclera that is below the cornea, but not the cornea itself. The reason for this is that at the same time that the lids are shut the eye is turned upward, so that the cornea is concealed beneath the upper lid. The same is the case during sleep. Hence the only part of the conjunctiva scleræ that is constantly exposed to

[15] I. e., hare's eye, from λαγώς, hare, because it was believed that hares sleep with their eyes open.

the air is that situated below the cornea. As a result of this exposure it is injected, and the patient suffers from the symptoms of a chronic conjunctival catarrh. In the higher degrees of lagophthalmus the cornea, too, is seen to lie in the slit which remains open when the lids are closed together; and, because the cornea is turned upward, it is the lower part of it that is found there. Lagophthalmus but seldom reaches a degree such that the cornea remains completely uncovered.

The cornea may suffer in two ways from being covered insufficiently; either its surface dries up wherever it is constantly exposed to the air, and keratitis e lagophthalmo ensues (see page 277 and Fig. 289); or the cornea protects itself against the exposure by a change in its epithelium which becomes thicker and epidermoid, so that the deeper layers of the cornea are preserved from desiccation (xerosis of the cornea, see page 219). But as opacity both of the epithelium and of the cornea itself is associated with this process, the sight is thereby prejudiced. In any case, therefore, vision is endangered in lagophthalmus if the latter is so considerable that the cornea is no longer sufficiently covered. Another result of lagophthalmus is epiphora, since complete closure of the lids is requisite for the normal conduction of tears into the nose.

The causes of lagophthalmus are: 1. Narrowing of the lids. This is in most cases caused by the loss of a portion of the skin of the lids in consequence of burns, ulcers (particularly lupus), operations, etc. Less frequent are the cases of congenital narrowness of the lids. These are characterized by the fact that the palpebral fissure still remains open a distance of some millimetres when the lids are shut tightly together, and that, nevertheless, no signs whatever are present of loss of the skin of the lids under the form of scars. In these cases the symptoms of a chronic blepharitis ulcerosa are often present. 2. Ectropion. 3. Paralysis of the orbicularis. 4. Constant patency of the eyes, occurring in persons who are very ill or who are unconscious, and due to a reduction of the sensitiveness of the cornea, so that the reflex acts of winking and of shutting the eyes are no longer initiated. 5. Enlargement or protrusion of the eye, so that the lids, in spite of being normal in size and mobility, are unable to cover it completely. Under this head should be specially mentioned Basedow's disease, in which the lagophthalmus is bilateral, so that bilateral blindness sometimes occurs as a result of it.

The *treatment* of lagophthalmus consists first of all in regarding the causal indication—i. e., in the removal of those conditions which prevent the complete closure of the lids. Under this head belong the remedying of contraction of the lids by blepharoplasty, the cure of ectropion, the treatment of facial paralysis, etc. Until we have succeeded in getting rid of the lagophthalmus, for which often quite a long time is required, the eye must be protected from its ill effects. This is accomplished by closing the palpebral fissure artificially with a bandage. For this purpose we first bring the

borders of the lids into perfect apposition, and keep them so by strips of sticking plaster which are attached vertically across the lids; over this is applied an ordinary protective bandage with dry cotton. In the lighter cases it is sufficient to apply this bandage at night, since then the danger of desiccation of the cornea is the greatest, and during the day the act of winking suffices to keep the cornea moist. But in the higher degrees of lagophthalmus, or in cases in which the cornea is already attacked, the bandage must be worn constantly. When the lagophthalmus is so great that the margins of the lids no longer come near touching and cannot be kept in contact by a bandage, we may give up the attempt to cover the cornea and simply keep it from drying by making a moist chamber. For this purpose we cover the eye with a watch-glass, the edges of which are fastened down tight by plaster to the circumference of the orbit. At some point in the concavity of the watch-glass is placed a little cotton wet with water. Because of the warmth of the eye the water evaporates, so that the air between the watch-glass and the eye is kept constantly moist.

In those cases in which the removal of the cause of the lagophthalmus is impossible, or is likely to require a very long time (as, for example, in the cure of a case of Basedow's disease), it would be disagreeable for the patient to have to wear a bandage for so long a time—a year or so. For these cases tarsorrhaphy (see § 836) is advisable. By this the palpebral fissure is abbreviated and the borders of the lids are brought nearer each other, so that the closure of the eye is facilitated.

V. Diseases of the Palpebral Muscles

1. *Orbicularis*

615. (*a*) *Spasm of the Orbicularis* (*Blepharospasm*[16]). This manifests itself by a squeezing of the lids tightly together. It is either a symptom accompanying other diseases of the eye (symptomatic blepharospasm), or it forms a distinct disease by itself (essential blepharospasm).

Symptomatic blepharospasm accompanies all irritative states of the eye, and is hence found in conjunction with the presence of foreign bodies in the conjunctival sac, with trichiasis, with the most various forms of inflammation of the eye, when an intense amount of light falls on the eye, etc. The violence of the blepharospasm is by no means in direct proportion to the severity of the disease of the eye, so that no sort of conclusion can be drawn from it with respect to the violence or the duration of the ocular affection that lies at the root of it. It often renders the examination of the eye extremely difficult. Blepharospasm is usually most violent and most obstinate in conjunctivitis eczematosa. It reacts unfavorably upon the eye trouble; moreover, it often leads to œdema of the lids, to blepharophimosis, and to spastic ectropion and entropion. The treatment of symptomatic

[16] From βλέφαρον lid, and σπᾶν, to draw tight.

blepharospasm consists in the removal of the ocular disease which lies at the root of it (cf. page 202).

Essential blepharospasm is distinguished from the symptomatic variety by the fact that in it the eyes themselves are found to be perfectly normal. In young persons, particularly of the female sex, it manifests itself by the eyes' suddenly shutting up and then remaining closed as if in sleep (*blepharospasmus hystericus*). In elderly people blepharospasm (*blepharospasmus senilis*) appears either under the form of clonic spasm—i. e., continual winking (nictitatio[17])—or as a tonic spasm by which the eyes are kept tightly closed for a length of time. Essential blepharospasm is extremely annoying to the patient; indeed, in severe cases it has, as far as he is concerned, almost the same results as a real blindness, since the patient cannot make use of his eyes when they are shut. Hysterical blepharospasm in time disappears of itself, while senile blepharospasm resists treatment for a long time, and sometimes, indeed, is absolutely incurable.

In *hysterical* blepharospasm both eyes shut up suddenly and usually without any known cause. It may take some hours, but it may also take days and even months before the eyes open again, which they do, and just as suddenly. Such attacks may be repeated quite often, and may also vary greatly in their duration. The spasm almost always affects both eyes; once only have I seen a unilateral hysterical blepharospasm. Other symptoms of hysteria are often present coincidentally with the blepharospasm. In a young girl who suffered with blepharospasm of this sort, and whom I had already treated in vain with different remedies, I was finally successful in relieving the blepharospasm by means of a single instillation of cocaine; but a few minutes after the girl had opened her eyes both legs became paralyzed, and remained so for several days.

In the examination of patients with hysterical blepharospasm it is often possible to find so-called pressure points—i. e., portions of the body upon which we simply have to press for the eyes to open as if by magic (Von Graefe). In the majority of cases the pressure points lie in the region supplied by the trigeminus as at the place of exit of the supra-orbital and infra-orbital nerves at the upper and lower margins of the orbit. But often these points are more difficult to find, being situated, for example, in the cavities of the nose, the mouth (when there are carious teeth), or the throat; sometimes the pressure points are not found in the region supplied by the branches of the trigeminus at all. Thus cases occur in which the blepharospasm can be made to disappear by pressure upon one of the costal cartilages or the vertebræ, or upon some spot of the arm or leg, etc. Often the patient himself calls the physician's attention to the position of the pressure points, the knowledge of which he has already utilized for his own relief. The most frequent cases are those in which the patient allays the blepharospasm by pressure upon the forehead, a pressure which acts upon the branches of the supra-orbital nerve. Young men wear a hat with a stiff crown which they press down hard upon their face; girls tie a ribbon tight about their head. But as soon as the pressure ceases—e. g., by taking off the hat—the eyes shut together again. In many cases the diversion of the attention to other things suffices to temporarily relieve the blepharospasm.

The form of hysterical blepharospasm, in which the eyes remain quietly closed without any apparent spasm, presents a great similarity to ptosis. We can, however,

[17] From *nictare*, to wink.

readily recognize it to be a spasm if we try to open the eye by lifting the upper lid; for we then feel the resistance that the orbicularis offers to the opening of the eye. As indicative of the contraction of this muscle, we see that the skin of the forehead is thrown into vertical wrinkles, and that the eyebrows are lower than usual. In ptosis the skin of the forehead, owing to the contraction of the frontalis, shows horizontal wrinkles, and the eyebrows are somewhat higher than usual (Fig. 292). In the normal state the situation of the eyebrows corresponds to the upper margin of the orbit.

[Some cases of hysterical blepharospasm have been caused in the present war by shell explosions.—D.]

Senile blepharospasm is often only one of the symptoms of a general spasm of the face (tic convulsif). Of the two forms of this spasm the clonic variety is the less disagreeable for the patient, because sight is but little interfered with by the constant winking. In tonic spasm the eyes shut suddenly and remain spasmodically closed for some minutes. If the patient is attacked by this spasm in a crowd, or while crossing the street, or under other such conditions, he is helpless and exposed to the likelihood of an accident. In senile blepharospasm, too, there are frequently pressure points which influence the spasm.

Just as the normal act of winking is set up in a reflex way by irritation of the terminal extremities of the trigeminus upon the surface of the eyeball, so, too, blepharospasm is in most cases of reflex nature. This is beyond doubt the case in symptomatic blepharospasm, in which the irritation of the trigeminus due to a foreign body, to inflammation, etc., is obvious. But besides this, a reflex action starting from the trigeminus must be assumed to exist in many cases of essential blepharospasm also. A proof of this is the fact that pressure upon branches of the trigeminus so often abrogates the blepharospasm, and that at the same time the pressure points themselves are often sensitive to pressure. *Treatment* has therefore the greatest prospect of success in those cases in which it is possible to find pressure points, as then we can attack directly the starting point of the reflex action. This is done by applying the galvanic current to the pressure points, or by injecting morphine at these spots. In a girl in whom pressure on the vertex relieved the blepharospasm the repeated inunction of an ointment (veratrine ointment) upon this spot sufficed to do away with the spasm. If no pressure points are discoverable, we must think of the surface of the eyeball itself as a source of the reflex action, and may try to render this insensitive by cocaine. It is probable that senile changes in the facial lie at the root of senile blepharospasm, and hence the outlook for a cure is small. We must try the galvanic current (anode in front of the lobe of the ear on the trunk of the facial, cathode on the nape of the neck). In addition we employ the remedies used against neuroses in general. Schloesser recommends the injection of alcohol along the trunk of the nerve at its point of exit from the stylo-mastoid foramen. Of operative measures that may be mentioned are stretching or resection of those branches of the trigeminus from which the reflex emanates and stretching of the trunk of the facial. It is only in senile blepharospasm that such heroic remedies will be adventured upon, as hysterical blepharospasm always passes off of itself in the course of time.

Children of from eight to fifteen years are frequently brought by their parents to the physician on account of a habit of *continual winking*. This happens not infrequently during the occurrence of a slight conjunctivitis and keeps on independently after the latter has been relieved. For the most part, in this case we have to deal with rather anæmic and nervous children. This affection—frequently attributed to badness by the parents—usually passes off of itself after some time.

Repeated *fibrillary contractions* of single bundles of the orbicularis, which are appreciated by the patients themselves, occur very frequently in perfectly healthy persons with normal eyes. No sort of significance is to be attributed to them.

616 (*b*) *Paralysis of the Orbicularis.*—If the case is one of recent paralysis, no changes are noticed while the eye is open; but if the patient has cause to shut his eye, it is apparent that the closure is but incompletely performed, because the lower lid cannot be lifted properly. This is particularly striking in the inner half of the lid. In consequence of the incomplete closure of the lid there is epiphora, which in light cases often constitutes the only complaint the patient makes. After the paralysis has lasted quite a long time further changes set in. The lower lid falls away from the eyeball and keeps drooping lower and lower all the time (ectropion paralyticum). The cornea during sleep is exposed to desiccation in its lowermost part, so that keratitis e lagophthalmo develops.

Paralysis of the orbicularis is caused by an affection of the facial nerve which innervates it. A lesion of the facial nerve may have either a central or a peripheral situation. In the former case it is located in the course of the nerve fibers extending from the cortex of the brain to the point where the nerve comes out at the posterior border of the pons, in the second case is located in the nerve trunk itself. In the case of a lesion situated above the nucleus (supranuclear lesion) and paralysis chiefly affects its oral branches, while the orbicularis is usually normal. Hence, in a paralysis of this muscle we have ordinarily to do with a lesion of the nerve in the nucleus itself or below it. Most frequently we have to deal with a so-called rheumatic paralysis, the real cause of which is unknown; but the paralysis may also be caused by injury (particularly fractures of the base of the skull and operations in the region of the parotid gland [and middle ear]), by caries of the petrous bone, by tumors, and by syphilis. The rheumatic paralyses of the orbicularis give a favorable prognosis, but even in them several months are required before the cure takes place.

The *treatment* must first of all endeavor to remove the cause of the paralysis. Symptomatic treatment consists mainly in the application of the electric current, both constant and induced. As long as the closure of the lids is imperfectly performed, the palpebral fissure must be kept closed by a bandage (see page 687), to prevent the development of ectropion and of keratitis. In severe cases the bandage must be worn constantly; in light cases it is sufficient to apply it at night only. If the paralysis proves incurable, tarsorrhaphy is indicated in order to make it possible to shut the eyes.

2. *Levator Palpebræ Superioris*

617. *Paralysis* of the levator palpebræ superioris manifests itself by a drooping of the upper lid (*ptosis*[18] [*or blepharoptosis*]). All degrees of ptosis occur, from a just noticeable depression of the upper lid, to a prolapse of it so complete that it hangs down quite relaxed and devoid of wrinkles, and covers the whole eyeball. The higher degrees of ptosis, in which the lid hangs down

[18] From πιπτειν, to fall.

in front of the pupil, interfere with vision, unless the patient lifts up the lid with his finger, or unless it is possible for him to draw it up sufficiently by a forced action of the frontalis muscle. By the contraction of the latter the forehead is wrinkled and the skin over it is thus contracted, so that the eyebrows and indirectly the upper lid as well are elevated (Fig. 292). But since this elevation is not sufficient, the patient is in addition compelled to throw his head back, because then in looking forward the eyes are directed down, and thus the pupils get to lie in the palpebral fissure even though it is low. The wrinkled forehead, updrawn eyebrows, and backward pose of the head are characteristic of persons with bilateral ptosis.

Ptosis is either acquired or congenital in its occurrence. *Acquired* ptosis may be caused by a lesion either of the muscle itself or of the nerve supplying it. The former condition is most frequently due to injuries, the latter to lesion of the oculo-motor nerve, since the nerve supplying the levator is a branch of the latter, and hence ptosis is often found in conjunction with paralysis of other muscles supplied by the oculo-motor nerve. The cases of isolated ptosis without any other signs of oculo-motor paralysis are caused mainly by central disease.

In *congenital* ptosis the levator palpebræ superioris is found to be either deficiently developed or entirely absent. In contradistinction to acquired ptosis, which usually affects only one eye, congenital ptosis is generally bilateral. Not infrequently is it transmitted by inheritance through several generations.

As regards the *treatment*, our endeavor in acquired ptosis, should be, to find out the cause of the paralysis and remove it by suitable measures. But if we have to do with a ptosis which has already become inveterate, or if it is congenital, an improvement of the condition can be obtained only by way of an operation (see §§ 842-845).

Congenital ptosis is frequently found in conjunction with other congenital anomalies. Among the latter are an inability to look up, accounted for by a deficient development or actual absence of the superior rectus (Steinheim); also epicanthus (§ 621).

There are cases in which the upper lid droops somewhat owing to congenital ptosis but rises when the mouth is opened or lateral movements of the lower jaw are made [jaw-winking]. The same connection between movements of the upper lid and of the lower jaw has been at times observed without any coexisting ptosis.

In *acquired ptosis* a similar sympathetic movement of the lid, and one that in this instance occurs conjointly with movements of the eyeball, is observed not infrequently. This is found in cases of oculo-motor paralysis of central origin, and it takes place in the following way: The ptosis attains its highest pitch in abduction of the eye; while in adduction (or in the attempt at adduction if the internal rectus is completely paralyzed), it diminishes, or disappears altogether, or even at times is converted into the opposed condition, so that the upper lid rises abnormally high. [This occurs not only in central oculo-motor paralysis, but also in peripheral paralyses of the eye muscles, including especially congenital paralysis of the external rectus due to actual deficiency of the muscle. In this latter set of cases, and rarely in some of the others, the ptosis

occurs in adduction, not in abduction. In some of these cases, at any rate, it is not a true ptosis that is in question, but a contraction of the orbicularis, marked by a sinking of the upper and a rising of the lower lid. There are also cases of *cyclic widening and narrowing* of the palpebral fissure, the widening, which is evidently due to spastic contraction of the levator, being associated with convergence of the eyes and miosis (Von Hippel).—D.]

There is a sort of ptosis that develops without known cause in women (very rarely in men) of middle age. It is always bilateral, and sets in so gradually that not till after

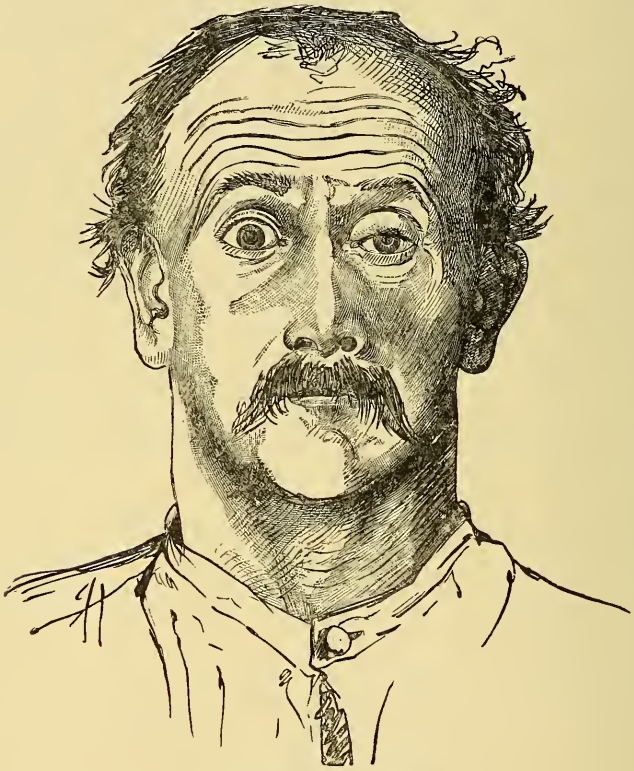


Fig. 292.—Left-sided Ptosis.

The ptosis was not complete, but just great enough for the upper lid to cover the pupil. Now as the left eye was the better of the two, the patient, in order to see with it, lifted his lid by contracting the frontalis muscle. He was not able, however, to do this on the left side alone, but wrinkled his forehead all over, so that the eyebrows are elevated on both sides. Hence, too, the right upper lid is drawn unusually high up, so that a strip of sclera can be seen exposed above the cornea, and the difference between the two palpebral fissures has become even more striking than before. The pupil of the right, amblyopic eye is dilated.

a series of years is it pronounced enough to cause any considerable interference with vision. In these cases it is not a paralysis of the nerve, but a primary atrophy of the muscle itself that is present (*ptosis myopthica*).

Mechanical means have been recommended for lifting the lid in ptosis, including the jamming of a monocle into the eye, and the pushing up of the upper lid by a fine, properly curved gold wire which is either attached to an eye-glass frame or springs apart on the plan of a spring speculum (Fig. 400), and which catches up a fold of the skin on the lid and by means of this lifts the lid. But these devices have met with but

scant approval and should be recommended only for cases in which the patient absolutely cannot make up his mind to have an operation done.

The name of ptosis is incorrectly used for conditions which have nothing to do with an affection of the levator palpebræ superioris—e. g., when the upper lid droops because it has become heavier in consequence of thickening due to trachoma, new growths, etc. [or when the upper lid is drawn down by a slight spasm of the orbicularis]. So, too, ptosis adiposa (page 665) does not belong with genuine ptosis.

Both paralysis and spasm are also observed in the unstriated levator or *musculus tarsalis superior* of Müller. Paralysis of this muscle underlies that slight ptosis which is one of the group of symptoms due to paralysis of the sympathetic (see page 451); and also underlies ptosis trachomatosa. Spasm of the muscle, manifested by drawing up of the upper lid and dilatation of the palpebral fissure, can be excited artificially by instilling cocaine. [See also page 451.] Moreover, according to some, the elevation of the upper lip lid in Basedow's disease is due to spasm of Müller's muscle.

[Such a *blepharodiastasis* or condition in which the palpebral fissure is wider open than usual may also be caused by a complete tenotomy with retraction of the superior or inferior rectus (see page 656). On the other hand, slight ptosis, or at least narrowing of the palpebral fissure, may be produced by advancement of these muscles or by paralysis not due to tenotomy (see page 656).—D.]

[An apparent ptosis is produced when the eye recedes into the orbit.—D.]

VI. Injuries of the Lids

618. Injuries of the lids of all kinds, including simple contusions, incised, lacerated, and contused wounds, burns by heat or caustics, etc., are very frequent. A peculiarity of these injuries that needs to be emphasized is that, because of the great elasticity of the skin of the lids and their loose attachments to the subjacent parts, both ecchymosis and œdema in the injured lids are usually much more considerable than after a similar injury in other parts of the body. Accordingly, we must not allow ourselves to be frightened merely by the great swelling and bluish-black discoloration of the lids, as these appearances are often enough produced by comparatively slight contusions. On the contrary, the diagnosis and prognosis should not be pronounced until after a careful examination. In this, three points are chiefly to be considered: Solutions of continuity of the skin of the lids, injury to the subjacent bones, and injury of the eyeball.

Solutions of continuity of the skin of the lids present a varying aspect according to their direction. Those which run horizontally—i. e., parallel to the line of fibers of the orbicularis—gape but little, so that the lips of the wound often lie in apposition spontaneously. But if the cut or rent runs in a direction perpendicular to the fibers of the orbicularis, the wound gapes widely in consequence of the retraction of the divided bundles of the muscle. Consequently, the cicatrices after horizontal wounds of the skin of the lids are scarcely visible, while those after vertical incisions are conspicuous and disfiguring. Hence, in operations upon the lids the rule is laid down that all incisions should, wherever possible, be made parallel to the course of the fibers of the orbicularis. The worst wounds are those which sever the lid

in a vertical direction through its entire thickness. If these do not unite by first intention, there remains an indentation of the border of the lid, or even a deep triangular incision in it (coloboma palpebræ traumaticum). By this the complete closure of the lid is rendered impossible, so that, in addition to the disfigurement, a permanent epiphora results from the injury.

The presence of an *injury of the subjacent bone* is determined by palpating the margin of the orbit with the finger through the swollen lid. A fracture of the orbital margin is manifested by unevenness and special sensitiveness at some spot, symptoms to which in many cases is added distinct crepitation. A certain sign of injury of the bone is *emphysema of the lids.* This consists in the entrance of air into the cellular tissue beneath the skin of the lid. The lids then have a peculiar soft feeling like a feather bed, and at the same time we get in the palpating finger a sense of crepitation due to the displacement of bubbles of air beneath the pressure of the finger. The air comes from the cavities surrounding the orbit—most often from the ethmoid cells. Its presence in the subcutaneous cellular tissue of the lids hence presupposes some abnormal communication between the latter and these cavities, such as can have originated only through a fracture of the bone. When then, by blowing the nose, straining, and coughing, the air in the nose and its accessory cavities is put under greater pressure, it is forced into the subcutaneous cellular tissue, and thus emphysema is produced (see § 723).

An added significance is imparted to wounds of the lids from the fact that by their mutilation the *eye itself* may be exposed to danger. Through cicatricial contraction of the lids, or the formation of fissures in them, lagophthalmus and consequently inflammation of the cornea may be set up.

The *treatment* of injuries of the lids is carried on according to general surgical rules. In simple ecchymosis we apply cold compresses with lead water. In emphysema of the lids the air contained in the tissues usually undergoes resorption without causing any ill results. To accelerate absorption a compressing bandage is indicated; at the same time the patient must avoid straining, blowing the nose, etc., in order not to drive fresh charges of air into the tissue. Recent wounds, the edges of which are not too greatly contused, are best united at once by sutures. In wounds the edges of which are destroyed by contusion and the like, we apply an antiseptic dressing and wait for the necrotic portions to be cast off. The same rule holds good for burns and injuries caused by caustic substances. After the elimination of those portions of the skin that have been destroyed, granulating raw surfaces are presented to view which cicatrize over and thus lead to a contraction of the lids. To combat this, we proceed precisely as has been laid down for the treatment of destruction of the skin by inflammation (see page 661).

619. Ecchymoses of the Lids.—*Ecchymoses* are usually pretty sharply limited at a line corresponding to the margin of the orbit, because the skin is attached to this by rigid connective tissue which prevents the further advance of the blood. On the other hand, the blood not infrequently travels beneath the skin of the dorsum of the nose over to the other side. An ecchymosis is then found in the lids of the other eye also. As the skin upon the dorsum of the nose is thick, it frequently does not permit the blood to be seen through it, so that we do not notice the bridge connecting the ecchymosis of one eye with that of the other. We might then easily be induced to believe that the injury had affected the other eye also, a thing which, however, can be excluded with certainty in many cases—e. g., when sugillation of the lids of one side sets in after enucleation of the other eye.

A similar migration of extravasated blood is observed in *fractures of the base of the skull.* The blood oozes forward from the site of the fracture and travels along the floor of the orbit. It then makes its appearance some time after the injury under the form of an ecchymosis in the lower part of the conjunctiva of the eyeball, and also on the lower lid close to the margin of the orbit, particularly in the region of the inner angle of the eye. This symptom, although it is not present in all cases, is of great importance for the diagnosis of fractures of the base.

Spontaneous ecchymoses sometimes occur in the lids, in the same way as they do in the conjunctiva, from violent straining, excessive coughing, and the like [also in infantile scurvy (Stephenson).]

Extravasations of blood into the lids, instead of disappearing by resorption, may go on to *suppuration,* so that an abscess of the lids develops. This is particularly to be apprehended when there is at the same time a solution of continuity of the skin of the lids, through which infectious germs may penetrate into their tissue.

VII. Tumors of the Lids

620. (*a*) **Benign Tumors.**—*Xanthelasma*[19] is a flat tumor of a dirty sulphur-yellow color and projecting but little above the skin of the lid. It is found most frequently on the upper and lower lids in the neighborhood of the inner angle of the eye. In this situation the tumors are often located symmetrically on the two sides of the eye, like the yellow spots above the eyes of the dachshund. Xanthelasmata occur in elderly persons, particularly of the female sex. They grow very slowly, and have no bad results besides the disfigurement they cause, which, moreover, affords the only reason for their removal by operation, as is sometimes done.

Molluscum contagiosum is a small, rounded tumor, the surface of which is somewhat flattened and has an umbilicated depression in its center. From this a substance resembling sebum is discharged upon pressure. This form of molluscum is contagious.

Molluscum simplex (fibrous molluscum) is a tumor of the skin which is attached by a pedicle to the skin of the lids and hangs down like a pouch

Warts and cutaneous horns are also observed on the lids.

Among *cysts,* milia, atheromata, and dermoid cysts occur. The latter, which may attain pretty large dimensions, will receive a more detailed description under the Head of affections of the orbit (§ 730). On the borders

[19] From ξανθός, yellow, and ἔλασμα, plate. It is also called xanthoma.

of the lids small, transparent cysts are frequently met with (Fig. 68), which have developed from occluded sweat glands in the border of the lid (glands of Moll).

The *vascular tumors* (angiomata) are found in the lids under the two forms of telangiectases and of tumores cavernosi. The former are bright-red spots situated in the skin of the lid itself, and are composed of dilated and tortuous blood-vessels. The latter lie beneath the skin of the lid, which they bulge forward and through which they can be seen shining with a bluish luster. They consist of closely aggregated, large, venous cavities, which can be felt and compressed through the skin; the arteries running to the tumors are dilated. Vascular tumors are usually congenital, but they develop still more extensively after birth, and sometimes attain such a size that they cover a great part of the face, and are also continued backward into the conjunctiva and the tissues of the orbit. Hence they should be removed as early as possible. In doing this our principal care must be to destroy the skin of the lids over as small an area as possible, as otherwise we might get shrinking of the skin, with ectropion and lagophthalmus. If the angioma is so small that we can remove it without sacrificing too much skin, it is best to extirpate it; or we can destroy such angiomata with fuming nitric acid or with the thermo-cautery or the galvano-cautery. In the case of very superficial angiomata the application of carbon-dioxide snow or treatment with radium is serviceable. In the case of large angiomata electrolysis is to be preferred (see § 826).

For other benign tumors of the lids, see page 697.

621. (*b*) **Malignant Tumors.**—*Carcinomata* occurring in the lids are, as a rule, epitheliomata, which start from the skin of the lid, particularly from that of the border. Subsequently they pass over upon the eyeball and even penetrate into the depth of the orbit. *Sarcomata* develop from the connective-tissue portion of the lids. The pigmented melano-sarcomata most often develop from a congenital nævus of the skin or conjunctiva. With malignant tumors we find an enlargement of the neighboring lymphatic glands, occurring first in the gland in front of the ear, afterward in the glands along the lower jaw and in the neck.

Superficial epitheliomata may be cured by treatment with Röntgen rays or with radium, but this takes a long time and does not afford a perfect protection against recurrences. Moreover, the sight may suffer through injury to the retina. [The treatment, however, may be applied in the desperate inoperable cases to relieve pain or as a forlorn hope.—D.] The quickest and surest treatment is extirpation, provided that it is made thoroughly, i. e., far enough into the sound parts; one should keep at least a centimetre outside of the visible or palpable border of the neoplasm. If in its performance so much of the lid must be sacrificed that the eyeball

as a consequence remains uncovered, a substitute for the palpebral skin that has been destroyed must be procured by a blepharoplasty done immediately after the removal of the new growth. In extensive tumors it is often necessary to remove the eyeball, and even the entire contents of the orbit.

The flat cancers of the skin that not infrequently occur upon the lids require particular mention. In these cases there is found a shallow ulcer with an uneven floor and irregular, rather hard walls. The infiltration of the walls of the ulcer is the only characteristic sign, for there is no tumor in the proper sense of the word, and for this reason inexperienced observers readily mistake the true nature of the lesion, which is nothing else than an epithelial carcinoma. The ulcer advances in one direction while it cicatrizes on the side opposite, for which reason it has been called by the name of *ulcus rodens*. Its progress, however, is extremely slow, so that such tumors often last many years before attaining any great size.

[Blastomycetic dermatitis (see page 662) may simulate a malignant tumor of the lids.—D.]

In consideration of the complicated structure of the lids in which such a manifold variety of tissue takes part, it should not surprise us that the most dissimilar kinds of tumors should at times come under observation in them. As rare occurrences there have been observed lymphomata, fibromata, enchondromata, myxomata, lipomata, cavernous lymphangiomata, plexiform neuro-fibromata (page 499), adenomata of the sebaceous glands, of the Meibomian glands, of the glands of Krause, of the sweat glands, and of the glands of Moll, and lastly glandular carcinomata.

VII. Congenital Anomalies of the Lids

622. By *coloboma of the lids* is meant a fissure in it, having approximately the shape of a triangle, the base of which corresponds to the border of the lid, while its apex looks towards the margin of the orbit. Coloboma is either congenital (coloboma congenitum) or is acquired, being then produced by injury (coloboma traumaticum). Congenital coloboma (Fig. 293) is on the whole, rare, and is observed oftener in the upper than in the lower lid. Sometimes it is found in conjunction with a dermoid tumor of the cornea (see page 223).

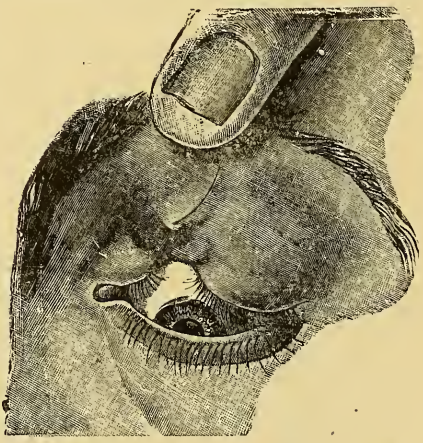


Fig. 293.—Congenital Coloboma of the Upper Lid.

Under the name of *epicanthus* is denoted a fold of skin which juts out on both sides of the dorsum of the nose and projects over the inner angle of the eye so as to partly cover it. In the Mongolian race a moderate degree of epicanthus is the rule, and produces the characteristic appearance of the palpebral fissure in these people. In the Caucasian race there

is not infrequently observed in children a slight degree of epicanthus, which disappears again when in the process of growth, the dorsum of the nose becomes more prominent. Higher degrees of epicanthus, which persist all through life, must be regarded as a malformation, and are sometimes found in connection with other congenital defects (e. g., ptosis). The fold of skin forming the epicanthus disappears if with our fingers we pick up the skin upon the dorsum of the nose into a vertical fold and thus shorten it horizontally. Upon this observation depends the operation for epicanthus recommended by Ammon, which consists in the excision of an elliptical piece of skin upon the dorsum of the nose. We may also excise the projecting fold of skin itself.

Among anomalies of the lids which are sometimes of congenital occurrence, the following also must be mentioned: Ptosis, distichiasis, abnormal narrowness of the lids, and, as the extreme degree of the latter condition, entire absence of the lids (ablepharia); furthermore, symblepharon, ankyloblepharon, and the condition in which the eye is completely covered by the external skin, the latter replacing the lids and forming a uniform covering for the aperture of the orbit (cryptophthalmus of Zehender); and, lastly, cysts in the lower lid, in the case of microphthalmus. [See page 482.]

CHAPTER XIII

DISEASES OF THE LACHRYMAL ORGANS

Anatomy and Physiology

623. Lachrymal Glands.—The lachrymal organs consist of the lachrymal gland and the lachrymal passages.

The *lachrymal gland* (glandula lacrimalis) is an acinous gland with short, branched gland tubules. It consists of two divisions, the larger, known as the superior lachrymal gland, lying in the upper outer angle of the orbit in a depression in the bony wall of the latter, the fossa glandulæ lacrimalis. Excretory ducts of the superior lachrymal gland pass downward and empty into the outer half of the superior fornix conjunctivæ.

The second division of the lachrymal gland—the inferior lachrymal gland—is much smaller, and consists only of one or two lobules, for which reason it is also known as the accessory lachrymal gland. Its lobules lie along the excretory ducts of the superior gland directly beneath the mucous membrane of the fornix (Fig. 294). If the upper lid is everted and at the same time the eye is made to look downward, we often see the conjunctiva of the fornix in the vicinity of the outer angle of the lid pushed forward by a soft mass which is in fact the accessory lachrymal gland. Krause's glands (see Fig. 283, *kr*, and Fig. 294) form a sort of continuation of the lobules of the inferior lachrymal gland along the fornix as far as its inner end. Their structure is that of the lachrymal gland, so that they may be regarded as the ultimate scattered outlying portions of the latter.

624. Lachrymal Passages.—The lachrymal passages begin with the *puncta lacrimalia*. These lie on the free border of the upper and lower lid (upper and lower punctum) and near the inner extremity of the lid at the spot where the tarsus terminates (Fig. 294). They are situated upon small elevations, the lachrymal papillæ (papillæ lacrimales), and form the orifices of the canaliculi lacrimales. These latter, starting from the puncta, run at first vertically for a short distance—i. e., in the upper lid run upward and in the lower lid downward—then they bend at a right angle and become directed toward the lachrymal sac. In so doing they first pass behind the caruncle, and converging more and more, at length reach the lachrymal sac. Into this they empty, either separately or after having united to form a short common trunk.

The *lachrymal sac* (saccus lacrimalis) lies in the inner angle of the eye in the cleft (fossa sacci lacrimalis) which the lachrymal bone forms for its reception. The lachrymal bone bounds the lachrymal sac (*S*, Fig. 284)

nasally, while to the front and temporally it is inclosed by the two branches of the ligamentum palpebrale mediale (*v* and *h*, Fig. 284). This relation of the lachrymal sac to the internal palpebral ligament enables us to determine the position of the former—a matter which is of importance when operations are concerned. If by drawing the lids outward we put them on the stretch and so cause the palpebral ligament to project, the lachrymal sac lies behind the latter, and in such a way as to rise just above it by its cupola or fundus (Fig. 294).

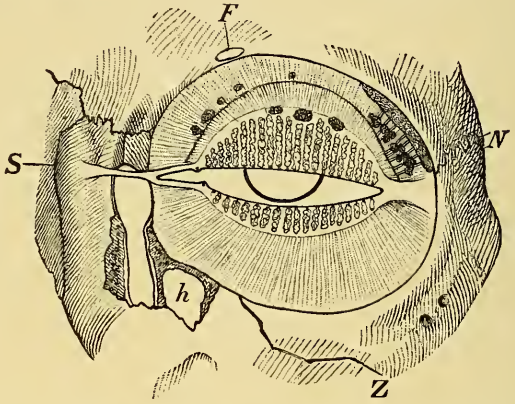
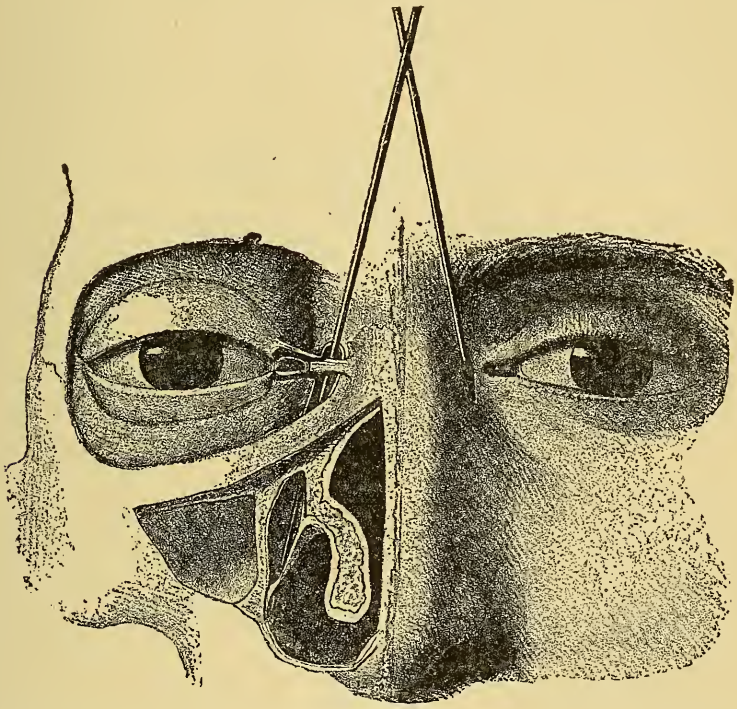


FIG. 294.—SEPTUM ORBITALE AND LACHRYMAL SAC. Natural size.

The skin and the muscular fibers of the orbicularis have been removed from the lids and the parts surrounding them, so that the septum orbitale lies exposed to view within the bony circumference of the orbital cavity. The septum orbitale consists of the tarsus, which is broader in the upper lid, narrower in the lower, and of the fascia tarso-orbitalis. The external extremities of the tarsi are attached by the broad, but not very dense rhaphe palpebralis lateralis to the malar bone, somewhat below the suture, *N*, between this bone and the zygomatic process of the frontal bone. The internal palpebral ligament is narrow, but stout; its anterior limb, which is the only one visible in the drawing, runs from the frontal process of the superior maxilla, *S*, outward, and divides so as to be inserted into the inner extremities of both the upper and lower tarsal cartilages. (At the point of insertion is seen the somewhat projecting papilla lacrimalis.) The fascia tarso-orbitalis, represented in the drawing by the radial lines of shading, runs from the convex border of both tarsal cartilages, and from the palpebral ligaments, to the margin of the orbit, and together with these parts closes in the orbit in front. The tarsal cartilages and the fascia are here supposed to be transparent. Hence, in the former there can be seen the Meibomian glands, which, in consonance with the varying breadth of the tarsus, diminish in height from the center of the latter to its two ends. Moreover, in the upper lid three acinous glands (cf. Fig 283, *w*) are visible along the upper border of the tarsus. Still higher up, a curved line shows the situation of the fornix conjunctivæ. Upon the fornix, especially in its nasal half, lie the acinous glands of Krause, while in the temporal half of the fornix are found lobules similar in character, but more densely packed, representing the inferior lachrymal gland. This adjoins the excretory ducts of the superior lachrymal gland, whose anterior border comes into sight just below the upper margin of the orbit. At the inner and lower margin of the orbit the bone has been chiseled away to show the lachrymal passages. The lachrymal sac lies behind the internal palpebral ligament, its apex rising a little above the latter. The line that in the drawing runs straight upward from the apex of the lachrymal sac to the horizontal suture is the suture between the frontal process of the superior maxilla and the lachrymal bone, upon which two bones the lachrymal sac rests (cf. Fig. 284, *F* and *T*). The lachrymal sac, after undergoing a slight constriction, passes into the nasal duct. To the outside of this is the antrum of Highmore, *h*, which has been opened up and is accessible to view. *Z*, suture between the superior maxilla and the malar bone. *F*, supra-orbital foramen.

At the spot where the cleft of the lachrymal bone merges into the bony canal the lachrymal sac passes into the nasal or *lachrymal duct* (ductus lacrimalis). The point where this transition occurs constitutes the narrowest part of the whole lachrymal channel (Fig. 294), and is therefore particularly liable to the formation of pathological contractions (strictures). From this point the lachrymal duct passes downward and empties into the

nasal fossa below the inferior turbinated body. In its downward course the lachrymal duct deviates a little backward and outward from the vertical. Hence, the two lachrymal channels diverge as they go down, the lachrymal sacs being less far apart than are the lower orifices of the lachrymal ducts. We can represent the course of the lachrymal channel on the living subject by placing a straight sound in such a way as to lie at its upper part upon the middle of the internal palpebral ligament, and below upon the furrow forming the boundary line between the cheek and the alæ



[Fig. 295.—Section of Canaliculi, Lachrymal Sac, and Nasal Duct. (De Wecker.) After Norris and Oliver.—D.]

of the nose [see Fig. 295]. This sound gives precisely the direction of the lachrymal duct (Arlt). If we place a sound in this way on each side of the nose, we see how the sounds diverge as they go down, and we can readily convince ourselves that the degree of divergence differs in different individuals. The divergence, in fact, depends upon the breadth of the root of the nose on the one hand, and upon the breadth of the inferior nasal orifice on the other. These facts are of importance with regard to the operation of sounding the lachrymal duct, in the performance of which the sound must be pushed along in the direction of the duct.

The mucous membrane of the lachrymal sac and that of the lachrymal duct forms one continuous whole. There is, therefore, no sharp dividing line between these two structures. They are mainly distinguished by the fact that the lachrymal sac lies against bone (the lachrymal bone) at one side only, and everywhere else is free, while the lachrymal duct is inclosed on all sides by bony walls. It follows from this that, in engorgement of the lachrymal channels with fluid, it is *only the lachrymal sac which is distended*, so as to appear as a visible swelling at the inner angle of the eye. The lachrymal duct cannot be distended; on the contrary, it is the favorite seat of *constrictions*, which again do not occur in the lachrymal sac. The formation of these constrictions is facilitated by the fact that a dense plexus of wide veins, analogous to the venous plexuses

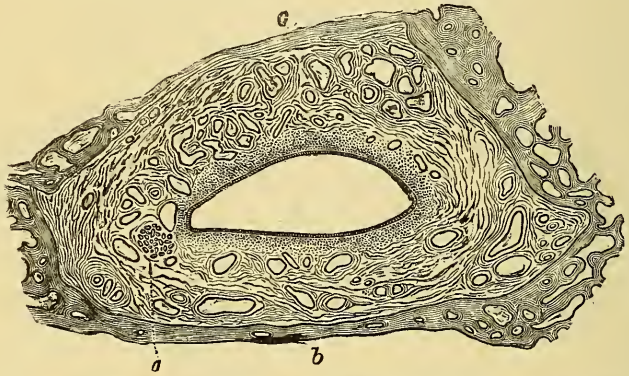


FIG. 296.—CROSS SECTION THROUGH THE LACHRYMO-NASAL DUCT. Magnified 11 × 1.

The lumen of the lachrymal duct is oblong and lined with cylindrical epithelium. The subjacent mucous membrane contains very many lymphocytes, i. e., it has an adenoid character. The submucous tissue is marked by its great abundance of blood-vessels. Most of these are veins; the few arteries are made obvious in the drawing by their double contour. At *a* lie the acini of a mucous gland, whose excretory duct emptying into the lachrymal duct is not traversed in the section here shown. Succeeding the submucous tissue is the wall of the bony canal; *b* is the side of this canal that is directed toward the maxillary antrum, *c*, the side that is directed toward the nasal cavity.

beneath the mucous membrane of the turbinated bodies, is interposed between the mucous membrane of the lachrymal duct and the bony wall (Fig. 296). Swelling of these veins alone is sufficient to contract or to close entirely the lumen of the duct.

The lachrymal passages are always filled with a little lachrymal fluid. If air is found in them, it is to be regarded as a pathological condition.

The mucous membrane of the canaliculi is lined with laminated pavement epithelium, that of the lachrymal sac and nasal duct with a double layer of cylindrical epithelium. The mucous membrane at different spots projects in the form of folds into the lumen of the lachrymal passages—a phenomenon which has been described as a formation of valves. The largest of these folds is Hasner's valve, at the lower orifice of the nasal duct. This, however, is not a true valve, any more than are the others—that is, not a valve which could close up the lumen of the lachrymal channel. On the contrary, it is simply a fold produced by the great obliquity with which the nasal duct

passes through the mucous membrane of the nasal fossa. Like the other folds of mucous membrane in the lachrymal passages, it is not of constant occurrence.

Duplication of the puncta and canaliculi, and also their absence, have been recorded as congenital anomalies.

625. Lachrymal Secretion.—Secretion of *tears* results either from psychic excitation ("weeping") or in a reflex way from irritation of the trigeminus or optic nerve ("watering of the eyes"). Psychic weeping occurs only in man, and even in man is absent in the case of infants. The tears contain only a few solid constituents, the main part of which is sodium chloride (hence "salty" tears). In the normal state the lachrymal gland secretes scarcely any more liquid than is lost by evaporation from the surface of the eyeball, so that but very small quantities of fluid are discharged into the nose. It is only when the secretion is much increased that any considerable quantity of tears is discharged into the nose, where its presence is manifested by repeated blowing of the nose.

The moistening of the eyeball is not due to the lachrymal glands alone. The secretion of the conjunctiva itself, and also of its glands, participates in the performance of this act. Hence it follows that even after removal or degeneration of the lachrymal gland the eye does not become dry.

In the *conduction of tears* into the nose there are two factors to be considered: The entrance of the tears into the lachrymal sac, and their transmission from the latter to the nose.

The conveyance of tears through the puncta into the lachrymal sac is effected by the act of winking. The tears accumulate in the horseshoe-shaped notch in the inner angle of the eye, which forms the lacus lacrimalis into which the puncta dip. Then a winking movement takes place, the fibers of the palpebral portion of the orbicularis contracting. These fibers spring from the internal palpebral ligament and when they contract draw the ligament away from the lachrymal bone. The wall of the lachrymal sac, being connected with the palpebral ligament, is drawn along with it, so that the lachrymal sac is dilated, and the contents of the canaliculi are sucked into the sac. The subsequent conveyance of the tears from the lachrymal sac into the nose is effected by the elasticity of the sac. In virtue of this elasticity, the sac when distended by the tears tends to contract again and thus expel the tears. Hence, in those pathological cases in which the lachrymal sac has lost its elasticity (atony of the sac) we observe that the conduction of tears downward is arrested, even though the nasal duct is completely pervious. The reason why the tears are not driven back again into the canaliculi by the contractions of the lachrymal sac, but pass on into the nasal duct, is that the latter is much wider than the narrow canaliculi.

I. Dacryocystitis Chronica [1]

626. Symptoms.—The patient comes with the complaint of the tears running over (epiphora [2]). On careful inspection we notice that the skin in the region of the lachrymal sac protrudes so as to appear fuller than on the other side. This swelling—tumor lacrimalis—is caused by the sac's being distended by an accumulation of its contents. [In mild cases, at the outset and sometimes for a long time, there may be epiphora and more or less constant obstruction, without evident distention of the sac.—D.] When pressure is made on the tumor the contents are discharged externally through the puncta, and appear, in recent cases, as a purulent, in older cases as a mucous or even limpid liquid. Sometimes, when pressure is made on the lachrymal sac, the contents are forced not through the puncta but downward through the nose. This is the case when, as exceptionally happens, the nasal duct is freely pervious. Associated with these symptoms, which prove distention of the lachrymal sac and abnormality in its secretions, there is usually a contraction (stricture) of the nasal duct. The proof of this is produced if with an Anel's syringe [Fig. 412, *h*] which has a fine but not sharp-pointed cannula we inject water into the lachrymal sac through the lower canaliculus, and at the same time make the patient bend his head forward so that the water which gets into the nose shall not run backward but forward. If stenosis of the nasal duct is present, the water does not flow freely into the nose, but only drop by drop or not at all, and regurgitates from the lachrymal sac through the upper canaliculus. [If in persons with no obstruction of the lachrymal passages we instill a mixture of adrenaline and cocaine into the eye so as to shrink up the mucous membrane of the tear passages and then instil a 30-per-cent solution of argyrol, the latter will gradually make its way down into the nose and, if the patient blows his nose, will stain the handkerchief. In a patient with lachrymal obstruction the argyrol goes down with difficulty or not at all. If in this case we inject the adrenaline-cocaine solution into the canaliculus with the syringe the argyrol that we have instilled will then usually go through to the nose, provided the obstruction is due simply to turgidity of the membrane, and not to a bony or fibrous stenosis.—D.] In the latter case in order to determine the site and extent of the stenosis we first make the passages insensitive by injecting a few drops of a cocaine solution and then try to explore the duct with a Bowman's sound, in doing which we come upon a spot where the duct is contracted or even altogether obliterated.

[1] To the older name blennorrhœa of the lachrymal sac, (dacryocysto-blennorrhœa), which I also formerly used, the objection must be made that it may give rise to misunderstanding, since gonorrhœal affections are frequently known as blennorrhœa, and also that the secretion which is expressed from the diseased tear sac is by no means always *βλέννα*, i. e., mucus, but is frequently pus or clear lachrymal fluid. Against the designation dacryocystitis catarrhalis it may be said that chronic diseases of the tear sac are not always of a simple catarrhal nature, but are sometimes tuberculous or trachomatous—a matter which often cannot be decided by a purely clinical examination.

[2] From *ἐπιφέρεσθαι* to rush upon. We also say *illacrimatio* or *stillicidium* (from *stilla*, drop, and *cadere* to fall).

627. Etiology.—The immediate cause of a chronic dacryocystitis is stricture of the nasal duct. This may be produced, for example, by swelling of the mucous membrane of the duct. The tears can then no longer be fully discharged into the nose. But as new tears are constantly forced, by the act of winking, into the lachrymal sac, the latter becomes more and more filled up and distended. The lachrymal fluid that thus accumulates in the sac soon decomposes. The tears, in fact, carry with them from the surface of the eyeball a quantity of germs, which find the best conditions for development within the sac in the fluid which stagnates there and is kept at the temperature of the body. And actually the contents of the sac in chronic dacryocystitis are seen under the microscope to swarm with micro-organisms of all kinds (especially the pneumococcus). The decomposed fluid irritates the mucous membrane of the sac, which inflames and throws out a pyoid secretion that mixes with the tears stagnating in the sac. The contents of the sac thus grow constantly more turbid and ultimately resemble pus. Chronic dacryocystitis is hence nothing but a catarrhal inflammation of the mucous membrane of the sac. Like all purulent or decomposed fluids, the secretion of the diseased sac is virulent in that it contains pus cocci in great numbers. Owing to this virulence, infection of defects in the cornea readily occurs in chronic dacryocystitis, so that an ulcus serpens develops.

The constriction of the nasal duct that, as has been said, forms the starting point for chronic dacryocystitis, develops, as a rule, in consequence of affections in the nasal cavity. Such affections are:

1. *Inflammation of the nasal mucous membrane.* Under this head belongs coryza in its various forms, whether acute or chronic, and whether a simple catarrhal trouble, or one of scrofulous or syphilitic origin. The inflammation of the mucous membrane of the nose is associated with swelling which, owing to the presence of the submucous cavernous tissue, may very readily reach quite a high degree, so that the nasal cavity is rendered impermeable by it. The inflammation and the swelling extend by continuity to the mucous membrane of the nasal duct, being particularly due to the engorgement of the numerous veins lying beneath the latter, an engorgement which of itself is sufficient to close the lumen of the duct. The inflammation of the mucous membrane then leads to cicatricial contraction of the latter and consequently to strictures in the nasal duct, so that the impermeability of the duct persists even after the inflammatory swelling subsides. In the ordinary form of ozæna (rhinitis atrophicans) there is no swelling even at the beginning, but a cicatricial contraction of the nasal mucous membrane, which may be continued from the inferior orifice of the nasal duct into the latter and thus lead to its constriction. 2. *Ulcers,* such as are apt to be associated with the scrofulous and syphilitic inflammations of the nasal mucous membrane, and also lupous ulcers. As the ulcers heal, cicatricial constriction or even obliteration of the nasal duct

takes place. This is the more to be apprehended if the subjacent bones are also implicated. 3. *Tumors.* These may block the lower orifice of the nasal duct, and thus cause stoppage of the flow of tears. The most frequent tumors of this kind are polypi

Blennorrhœa of the lachrymal sac affects the female more frequently than the male sex, perhaps on account of the more frequent use which the former makes of the lachrymal apparatus. Persons having the bridge of the nose flattened (flat noses and "saddle-noses," particularly when due to hereditary syphilis) also are predisposed to this affection.

Sometimes a chronic dacryocystitis is observed even in new-born children and this may actually be associated with perforation and the formation of fistulæ. The cause of the disease is a belated perforation of the thin diaphragm of mucous membrane which in the fetus closes the lower orifice of the nasal duct. The result is decomposition of the gelatinous contents of the nasal passages and hence inflammation of the latter (Rochon-Duvigneaud). Usually repeated expression of the lachrymal sac suffices to cure the disease; if not we must make the nasal duct pervious by the use of sounds.

Trachoma and *tuberculosis* of the lachrymal sac occur as secondary affections. [Trachoma of the sac is said to be frequent in the Near East where trachoma generally is endemic. Sacs thus diseased are very friable and rupture easily, so that lavage is dangerous (Butler). D.] Tuberculosis of the sac may originate from tuberculosis of the conjunctiva or from tuberculosis (lupus) of the nasal mucous membrane. The lachrymal sac feels very much thickened, and upon opening it we find its inner surface lined with discolored granulations (cf. page 209).

In both the *puncta* and *canaliculi* contraction and even obliteration are sometimes observed, the consequence of which is also epiphora. The most frequent cause giving rise to these conditions is injury of the mucous membrane of these structures due to sounding. To remove them we must endeavor to enter the canaliculus with a conical sound and thus dilate it, or, if necessary, we must slit it up. Occlusion of the canaliculi may also be produced by foreign bodies or by *concretions.* The latter are of a gray or grayish-green color and of friable or hard consistence, and prove to be conglomerated masses consisting of a fungus, the streptothrix Fœrsteri. [The sporotrichon may also form concretions in the canaliculi in cases of sporotrichosis (Bedell—cf. page 210).] In some cases cystoid dilatation of the canaliculi has been found. This arises from an obliteration of the canaliculus at both extremities and the accumulation of fluid in its cavity, so that the tube is gradually distended till it forms a cyst.

In the *puncta* there can be frequently made out a change of position of such a character that the lower punctum is turned outward (forward) instead of looking upward (*eversion of the punctum*). This represents the very first stage of an ectropion —a condition which bears within itself the germ for its own development (see page 681).

628. Course.—Chronic dacryocystitis is an eminently chronic disease, whose course is counted by years. A spontaneous cure may indeed occur, if, as the swelling of the mucous membrane of the nasal duct abates, the lumen of the duct becomes free again, and at the same time the catarrhal inflammation of the sac subsides. This, however, occurs but rarely, because strictures of the nasal duct have formed in the meantime. Hence the rule is that without artificial aid no cure takes place, but the following course of events ensues: The secretion at first purulent becomes after a time

mucous and viscid; at length, owing to atrophy of the mucous membrane, secretion ceases altogether. Then the distended sac contains simply a clear liquid—namely, the tears which have accumulated in it. But still the epiphora keeps on, because the constriction of the nasal duct prevents the conduction of the tears into the nose.

As a result of the constant distention of the lachrymal sac by fluid, its walls at length lose their elasticity. When this condition, called *atonia sacci lacrimalis*, has set in, the tears are no longer carried down into the nose, even if the nasal duct again becomes perfectly pervious. The distention of the lachrymal sac may keep on increasing more and more, so that it presents a fluctuating tumor of the size of a walnut and over. This either projects far forward, or it often extends deep into the orbit, so that the eyeball is displaced by it (exophthalmus). It is filled with a clear fluid; hence the name *hydrops sacci lacrimalis*.

Chronic dacryocystitis causes trouble chiefly through the epiphora, which compels the patient to dry his eye frequently. The epiphora increases in cold weather, and in wind, smoke, etc. If it lasts a long time it leads to chronic catarrh of the conjunctiva and to blepharitis ulcerosa. If these two conditions are found in one eye alone, they must always excite the suspicion of there being a lesion of the lachrymal sac. In the subsequent course of the disease the moistening of the lower lid by the tears leads to eczema of the lid, contraction of the skin covering it, and ultimately to ectropion. By these conditions, again, the epiphora itself is increased. In the presence of chronic dacryocystitis ulcers of the cornea may develop from erosions of the latter, and operation wounds also may readily become infected.

629. Treatment.—This must first have regard to the nasal disease which underlies the trouble, and, if this is still present, must use the proper means for its relief. As for the lachrymal sac itself, the patient is to be told to evacuate it very frequently by pressing the finger on the inner canthus. In this way the accumulation of secretion and its consequent decomposition, together with the dilatation of the sac, are combated. In addition it is advisable to cleanse the sac by syringing with an Anel's syringe (§ 811).

[A great many obstructions of the milder type, especially in the earlier stages, can be relieved by syringing. A 30-per-cent solution of argyrol is instilled, and then the canaliculus is syringed with a mixture of adrenaline and cocaine. After the first or second treatment the argyrol usually goes through to the nose, at first imperfectly, later freely. If there is obstruction in the nose itself, adrenaline may be applied to the inferior turbinate before syringing. The physician may do the syringing once or twice a week, according to circumstances and directs the patient to instil two or three times a day a solution of adrenaline followed by a 20-per-cent solution of argyrol.—D.]

In simple eversion of the punctum, without ectropion proper the epiphora can be relieved by slitting the canaliculus (§ 812). The tube is thus converted into an open groove, which looks backward and hence dips into the lacus lacrimalis and takes up the tears.

The main burden of treatment lies in the removal of the stricture of the nasal duct, by gradual dilatation with sounds according to Bowman's method (see § 813).

The duration and the success of the treatment with sounds depend upon the nature of the contraction. The most favorable cases are those in which the contraction is caused by simple inflammatory swelling of the mucous membrane; those in which cicatricial strictures are present are less favorable, and the least favorable are those in which the nasal duct is completely obliterated at some spot. Cases of the latter kind do not, for the most part, admit of a permanent cure. Even in the most favorable cases the duration of the treatment amounts to from four to six weeks, and it usually requires several months. If strictures due to cicatrices are present, recurrences may set in, owing to renewed contraction of the cicatricial tissue; and, in fact, this unfortunately occurs so often that permanent cures form the exception.

Those cases which cannot be cured by treatment with sounds require, if they give rise to considerable annoyance, *extirpation of the lachrymal sac* (see § 816).

Extirpation of the lachrymal sac is suitable in those cases in which the treatment by sound is likely to be without result. This is the case where there are very extensive cicatricial contractions or complete obliteration of the nasal duct. This is still more the case if there are at the same time demonstrable changes in the bone, shown either by our coming upon bared and roughened bone in the act of sounding, or by there being an externally visible implication of the bone manifested by a sinking in of the nose (in consequence of syphilis). Furthermore, cases of atony and dropsy of the lachrymal sac, are suitable ones in which to perform extirpation of the sac, since in these cases a normal escape of tears could not take place even after the nasal duct has been made pervious. Moreover, removal of the tear sac is very frequently done in cases in which treatment by sounds would be feasible, but in which the patient's external circumstances make this very protracted method of treatment impossible. This is particularly the case in patients of the working class.

While the treatment by sounds in favorable cases restores the normal conduction of the tears, the possibility of this restoration is forever prevented by the operation for destruction of the lachrymal sac. Hence a condition of epiphora always remains, which, however, reaches a troublesome degree only when, in consequence of irritation of the conjunctiva, there is an excessive secretion of tears, and this can be relieved by extirpation of the inferior lachrymal gland (see § 818). [This seems rarely necessary. In order to obviate the possibility of this continuous epiphora, other operations, e. g.,

Toti's and *West's* (see § 817) have been devised, by which permanent drainage is maintained.—D.] To compensate them for the epiphora the patients are relieved of the presence of a constantly suppurating cavity which continually exposes them to the danger of getting an ulcus serpens of the cornea, and also usually gives rise from time to time to acute phlegmons (dacryocystitis acuta).

[There are cases in which the dacryocystitis is of such mild type that we do not feel justified in removing the sac, but in which the dacryocystitis may be a source of temporary danger, e. g., in the case of operations on the eyeball, in which it might give rise to infection. In such cases we may perform *incision of the sac* or *cauterization* or *ligature of the puncta* (see §§ 814 and 815).—D.]

II. Dacryocystitis Acuta (or Phlegmonosa)

630. Symptoms.—In an individual who suffers from chronic dacryocystitis, a violent inflammation may suddenly develop in the region of the tear sac. The skin in its vicinity is then reddened and greatly swollen; the swelling also extends to the lids, and even to the conjunctiva, in which there is chemosis. The inflammation is accompanied by fever and violent pain, so that the patient is deprived of sleep for several nights. After some days the skin at the apex of the swelling takes on a yellowish discoloration, and finally becomes perforated, when quite a large quantity of pus is discharged. Upon this the pain abates and soon ceases altogether, and the swelling also rapidly goes down. Later on, there is discharged from the perforation a fluid which at first is purulent, afterward mucous, and at length perfectly clear like water. Ultimately nothing but the tears which are forced into the sac run out through the perforation, which is hence called a *lachrymal fistula*.

As long as the fistula remains open the patient is safe from any new attack of inflammation. But if the fistula closes and tears reaccumulate in the sac, recurrence of the acute dacryocystitis may ensue.

Acute dacryocystitis consists in a purulent inflammation of the connective tissue surrounding the lachrymal sac. This inflammation leads to purulent disintegration of the submucous tissue with the formation of an abscess which ruptures externally. Acute dacryocystitis is accordingly a phlegmon. Chronic dacryocystitis, on the contrary, is a catarrhal inflammation of the mucous membrane itself, in which the purulent secretion of the latter is deposited upon the surface only. The connection between the two diseases consists in the fact that chronic precedes acute dacryocystitis and gives rise to it; for the catarrhally inflamed sac is filled with decomposed secretion, and it only requires the presence of a small defect in the epithelial covering of the mucous membrane of the sac to enable the micro-organisms of the secretion to penetrate into the submucous tissue, where they excite suppuration and cause dacryocystitis.

Acute dacryocystitis almost always originates from a chronic dacryocystitis; consequently the patients state that epiphora has existed for quite a long time before the acute inflammation, which they often call by the name of erysipelas. It is only

in very rare cases that a caries of the lachrymal bone gives rise to acute dacryocystitis. The diagnosis of dacryocystitis is easy to make from the situation of the swelling, which corresponds to the region of the lachrymal sac. A characteristic feature is that the swelling extends up but little above the inner palpebral ligament. Swellings which lie mainly above the palpebral ligament are to be regarded as connected not with the lachrymal sac but mostly with the frontal sinus (discharge of an empyema beneath the skin). It is the exception for abscesses of other kinds, e. g., a furuncle in the skin or a periostitic abscess, to occur in this spot. The spot where the pus breaks through, however, does not always correspond with the position of the sac. It usually lies beneath, being quite a distance below and to the outside of it. The reason for this is that pus sinks down beneath the skin and at the same time travels outward along the inferior margin of the orbit, because the skin along this is attached to the bone by rather firm connective tissue. The farther the pus sinks before it makes its way through the skin the longer will be the fistulous canal that remains. That an opening in the skin which lies far below and to the outer side may still be a lachrymal fistula can be demonstrated by our being able to push a sound through from the opening into the lachrymal sac. If we should not succeed in this, we inject a colored liquid from the canaliculus into the sac; we then see it flow out again by the orifice of the fistula.

Later on in their course, lachrymal fistulæ contract and sometimes become so minute that they are permeable by nothing but a fine bristle. The external orifice of these *capillary fistulæ*, as they are called, is then scarcely visible with the naked eye. We simply notice a drop of clear lachrymal fluid appearing from time to time upon the skin beneath the lachrymal sac; it is only upon careful examination that we notice the capillary opening.

631. Treatment.—If we are dealing with an acute dacryocystitis in its very inception, we may try to prevent the development of an abscess. With this end in view, we sedulously express the fluid from the lachrymal sac, inject it with antiseptic solutions, and in the intervals apply a pressure bandage, which constringes it.

If the inflammation has passed the initial stage, it is idle to endeavor to prevent the formation of an abscess; besides, the methods of syringing, expression, and compression, given above, could not be employed, on account of the swelling and painfulness of the parts. The only thing to do now is to hasten the formation of the abscess, an object which is best attained by the use of moist and warm compresses. As soon as fluctuation makes itself apparent, we incise the lachrymal sac and in addition treat by sounding or more radical means the chronic dacryocystitis that forms the basis of the condition (see § § 815 and 816).

632. Diseases of Lachrymal Gland.—It is extremely rare for the *lachrymal gland* to be the seat of disease. Among such diseases belong: 1. Inflammation (dacryo-adenitis). This may go on to resolution; in other cases, suppuration of the gland with discharge of the pus externally has been observed, a fistula of the lachrymal gland remaining afterward. There have even been described cases of bilateral dacryo-adenitis which ran either an acute or more frequently a chronic course; some of these cases were complicated with simultaneous swelling of the parotid or other salivary glands (Mikulicz's disease; see § 733). 2. Tuberculosis and syphilis of the gland. 3. Neoplasms, including carcinomata, adenomata, cylindromata, lymphomata, chloromata, sarcomata, and mixed tumors resembling those of the parotid gland. 4. Cystoid dila-

tation of a duct of the gland—a condition called dacryops. 5. Atrophy of the gland in xerophthalmus (Arlt; see page 221).

The superior lachrymal gland sometimes requires extirpation (see § 818).

Removal of the *inferior lachrymal gland* (see § 818) is done in order to relieve a troublesome epiphora, when other measures have failed; as, for example, when after a chronic dacryocystitis the permeability of the nasal duct has been restored by sounding and nevertheless the epiphora continues, or when as a result of extirpation of the lachrymal sac drainage of the tears in the regular way has become impossible.

Hence I am accustomed to perform extirpation of the inferior lachrymal gland right after an extirpation of the lachrymal sac. [In the opinion of many, including the translator, this is not often necessary, simple extirpation of the sac not being followed by troublesome epiphora.—D.]

633. Epiphora and Absence of Lachrymal Secretion. —Disturbances in the function of the lachrymal apparatus find expression either under the form of epiphora or of absence of the tears. *Epiphora* is an exceedingly frequent symptom of the most various condition, and is either based upon an increase in the secretion or a hindrance to the discharge of the tears. The former occurs physiologically in weeping, and also in the presence of all sorts of irritants affecting the terminal expansion of the trigeminus and its vicinity. Among these are cold wind, air rendered foul by smoke, etc., foreign bodies in the conjunctival sac, inflammations of the eye and its adnexa, affections of the nose, and neuralgia of the first and second branches of the trigeminus. Furthermore, irritation of the retina by strong light causes increased tear secretion. Interference with the conduction of tears into the nose may have its cause either in interference with the normal closure of the lid or in anomalies of the lachrymal passages. Among affections producing the former condition are to be mentioned paralysis of the orbicularis, shortening or ectropion of the lids, notching of the border of the lid, and even simple eversion of the lower punctum; to the latter belong all the affections of the lachrymal channels which have been treated of in this chapter. In old people it is an extremely common thing for the eyes to water especially out in the open air in cold weather, without there being any discoverable lesion of the conjunctiva or the tear passage. For this senile epiphora instillation of the collyrium adstringens luteum [see page 150] is the best remedy. If we find such an epiphora without discoverable cause in younger persons, there is probably an excessive irritability of the mucous membrane of the nose which excites increased secretion of tears in a reflex way. So, too, pungent odors, such as the vapor of ammonia, horse-radish, etc., which irritate the terminal extremities of the trigeminus in the nasal mucous membrane, often bring the tears to our eyes; and lachrymation is likewise very often present in severe coryza. A like connection also exists in the contrary sense, in that a bright light striking upon the eye excites an impulse to sneeze, as is observed especially in children with photophobia, when the attempt is made to open their eyes for purposes of examination. In cases of epiphora for which no other cause can be found, it is requisite to examine the nose carefully and treat it according to the indications.

The opposite condition, namely, *abolition* of the lachrymal secretion, is extremely rare. It is found in xerophthalmus, in consequence of occlusion of the excretory ducts of the lachrymal gland; also in paralysis of the trigeminus, and in facial paralysis when the lesion is situated very high up in the nerve. It is hence supposed that the fibers destined for the innervation of the lachrymal gland start from the brain along with the facial, and run with the nervus petrosus superficialis major to the nasal ganglion of the trigeminus, by the second branch of which they reach the lachrymal gland through the nervus subcutaneus malæ (Goldzieher). A purely nervous disturbance lies at the bottom of those cases in which persons declare that formerly they wept a good deal, but that for a long time past they have not been able to weep, even when very much distressed.

CHAPTER XIV

DISTURBANCES OF MOTILITY OF THE EYE

Anatomy and Physiology of the Ocular Muscles

634. The ocular muscles are distinguished into extrinsic and intrinsic. The latter, also called the interior muscles of the eyes, are the sphincter pupillæ and the ciliary muscle. Of these we shall have something to say later; at present we shall concern ourselves only with the extrinsic muscles. These are six in number, four straight and two oblique.

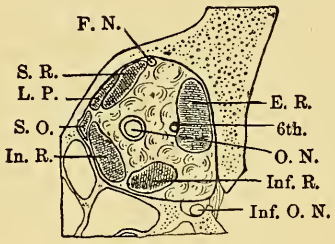


Fig. 297.

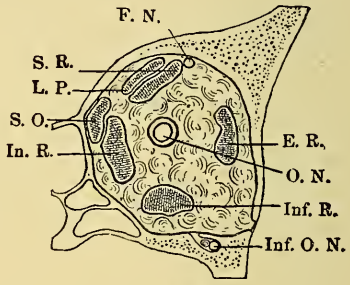


Fig. 298.

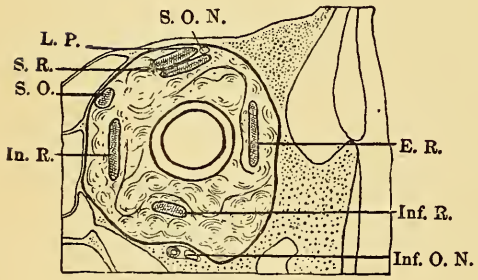


Fig. 299.

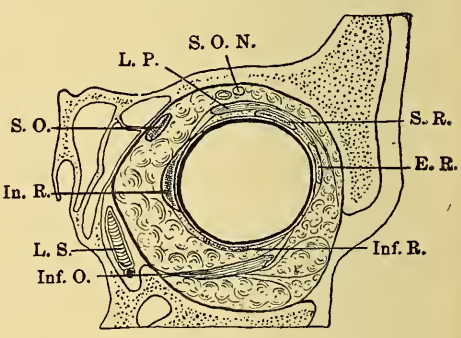


Fig. 300.

[Figs. 297-300.—Frozen Frontal Sections of Left Orbit seen from before. (After Dwight from Norris and Oliver.)

Fig. 297.—Section about 12 mm. behind globe. *S. R.*, superior rectus, *L. P.*, levator; *S. O.*, superior oblique; *In. R.*, internal rectus; *Inf. R.*, inferior rectus; *E. R.*, external rectus; *O. N.*, optic nerve; *F. N.*, frontal nerve; *Inf. O. N.*, infra-orbital nerve; *6th*, sixth nerve.

Fig. 298.—Section about 5 mm. behind globe. Letters as in preceding figure.

Fig. 299.—Section about 3 mm. in front of back of globe. Letters as before.—*S. O. N.*, supra-orbital nerve.

Fig. 300.—Section near equator of globe. Letters as before.—*Inf. O.*, inferior oblique. *L. S.*, lachrymal sac.—D.]

The four straight muscles are the rectus medialis sive internus, lateralis sive externus, superior, and inferior. All four take their origin from the apex of the orbit along the bony circumference of the foramen opticum (*F*,

Fig. 301), and from this point run forward, diverging as they go. They thus bound a funnel-shaped space, the muscular funnel (*t t*) [see also Figs. 297-300], the apex of which lies at the foramen opticum, while the eyeball forms its base and the optic nerve (*o*) runs along its axis. The external and internal recti muscles (*i* and *e*) are inserted into the sclera to the outer and inner side of the cornea; the superior rectus (*su*) has its insertion (s_1) above, the inferior rectus below the cornea. The attachment is effected by means of short tendons, which spread out in the form of a fan and become fused with the sclera, which in this way is thickened in its most anterior portion.

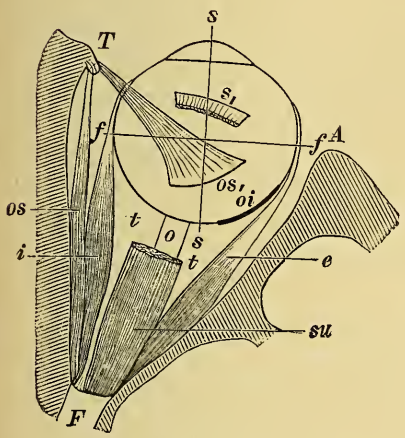


Fig. 301.

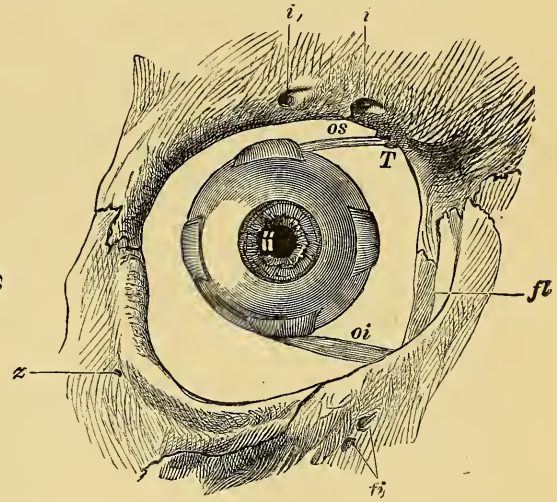


Fig. 303.

Fig. 301.—Horizontal Section through the Orbit (Schematic). Natural size.

The apex of the orbit is formed by the foramen opticum, *F*. The external wall of the orbit does not reach as far forward as the internal, so that its anterior margin, *A*, lies in the same plane as the equator of the eyeball. At the anterior extremity of the internal wall of the orbit is shown the trochlea, *T*, which in reality lies in the upper and inner angle of the orbit, and hence would not be met with in the cross section of the orbit that is here represented. The external rectus, *e*, and the internal rectus, *i*, bound the muscular funnel, *t t*. A portion of the superior rectus (*su*) is cut away to show the optic nerve, *o*. s_1 is the insertion of the superior rectus upon the eyeball, lying oblique to the margin of the cornea. os_1 is the fan-shaped insertion of the tendon of the superior oblique, *os; oi*, the line of insertion of the inferior oblique. *f f* is the frontal, *s s* the sagittal axis of the eyeball.

Fig. 302.—Anterior Orifice of the Orbit with the Eyeball. Natural size.

The tendons of the four recti muscles are cut off near their insertion upon the eyeball, but the inferior oblique, *oi*, and the tendon, *os*, of the superior oblique are left entire. The latter comes out from the loop of the trochlea, *T*. To the temporal side of the trochlea lies the supra-orbital notch, i_1, and somewhat to the outside of this there is a foramen, i_1, which is not regularly present, for a branch of the supra-orbital nerve. In this case the infra-orbital foramen, *fi*, is also abnormally divided into two distinct foramina. *z* is the orifice of the zygomatico-facial canal, *fi* is the lachrymal fossa. Comparison with Fig. 294 shows that the orbit represented in the former is much lower than the one here depicted, but is broader in the horizontal direction.

The two oblique ocular muscles are the obliquus superior and inferior. Their course is more complicated than that of the straight muscles. The *superior oblique* (*os*, Fig. 301) also arises from the margin of the optic foramen and runs forward upon the upper and inner wall of the orbit as far as the trochlea, before reaching which it passes into its tendon. The trochlea itself (*T*, Figs. 301 and 302) lies a little behind the upper and

inner margin of the orbit. It consists of a firm fibrous loop through which the tendon of the muscle is carried in such a way as to be able to glide up and down in it. After traversing the trochlea, the tendon bends backward at an acute angle, and passes beneath the superior rectus to the eyeball. Here it spreads out in the form of a fan, and is inserted in the upper half of the eyeball about in the vertical meridian and behind the equator (os_1, Fig. 301).

The *inferior oblique* arises from the lower margin of the orbit near its inner extremity (*oi*, Fig. 302). From here it runs upward and outward [beneath the inferior rectus] and arrives at the outer side of the eyeball, into which it is inserted about in the horizontal meridian and also behind the equator (*oi*, Fig. 301).

The tendons of the ocular muscles before reaching the sclera must pass through the fascia of Tenon (fascia bulbi) which surrounds the eyeball. In the spot where a tendon of the muscle perforates the fascia, the latter does not present a corresponding aperture, but is reflected backward upon the tendon (*e* and e_1, Fig. 1). It ensheathes the tendon and further back is continuous with the fascia which envelopes the muscle itself. By these "*lateral invaginations*," therefore, the tendons are connected with Tenon's capsule—a fact which is of importance with regard to the operation for squint.

The *lines of insertion* of the four recti muscles are situated at unequal distances from the margin of the cornea, and usually, too, are not quite concentric with it. Moreover, they are not perfectly symmetrical in their relation to the horizontal and vertical meridians. The mean variations in regard to the position of the lines of insertion are shown as accurately as possible in Fig. 303, which represents the anterior half of the eyeball projected upon a plane. In it are marked the distances of the lines of insertion from the cornea in millimeters, as I have found them from the mean of a great number of measurements.

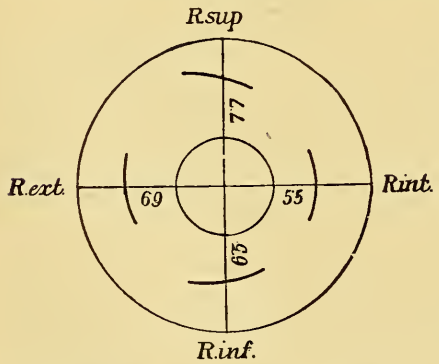


Fig. 303.—Lines of Insertion of the Four Recti Muscles projected upon a Plane. Natural size.

The muscles are surrounded by *fasciæ* which are continuous anteriorly with Tenon's capsule at the spot where the latter is reflected upon the tendons of the muscles. Lateral prolongations of the fasciæ unite the muscles together, and also pass from them to the bony wall of the orbit. By means of this system of fasciæ pervading the orbit, the contents of the latter are fixed in place. It is owing to them that the eye does not leave its place when performing its movements, but turns about a fixed center. The continuations of the fasciæ, passing from the muscles to the walls of the orbit, act as a sort of restraining apparatus [*check ligaments*] which prevent extreme excursions of the eyeball (Merkel, Motais). They are most strongly developed upon the internal

and external recti (*fi* and *fe*, Fig. 284). [The superior oblique has two check ligaments, one attached to the direct, the other to the reflected portion of the tendon. The inferior oblique has no check ligament, its place being taken by the ligament attached to the reflected tendon of the superior oblique.—D.] The levator palpebræ superioris, too, which is associated in action with the superior rectus, is united to it directly by bands of fascia [See page 656. The inferior rectus is similarly connected with the inferior oblique]. Furthermore, bands of fascia pass from the levator to the skin of the upper lid and also to the upper retrotarsal fold, so that these structures follow the movements of the eyeball and the upper lid when these are elevated. An analogous apparatus comes into play when the eyeball is depressed, bands of fascia running from the inferior rectus into the lower lid and to the lower retrotarsal fold [see page 656].

635. Actions of the Muscles.—The movements of the eyeball take place freely in all directions as in a ball-and-socket joint (arthrodia). The movements are performed in such a way that the eyeball, as a whole, undergoes no change of place; it simply rotates about a center of movement which corresponds approximately to the center of the eye.

We can imagine all movements of the eyeball resolved into components which correspond to three *primary axes*. These are perpendicular to each other and cross at the center of movement. One of these is vertical; the movements which take place about it are the lateral movements of the eye—that is, the movement to right and left, or to the outside (abduction) and inside (adduction). The frontal axis (*f f*, Fig. 301), runs from right to left and the movements that correspond to it are those of elevation and depression of the eyeball. The sagittal axis (*s s*, Fig. 301) runs from before backward, and coincides with the line of vision. The movements which take place about it are known under the name of wheel rotation [torsion] of the eyeball; by virtue of them the upper extremity of the vertical meridian of the eyeball is inclined outward or inward.

A simple action—simple in the sense of rotating the eyeball about only one of the three primary axes—belongs only to the internal and external recti, which act merely to turn the eye inward and outward. The action of the other four ocular muscles is a complicated one, and if we seek to determine the axes about which they actually rotate the eyeball, we find that these do not coincide with any one of the three primary axes. Fig. 304 represents schematically the action of these muscles decomposed into its components.

The *superior rectus* runs from the apex of the orbit not only forward but also a little outward, in order to reach the eyeball. Its direction, hence, does not coincide exactly with the sagittal axis of the eye, but forms with it an angle, directed backward and inward (Figs. 301 and 305 A). Hence, as its insertion falls in front of the center of rotation of the eye, it will not only elevate the latter, but also adduct it, too. For the same reason it also so rolls the eye that the upper end of the vertical meridian tilts inward [intorsion].

The *inferior rectus* likewise deviates somewhat to the outside in its course forward. Hence, besides lowering the eye, it has an adducting action. Furthermore, it so rolls the eye that the upper end of the vertical meridian tilts outward [extorsion].

To learn the action of the *superior oblique* we have merely to consider that section of it which lies between trochlea and eyeball; the trochlea being, so to speak, the physiological origin of the muscle. Its action primarily consists in rolling the eye, so that the upper end of the vertical meridian tilts inward. Since, furthermore, it is inserted into the posterior half of the eye, and this insertion lies below the trochlea (*os*, Fig. 302), the posterior half of the eye is raised and the cornea consequently depressed when it contracts. The superior oblique also produces abduction of the eye, since it is inserted behind the center of rotation of the eye (Fig. 301, os_1), and in its contraction draws the posterior half of the eye inward, so that the cornea goes outward. The superior oblique accordingly acts to roll [intort], depress, and abduct the eye.

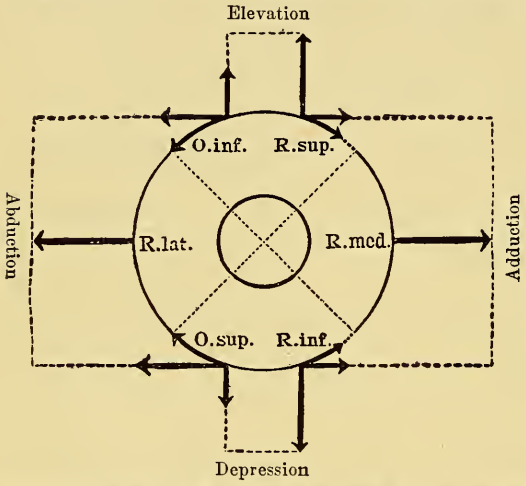


FIG. 304.—SCHEMATIC PLAN SHOWING THE ACTION OF THE OCULAR MUSCLES. (After Marquez.)

The action of each muscle is shown by arrows, which represent this action as decomposed into its components, according to the parallelogram of forces. The components representing torsion are drawn on the outer circle and indicate with their apices the position of the extremity of the vertical meridian, when the upper end of the latter has been tilted by the muscle inward or outward, as the two dotted lines crossing in the center of the figure represent. [The curved arm, in other words, shows the direction in which the vertical meridian is tilted and the amount of tilting. —D.] The inferior oblique, in accordance with its elevating action, is drawn on the upper rim of the outer circle, and the superior oblique, because of its depressing action, is drawn on the lower rim. There are shown by this plan: (1) That the action of the rectus medialis and rectus lateralis alone is simple, that of all the other muscles being made up of the [two or] three components. (2) That for all movements, except the purely lateral ones, [two or] three muscles act in concert. (3) That when this occurs, two out of the three components of each muscle are in large part nullified, because opposed by those of other muscles, so that in general only the third set of components, which have the same direction and hence act as if added together, come into play. [See also page 719.—D.]

The *inferior oblique* accomplishes a torsion of the eye opposite to that effected by the superior oblique—i. e., a torsion in which the upper extremity of the vertical meridian is inclined outward. Since its origin in the margin of the orbit lies below its insertion upon the posterior half of the eyeball (*oi*, Fig. 302), it draws this half downward and thus elevates the cornea. Furthermore, since it draws the posterior half of the eyeball inward, it abducts the eye. The action of the inferior oblique is accordingly to roll [extort], elevate, and abduct the eyeball.

In the case of muscles whose method of action is complicated (and, with the exception of the first pair, this is the case with all the muscles), the *effect of the individual components* of which the total action is made up varies in intensity according to the position which the eyeball happens to occupy. As an illustration, we may explain how this is in the case of the superior rectus. When the eye is looking straight forward, so that its visual line coincides with the sagittal axis of motion, *S S*, (Fig. 305 A), the plane of action of the superior rectus forms with both the visual and the sagittal axis an angle of about 23°—this angle having its branches directed backward. Consequently, the muscle, besides causing an elevation of the eye, also adducts and rolls [intorts] it. If now the eye is turned out 23° (Fig. 305 B), the plane of action of the muscle coincides with the visual plane, *G* G_1. Then the action of the muscles will be

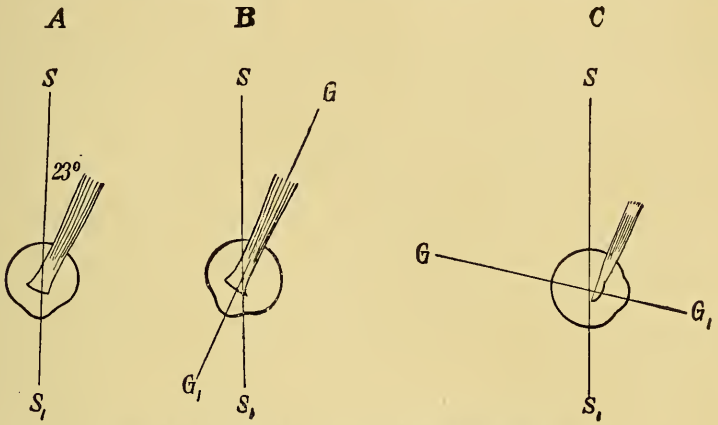


Fig. 305.—Method of Action of the Superior Rectus.
A, in looking straight forward; B, in abduction; C, in adduction; *S* S_1, sagittal axis of motion; *G* G_1, visual axis.

simply one of elevation, since the other two components disappear. On the other hand, the more the eye is turned inward, the more do the two components of adduction and torsion preponderate. They would reach their maximum if the eyeball could be turned far enough inward for its visual axis, *G* G_1, to form a right angle with the plane of the muscle (Fig. 305 C); and, on the other hand, the action of elevation would then have become reduced to nothing. In like fashion, the action of the individual components can be deduced from the position of the eye for every other ocular muscle as soon as the course that it takes is accurately known. This fact is of importance for the diagnosis of paralyses of the ocular muscles, the failure of the paralyzed muscles being particularly marked in the direction of one or the other of the components of its action, according to the different way in which the eye is directed.

[**636. Main and Subsidiary Actions.**—Extending the foregoing analysis to all the muscles, we arrive at the following conclusions :

1. Each eye muscle except the internal and external rectus has a main and a subsidiary action.

2. The main action increases as the subsidiary decreases, and vice versa.

3. The main action increases in one special direction of the gaze.

These facts are shown in the table subjoined.

	Main Action	Subsidiary Action
Superior Rectus	Moves eye up; action increases as eye is turned out; becomes nil when eye is turned in.	Adducts eye and rotates vertical meridian inward; action increases as eye is turned in. Raises upper lid.[1]
Inferior Oblique	Moves eye up; action increases as eye is turned in; becomes nil when eye is turned out.	Abducts eye and rotates vertical meridian outward; action increases as eye is turned out.
Inferior Rectus	Moves eye down; action increases as eye is turned out; becomes nil when eye is turned in.	Adducts eye and rotates vertical meridian out; action increases as eye is turned in. Pulls down lower lid.[1]
Superior Oblique	Moves eye down; action increases as eye is turned in; becomes nil when eye is turned out.	Abducts eye and rotates vertical meridian in; action increases as eye is turned out.—D.]

637. Antagonists.—If we call muscles which rotate the eye in opposite ways about one of the main axes *antagonists*, the muscles may be grouped as follows:—

The internal and external recti rotate the eye about the vertical axis exclusively and are, therefore, the only perfect antagonists.

The superior and inferior recti rotate the eye in opposite ways about the frontal axis (to produce elevation and depression) and about the sagittal axis (to produce torsion), and to this extent are antagonists; but both act as adductors.

The superior and inferior obliques rotate the eye in opposite directions about the sagittal axis (to produce torsion) and the frontal axis (to elevate and depress), acting thus as antagonists; but both act alike to produce abduction.

[The four recti are antagonistic to the obliques in the sense that the former retract the eye, and the latter draw it forward.—D.]

[Opposing muscles act not only to check, reverse or modify the movement initiated by the antagonist, but also by their counteraction make the movement of a moving eye steadier (see pages 719 and 721) and furthermore maintain the *balance* of the eye when at rest. Quite certainly all the muscles take part in this action, so that when the eye is at rest it is maintained in position by the balanced contraction of all six muscles. This is especially the case when no one of the six is predominantly acting, but all are equally relaxed, yet maintain their normal tonus. When this is the case, the eye is said to be in the *primary position* (or *position of equilibrium*). In normal subjects each eye is then directed straight ahead in the horizontal plane, the visual axes being horizontal and parallel with the sagittal plane. When otherwise directed the eye is said to be in a *secondary position*.—D.]

[**638. Monocular Movements.**—Rarely does one muscle alone effect a movement of the eye. This [possibly] occurs when the eye is carried straight out or straight in from the primary position, in which event simply the external or internal rectus [may] act. But in all other movements

[1] From fascial attachments of the tendon to the lid.

two or even three of the eye muscles always take part. [In fact, as the following scheme shows, in probably every movement of the eye, including simple adduction and abduction, from three to five muscles take part, some acting directly, others serving to steady the eye in its course.

ABDUCTION

The eye is carried straight *out* by the external rectus assisted especially toward the end of its course, by the two obliques.[2] The latter nullify each others' vertical and torsion action, so that the eye moves straight out, and its vertical meridian remains vertical. The external rectus pulls the outer canthus outward.

ADDUCTION

The eye is carried straight *in* by the internal rectus assisted, especially toward the end of its course by the superior and inferior recti.[2] The latter nullify each others' vertical and torsion action, so that the eye moves straight inward, and its vertical meridian remains vertical. The inner canthus is pulled back and inward.

ELEVATION

The eye is carried *straight up* by the superior rectus and the inferior oblique, the external and internal recti probably acting to steady it in its course. The two elevating muscles counteract each other so far as regards lateral action and torsion, so that the eye moves straight up and the vertical meridian remains vertical. The superior rectus helps to lift the upper lid.

In moving *up and out,* the eye is carried up mainly by the superior rectus, out by the external rectus assisted by the inferior oblique. The vertical meridian is tilted outward.[3]

In moving *up and in,* the eye is carried up mainly by the inferior oblique, in by the internal rectus assisted by the superior rectus. The vertical meridian is tilted inward.[3]

DEPRESSION

The eye is carried *straight down* by the inferior rectus and the superior oblique, the external and internal recti probably acting to steady it in its descent. The lateral and the torsion actions of the two depressors nullify each other, so that the eye moves straight down and its vertical meridian remains vertical. The inferior rectus assists in depressing the lower lid.

In moving *down and out,* the eye is carried down mainly by the inferior rectus, out by the external rectus assisted by the superior oblique. The vertical meridian is tilted inward.

In moving *down and in,* the eye is carried down mainly by the superior oblique, in by the internal rectus aided by the inferior rectus. The vertical meridian is tilted out.—D.]

[For the amount that the eye can be carried in the several directions, see § 676.—D.]

639. Binocular Movements.—When we look with both eyes the muscles of one eye work with those of the other in such a way that the

[2] [When the eye is abducted the superior and inferior recti also act, but as they have little or no adducting power when the eye is turned out, and as their torsion and vertical actions nullify each other, all they can do is to steady the eye in its course by their mutually counteracting pull. The same is true of the two obliques when the eye is adducted.—D.]

[3] [When the eye is abducted the lateral and torsion action of the superior rectus is slight and that of the inferior oblique marked; on the other hand, the elevating power of the latter is nil and that of the superior rectus considerable. Hence if both muscles act, the eye is carried up almost exclusively by the superior rectus while the preponderating lateral action of the oblique carries it still further out, and tilts its meridian outward. Similar reasoning applies to the statements made regarding the lateral movements and the position of the vertical meridian in the other directions of the gaze.—D.]

visual lines of the two eyes intersect in the object of fixation. To effect this the eyes either both move in the same direction (i. e., both to the right, both up, etc.) [parallel (or conjugate) movements] or both move so as to produce convergence or divergence [disjunctive movements].

[**640. Field of Action of Muscles; Cardinal Directions.**—The muscles of the two eyes may be grouped according to their main action as follows:

I. Lateral Rotators

Right Rotators	*Left Rotators*
(Carrying both eyes to right.)	(Carrying both eyes to left.)
R. external rectus	R. internal rectus
L. internal rectus	L. external rectus

II. Elevators

Right-hand Elevators	*Left-hand Elevators*
(Acting as elevators mainly when eyes are directed to right.)	(Acting as elevators mainly when eyes are directed to left.)
R. superior rectus	R. inferior oblique
L. inferior oblique	L. superior rectus

III. Depressors

Right-hand Depressors.	*Left-hand Depressors.*
(Acting as depressors mainly when eyes are directed to right.)	(Acting as depressors mainly when eyes are directed to left.)
R. inferior rectus	R. superior oblique
L. superior oblique	L. inferior rectus

The twelve muscles of the two eyes are thus divided into three groups of four each; and each group comprises two pairs of muscles, one muscle of each pair being in the right eye, the other in the left. The two muscles of each pair are called *associates.*

It will be seen that each pair of associates has its predominant *field of action* (i. e., direction of the gaze in which it is most active), viz.

Eyes directed to—	*Muscle predominantly acting—*	
Right	R. external rectus	L. internal rectus
Left	R. internal rectus	L. external rectus
Up and right	R. superior rectus	L. inferior oblique
Up and left	R. inferior oblique	L. superior rectus
Down and right	R. inferior rectus	L. superior oblique
Down and left	R. superior oblique	L. inferior rectus

These six directions of the gaze (right, left, up and right, etc.), are of prime importance in the diagnosis of paralysis and are hence called the *cardinal directions* of the gaze.—D.]

[**641. Conjugate Movements.**—If in the summary of monocular movements given on page 718 we substitute for the expressions in and out their equivalents right and left, as the case may be, we find that the conjugate movements of the two eyes are performed as follows:

Lateral Movements

The eyes are carried to the *right* by the two right rotators, to the *left* by the two left rotators, the eyes being steadied in their course by the elevators and depressors. The vertical meridians remain vertical.

Elevation

The two eyes are carried *straight up* by the four elevators, being also steadied in their course by the external and internal recti. The vertical meridians remain vertical.

In moving *up and right*, the eyes are carried up mainly by the two right-hand elevators, to the right by the two right rotators assisted by the two left-hand elevators. The vertical meridians are both rotated to the right.

In moving *up and left*, the two eyes are carried up mainly by the two left-hand elevators, to the left by the two left rotators assisted by the two right-hand elevators. The vertical meridians are both rotated to the left.

Depression

The two eyes are carried *straight down* by the four depressors, being also steadied in their course by the external and internal recti. The vertical meridians remain vertical.

In moving *down and right*, the eyes are carried down mainly by the two right-hand depressors, to the right by the two right rotators assisted by the two left-hand depressors. The vertical meridians are both rotated to the left.

In moving *down and left*, the eyes are carried down mainly by the two left-hand depressors, to the left by the two left rotators assisted by the two right-hand depressors. The vertical meridians are both rotated to the right.—D.]

642. [Disjunctive Movements of the Two Eyes.—The disjunctive movements of the eyes are—

1. *Convergence*, effected by an equal and simultaneous contraction of both internal recti. It may be combined with a parallel movement, the eyes being converged and also moved right, left, up, or down.—D.]

[Owing to a combination of this sort, it may appear that one eye stands still while the other converges. Thus, if a prism is placed, base out, before the left eye, it will seem as if the latter alone turned inward in obedience to the converging impulse, while the right eye remained fixed. In reality, both eyes converge to an equal amount, say 5°, then both rotate 5° to the right, the net result of the movement being that the right eye, although converging, looks straight forward. The same thing happens when an object which the eyes are following, is brought toward them, not in the median line, but in line with the right eye.—D.]

[The nearest point on which the two eyes can be converged by the utmost effort is called the convergence *near point*, and the furthest point to which they can be directed by the utmost relaxation of the converging muscles is the convergence *far point*.—D.] The latter either lies at infinite distance, in case the visual lines are parallel, when the convergence is completely relaxed, or it lies within infinite distance (i. e., is positive) or beyond (i. e., is negative). The latter expression means that a certain degree of divergence is possible, which indeed is the rule for normal eyes. The difference between the maximum and minimum of convergence (far point and near point of convergence) is the *amplitude of convergence*, which, in cases where the eyes can be made to diverge beyond parallelism, is composed of a positive and of a negative portion. This way of regarding the convergence is like that which since Donders's time has obtained with respect to the accommodation (see § 759), and is intended to facilitate the comparison of these two

functions which are so intimately related. With the same object in view Nagel has introduced the term *metre-angle*. Let *o o* (Fig. 306) be the base line, i. e., the line connecting the centers of rotation of the two eyes, and *M C* the median line. The angle of convergence is the angle through which the eye has to be deflected from the position of parallelism in order to be directed toward a point, *C*. That is, it is the angle *w*, or, what is the same thing, the angle w_1. Its magnitude is in inverse proportion to the distance of the object looked at (precisely as in the case of accommodation). The angle of convergence required in order to sight a point situated one metre in front of the eyes is called a metre-angle (mw [or MA]); and this constitutes the unit for the numerical denotation of the degree of convergence. When the object sighted is at 2 metres distance the convergence amounts to 0.5 MA; when the point is at 50 cm., the convergence is 2 MA, etc. This method of denoting the degree of convergence has the advantage that it parallels in all respects the expressions used to indicate the work done in accommodation. Thus for a distance of 50 cm. a convergence of 2 MA and an accommodation of 2 D are required. The magnitude of the metre-angle, measured in degrees, varies in different persons, since it depends upon the length of the base line; on an average (with a base line of 64 mm.) it amounts to 1° 50′. [The average normal convergence is 16 MA, the divergence 1 MA.] The angle *oCM* or W_1, that each eye makes in converging on its fellow, is the *convergence-adduction*. Its usual maximum varies between 24° and 27°—rarely reaching 30°. It is thus not much over one-half the maximum *latero-adduction* or angle through which the eye can be carried when turning inward when making a parallel (conjugate) movement with its fellow (see § 676 and for the precise measurement of the convergence-adduction see §§ 677 and 679).—D.]

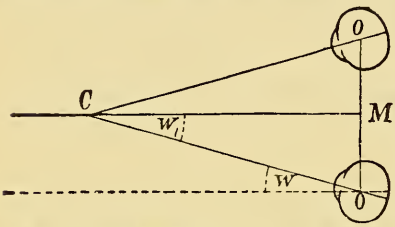


FIG. 306.—REPRESENTATION OF THE METRE-ANGLE.

[2. *Divergence*, effected by a simultaneous and equal relaxation of both internal recti (passive divergence), assisted probably by a simultaneous and equal contraction of both external recti (active divergence).—D.]

[The utmost amount by which each eye can diverge beyond parallelism (*divergence-abduction*) is not over 1.5°–2°. It is thus very much less than the *latero-abduction*, or the angle through which each eye can be turned out when moving in parallelism with its fellow (see § 676 and cf. §§ 677 and 679).—D.]

[3. *Divergence in a vertical plane*, effected by simultaneous contraction of the elevators of one eye and the depressors of the other.—D.]

643. **Nerves Governing the Ocular Muscles and Movements.**—The *innervation* of the ocular muscles is accomplished by three nerves. The oculo-motor nerve supplies the internal, superior, and inferior recti, and also the inferior oblique. Moreover, the levator palpebræ superioris and the interior muscles of the eye, namely, the sphincter pupillæ and the ciliary muscle are innervated by it. The abducens nerve is reserved for the external rectus, the trochlear nerve for the superior oblique. The nuclei for the three nerves supplying the eye muscles lie [beneath the aqueduct and] upon the floor of the fourth ventricle. [See Fig. 307.]

The association of the eye movements is regulated by the *association centers*, which are centers of a higher order than the nerve nuclei. According to the necessities of the case, they innervate certain muscles or groups of muscles of one eye simultaneously with those of the other. The internal rectus of the right eye, for example, may be set into action at the same time with the internal rectus of the left eye, so that a movement of convergence takes place; but, on the other hand, it may also act in concert with the external rectus of the left eye, so that both eyes are turned to the left.

The nuclei from which the nerves of the ocular muscles arise lie one beneath the other, and are connected by transverse fibers which run from the nuclei of one side to

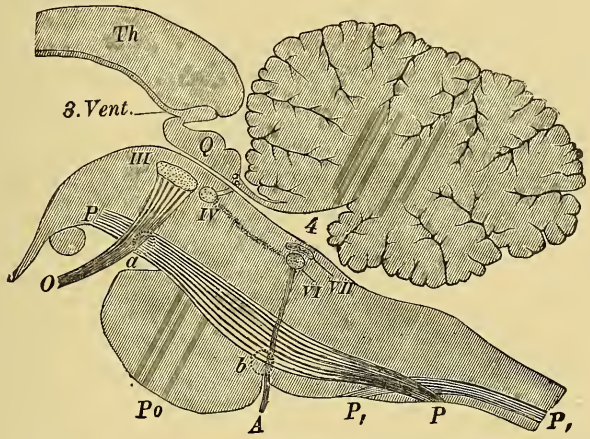


FIG. 307.—NUCLEI OF ORIGIN OF THE NERVES OF THE OCULAR MUSCLES. SCHEMATIC SAGITTAL SECTION THROUGH THE CAUDEX CEREBRI. Natural size.

The oculo-motor nucleus, *III*, lies beneath the anterior pair of tubercles of the corpus quadrigeminum, *Q*. The fibers coming from this nucleus run, converging downward, and emerge as a united nerve trunk, *O*, at the anterior border of the pons, *Po*. Directly behind the oculo-motor nucleus lies the nucleus, *IV*, of the trochlear nerve, from which the trunk of the nerve passes upward. The two points (drawn of light color in the figure) directly above it, and at the posterior margin of the corpus quadrigeminum, represent the cross sections of the trunks of the trochlear nerves as they decussate in the velum medullare anticum. The nucleus of the abducens, *VI*, lies upon the floor of the fourth ventricle, *4*, directly beneath the nucleus of the facial nerve, *VII*. The dotted band running from the nucleus of the abducens to the nucleus of the trochlear nerve represents the posterior longitudinal fasciculus connecting the nerve nuclei. The trunk of the abducens, *A*, emerges at the posterior border of the pons. *a* shows the site of a lesion which, through destruction of the oculo-motor nerve, *O* and the pyramidal tract, *P P*, would result in a crossed paralysis of this nerve and of the extremities. In like fashion a lesion at *b* would produce crossed paralysis of the abducens and of the extremities. P_1 P_1, pyramidal tract of the other side; *Th*, optic thalamus.

those of the other, and by longitudinal fibers (the posterior longitudinal bundle), which joins the proximal to the distal nuclei. From the nuclei fibers ascend to the cortex, going to the centers for the voluntary associated movements of the eyes in the gyrus angularis (Bernhiemer). [It seems likely that the angular gyrus is rather the center for reflex or involuntary associated movements, the centers for willed movements of the eyes, conjugate, convergent, and divergent being situated farther forward in the cortex (according to Sherrington, Bechterew, and others, in the frontal lobe).—D.]

The nerve nuclei themselves lie beneath the aqueduct of Sylvius and on

the floor of the fourth ventricle on both sides of the rhaphe. The most anterior is the nucleus of the *oculo-motor nerve* (*III*, Fig. 307), which lies beneath the aqueduct in the region of the anterior corpora bigemina. It consists of several paired and one unpaired group of ganglion cells. And in a physiological sense it must be regarded as composed of a number of partial nuclei. But which separate group of ganglion cells belong to each muscle innervated by the oculo-motor nerve has not yet been precisely determined for man. For monkeys, in which the relations are probably similar, Bernheimer, on the basis of experimental investigations, has worked out the plan shown in Fig. 308. This confirms what clinical experience has already shown, that the nuclei of origin of muscles that are synergetic in action are in juxtaposition—e. g., the nuclei for the pupil, for accommodation, and for convergence (internal recti); the nuclei of the superior rectus and inferior oblique (subserving elevation of the eye); and the nuclei (subserving depression of the eye) of the inferior rectus and the trochlearis, which latter does not belong to the domain of the oculo-motor nerve.

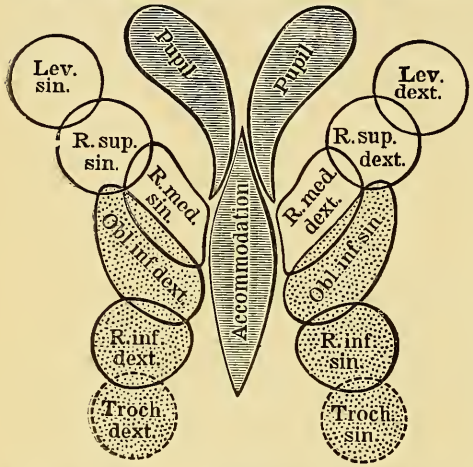


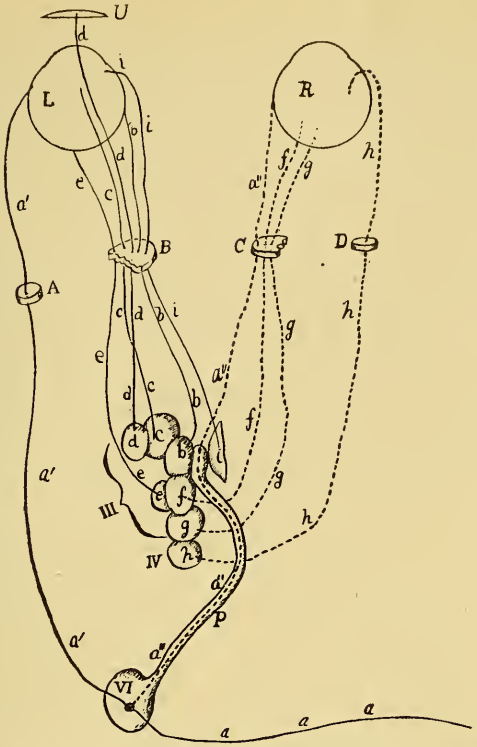
FIG. 308.—SCHEMATIC REPRESENTATION OF THE NUCLEAR REGION OF THE OCULO-MOTOR AND THE TROCHLEAR NERVES IN A MONKEY. Seen from above. (After Bernheimer.)

The median nuclei are shaded; they are designed for the interior muscles of the eye, the two anterior (paired and composed of small cells) being designed for the pupil, the posterior (unpaired, large-celled median nucleus) for accommodation. The groups of ganglion cells situated on either side of the median nuclei form together the right and left main lateral nucleus. They comprise the region of origin of the nerves for the exterior ocular muscles, and immediately adjoining them behind is the nucleus of the trochlear nerve. [All [4] the fibers from the partial nuclei of the interior muscles, the levator and the superior rectus, most of those from the partial nucleus of the internal rectus, and a few of those from the partial nucleus for the inferior oblique, run to the eye of the same side; the remaining fibers from these latter two nuclei and all the fibers from the nuclei of the inferior rectus and the superior oblique run to the eye of the other side. See also Fig. 318.—D.] The actual disposition of the nucleus is such that the partial nucleus for the levator represents the proximal, that for the inferior rectus the distal end of the main lateral nucleus, and the median nuclei lie between the proximal ends of the lateral nuclei. In reality no sharp differentiation of the partial nuclei within the main lateral nucleus exists, and hence the situation of the individual partial nuclei cannot be determined precisely but approximately only.

The fibers coming from the oculo-motor nucleus pass down through the crus cerebri, part of the fibers remaining on the same side, another part crossing to the other side. At the base of the brain the oculo-motor fibers become visible on the anterior border of the pons (*O*, Fig. 307). From this point the oculo-motor nerve runs in the wall of the cavernous sinus (Fig. 310) and through the superior orbital fissure into the orbit.

The nucleus of the *trochlear nerve* (*IV*, Fig. 307) follows almost directly upon the posterior extremity of the oculo-motor nucleus, so that it might almost be regarded as the last partial nucleus of the latter [cf. Fig. 309]. It lies beneath the posterior tubercles of the corpus quadrigeminum. But the fibers which arise from it do not join with the trunk of the oculo-motor nerve which runs downward, but pass in the opposite direction upward and backward, into the velum medullare anticum. In this they pass over to the other side and thus decussate with the fibers of the opposite nerve, and then come out upon the base of the brain, winding about the crus cerebri and passing outward.

[4] [This description has been modified somewhat from the original to make it conform to Bernheimer's statements.—D.]



[Fig. 309.—Distribution of the Nerves running to the Motor Apparatus of the Eye according to Bernheimer. Left Nucleus.

A, section of left 6th nerve; *B*, section of left 3d nerve, comprising only that portion which contains the direct fibers (from the left nucleus); *C*, section of right 3d nerve, comprising only that portion which contains the crossed fibers (from the left nucleus); *D*, section of right 4th nerve. *R*, right eye. *L*, left eye. *U*, right upper lid. *III*, left oculo-motor nucleus. *IV*, left trochlear nucleus. *VI*, left abducens nucleus, connected with *III* by posterior longitudinal bundle, *P*. *a*, *a*, *a*, association fibers, from right hemisphere (angular gyrus) to *VI*, bifurcating there to form *a'*, *a'*, *a'*, fibers to the left external rectus and *a''*, *a''*, *a''*, fibers to right internal rectus. Latter fibers pass through *P* to *III* and thence through *C* to the muscle. These fibers subserve parallel movements only, i. e., rotation to the left in conjunction with the left external rectus. *b*, *b*, *b*, fibers to left internal rectus (subserving convergence only, not parallel movements). *c*, *c*, *c*, fibers to left superior rectus. *d*, *d*, *d*, fibers to left levator palpebræ. *e*, *e*, *e*, fibers, (few in number) to the left inferior oblique. *f*, *f*, *f*, fibers (many in number) to right inferior oblique. *g*, *g*, *g*, fibers to right inferior rectus. *h*, *h*, *h*, fibers to right superior oblique. *i*, *i*, *i*, fibers to left iris and ciliary muscle.—D.]

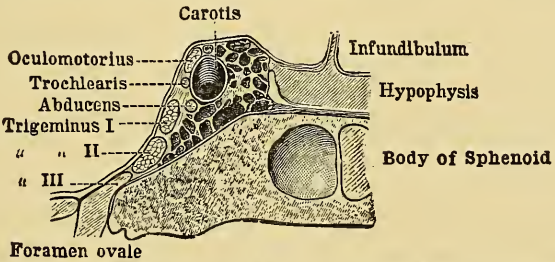


Fig. 310.—Frontal Section through the Sinus Cavernosus. (After Merkel).

The sinus lies upon the lateral division of the body of the sphenoid bone. It is traversed by numerous septa which are attached also to the inner and lower wall of the carotid artery, which forming an S-shaped bend lies in the sinus. The outer and upper wall of the carotid artery is adherent to the wall of the sinus. Imbedded in this wall lie the three nerves supplying the eye muscles, and further below lie the first and second branches of the trigeminus. The third branch of the trigeminus leaves the cranial cavity through the foramen ovale.

The nucleus of the *abducens* (*VI*, Fig. 307) lies pretty far behind the nuclei of the other two nerves, and in the immediate vicinity of the facial nucleus (*VII*, Fig. 307), a little in front of the striæ medullares. The nerve fibers arising from the nucleus pass downward between the bundles of the pyramidal tracts and become visible at the posterior border of the pons (*A*, Fig. 307). Both the trochlear and the abducens nerves, as soon as they have reached the base of the brain, run forward like the oculomotor nerve and pass through the cavernous sinus (Fig. 310) and through the superior orbital fissure into the orbit.

644. Projection and Orientation.—Orientation in space—i. e., the ascription of objects seen to the place where they actually belong—is effected in the following way: The objects of the external world form images upon the retina. To find the situation of the retinal image of any object whatever, we only need draw a line from the object to the retina through the nodal point of the eye (*k*, Fig. 311), since those rays that pass through the nodal point (principal rays) pass unrefracted to the retina. Thus the image of the object of fixation, *o* (Fig. 311), lies at *fc* (the fovea

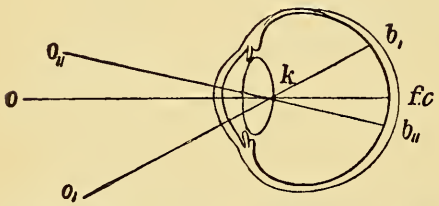


Fig. 311.—Projection of Retinal Images Externally.

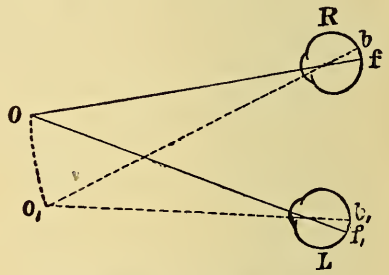


Fig. 312.—Binocular Single Vision.

centralis). Objects, such as o_1, situated beneath the point of fixation, throw their image above the fovea centralis, at b_1; and, on the other hand, the object o_{11} lying above the point of fixation has its image at b_{11} beneath the fovea. We ourselves judge of the place in which an object is by following out a reverse process. We refer the object to the extremity of a line which passes outward from the retinal image and through the nodal point. This process of determining the place of external objects, which is learned by experience, is called *projection* (of the retinal images outward). By virtue of it we see the objects in the outer world arranged side by side just as their images are upon our retina, only in reverse order; whatever forms an image to the right of the point of fixation, is seen upon the left of the latter, etc. We are thus informed with certainty in regard to the position of objects relative to each other (*objective orientation*). But for perfect orientation it is further requisite that we should assign to its correct situation in space the whole mosaic of images that we project from our retina into the outside world, and which is already properly constructed as far as the relation of its own parts to each other is concerned. Not till we do

this can we have any conception, corresponding to the real state of things; a conception, that is, of the position of objects not only with reference to each other but also with reference to our own body (*subjective orientation*). Such subjective orientation depends upon our having a knowledge of the position of our own body in space, and of the position which the eyes occupy in our body. The former is accomplished by virtue of the sense of equilibrium, the latter by the muscular sensations which originate in the ocular muscles, and which inform us how our eyes are directed with relation to our body. By means of subjective and objective orientation together, we are able to recognize correctly the absolute position in space of any object that we see.

As a rule, we see with both eyes at once, these being so placed by means of their associated movements that their visual lines cross in the object looked at—i. e., we "fix" [or "sight"] the object with both. The object *o* (Fig. 312) then forms an image in the fovea centralis (*f* and f_1) in both eyes. An object, o_1, situated to the left of the point of fixation, would cast an image at *b* and b_1, to the right of the fovea in both eyes, and in both eyes, moreover, equally far to the right of it. These images, as well as all others that are situated on symmetrically disposed spots of the two retinæ, are, according to the law of projection, located by both eyes, at the same point of the outer world (*o* o_1, etc.), and hence are seen single (*binocular single vision*).

645. Binocular Vision.—The fact of a person's seeing single with two eyes [5] may be accomplished in two ways. Either he fixes correctly with both eyes and refers to the same spot the impressions produced in both (binocular single vision) or one of the two eyes fails to see, either because it is blind or because it suppresses the impression that it receives (monocular vision).

How can we know which of these two conditions is present in any given case? One way is by determining whether the object is fixated properly with both eyes, for it is only under these conditions that binocular single vision is possible. If a manifest deviation of one eye is present, no further test is necessary. The case is different if there is no deviation that we can see or at least none that we can be certain of. Then we make the following test: We make the patient fixate an object, e. g., a lighted candle, some metres distant, and we cover first one eye, then the other. If the two eyes are rightly placed, each will remain fixed in its position after the other is covered. But let us assume that the right eye deviates a little outward when the left is fixed upon the object. If, now, the former is covered the left will continue in the act of fixation; but if the left is covered, the right has to be brought into the position of fixation by a movement of adduction. Hence, in covering the eye which is performing fixation, we notice a movement of adjustment in the nonfixing eye, the direction of which movement is precisely opposite to that of the previous deflection. This movement of adjustment is still distinctly visible when the deflection itself is too slight to be recognized with certainty.

[5] [The patient often gives wrong information on this point, alleging that he sees single either because through his own inattention or faultiness in our methods of examination the fact of his seeing double is not brought home to him, or because, as happens in periodic and intermittent squint, he sees single at one moment and double at another.—D.]

Now in all cases in which correct fixation by the two eyes is not present—and that, whether the deviation is a manifest one or is demonstrated only by the test above given—, binocular single vision cannot exist. If, nevertheless, there is single vision, this can be accounted for only upon the assumption that the image formed in the deviating eye is either not perceived or is suppressed. If, on the other hand, correct fixation is proved to exist in both eyes, we may assume that the single vision is a binocular one.[6] In order to be quite sure, we may apply another test, so as to determine whether the single vision depends upon the fusion of the two images or upon the suppression of one. We hold a prism with its base down before one of the eyes (Fig. 343). If there was a binocular vision before, two images would now necessarily be seen standing one above the other (o and o_1). But if there is still single vision, this can only occur because the image formed in one of the eyes is either not perceived or is neglected. [Another test is bar-reading and the use of the red glass (see § 675).]

It is only a person who has binocular single vision that has also real solid or *stereoscopic* vision. Hence, we may also test binocular vision with stereoscopic pictures, special samples of which have been made for this purpose [or with the amblyoscope (see § 675)]. A particularly delicate test of stereoscopic vision—i. e., of the perception of degrees of depth—is by means of *Hering's test with falling bodies.* The person under examination looks with both eyes through a long tube at a slender thread stretched vertically. Little balls (glass beads or peas) are dropped alongside of the thread, sometimes in front of it, sometimes behind. A man having proper binocular vision will tell every time, and without delay and without mistake, whether the balls have fallen in front or behind the thread; but one having only monocular vision can at best guess at what takes place, and hence often makes a mistake.

646. Binocular Diplopia.—Interference with binocular single vision is manifested by binocular diplopia, which makes its appearance when the visual axis of one eye deviates from the object of fixation. For example,

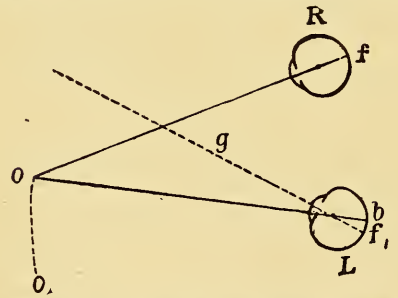


Fig. 313.—Homonymous Double Images.

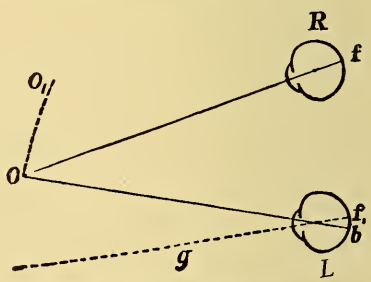


Fig. 314.—Crossed Double Images.

the right eye, R (Fig. 313), sights the point, o, while the visual axis, g, of the left eye, L, deviates inward, because the eye has a convergent squint. The point, o, then forms an image at the fovea, f, in the right eye, but in the left it forms an image at b to the right of the fovea, f_1. With the right eye the object is seen in its right place, o. With the left eye, too, the object would be seen at a point opposite the retinal image, b, that is, in its proper situation, o, and hence would be seen single with binocular

[6] [This is by no means a safe assumption. The test above made is simply one of binocular fixation (see § 674, description of the screen test), and not at all one of binocular vision. It is quite possible, especially in a case of cured squint, for a person to have binocular fixation and yet also have complete suppression of one image and hence only monocular vision.—D.]

vision, if the person possessing such an eye were not in error with regard to the way in which the left eye is directed. He knows nothing of the deviation of this eye inward, but has the idea that, like the right, it has its visual axis adjusted for the object. He therefore expects the image of the object to be at the fovea in the left eye as well as in the right. But as this is not the case, and the image, *b*, lies to the right of the fovea, he thence concludes that the object, *o*, has become displaced toward the left—that is, to o_1—since he knows from former experience that all objects situated to the left of the point of fixation throw their images to the right of the fovea. In this case, accordingly, the subjective orientation is not correct; the entire mosaic of retinal images in the left eye is located in space too far to the left, because the person who has such an eye has an erroneous impression in regard to the way in which it is placed in his head (Nagel, Alfred Graefe).

The double images that have been here selected as an example are known as *homonymous*, because the image, *o*, seen upon the right side belongs to the right eye and the one, o_1, seen on the left belongs to the left eye. In practice this fact is determined by covering, for a moment, first one eye and then the other, and asking the patient which of the two images in each case disappears. We can also place a colored glass before one eye and have the patient tell which of the two images is colored, and which appears of its natural hue. Homonymous double images depend, as the preceding demonstration shows, upon undue convergence of the eyes.

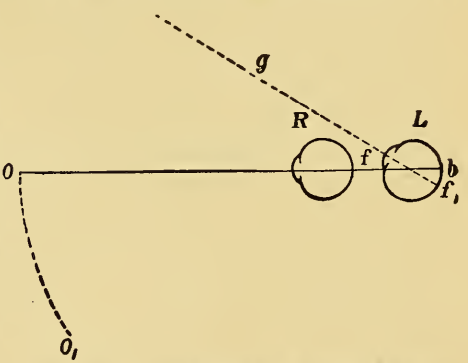


FIG. 315.—DOUBLE IMAGES, WITH DIFFERENCE IN LEVEL.

Heteronymous or *crossed* double images are produced when there is a relative divergence of the eyes. In Fig. 314 the left eye, *L*, deviates outward. The image of the point, *o*, therefore falls to the left of the fovea; f_1, at *b*, for which reason the object itself is erroneously seen at o_1, to the right of the point of fixation, *o*. In this case the left image corresponds to the right eye, while the right image corresponds to the left eye.

A *difference in the level* of the double images occurs when the eyes themselves stand on different levels. In Fig. 315 the eyes are represented as behind each other instead of side by side. The right eye, *R*, sights the object correctly, but the left eye, *L*, is deviated upward. Hence, the image, *b*, of the point, *o*, falls above the fovea, f_1, in the left eye, and the person who has such an eye imagines, because he believes that the eye is correctly

placed, that he sees the point, o, at o_1, below its true situation, since when the eyes are correctly placed all objects situated below the visual plane cast their images upon the upper half of the retina. The image which stands lower, therefore, always belongs to the eye which stands higher, and vice versa. [We arbitrarily denote the vertical diplopia as *right* or *left* according as the image formed by the right or the left eye is lower, indicating that the eye itself is higher. (Right and left diplopia, therefore, correspond to right and left hyperphoria (see § 669).—D.]

Double images may also be *inclined* in such a way that their upper or lower extremities are approximated. This is the case when one of the two eyes has undergone a rotation about its sagittal axis and the other has not. In Fig. 316 A, R and L represent the posterior halves of the two eyes seen

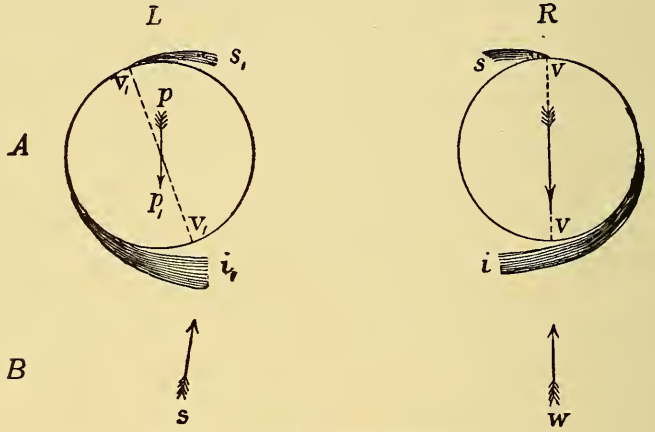


FIG. 316.—DOUBLE IMAGES WITH OBLIQUITY.

from behind and imagined to be transparent, so that the inverted image of an arrow is seen as it is situated upon the retina. In the right eye the vertical meridian of the retina, $v\ v$, really does stand vertical, but in the left eye ($v_1\ v_1$) it is supposed to be inclined. The image of a vertically directed arrow is also vertical in the two retinæ; hence, in the right eye it coincides with the vertical meridian, but in the left eye it forms an angle with the vertical meridian, because this is inclined. Since, now, the left eye has been previously accustomed to consider as vertical only those objects the images of which coincide with the vertical meridian, it will consider the arrow as having an oblique position.[7] Hence, two images of the arrow are seen (Fig. 316 B, w and s), of which that belonging to the left eye stands obliquely. [Necessarily, according to the laws of projection, the image, s, seen by the left eye, appears tilted the opposite way to that in which the

[7] [This applies only to a pathological tilting in which the vertical meridian of one eye is no longer parallel with that of the other. The physiological tilting which occurs in the oblique positions of the gaze (see remarks under Actions of the Ocular Muscle, pages 719–721), and in which the vertical meridians of the two eyes, although tilted, remain parallel to each other, is not associated with apparent tilting of the object looked at.—D.]

eye itself is tilted. For the lower end, p^1, of the retinal image is formed on the outer as well as the lower part of the retina. Consequently it is projected not only up, but also in (to the right), so that the point of the arrow appears located in space above and to the right of the arrow's center. So too, the upper end, p, of the retinal image, which falls on the inner part of the retina, is projected out (to the left) as well as down, and appears in space, therefore, to the left of the arrow's center. Hence, while the eye itself is tilted to the left, the image formed by it is tilted to the right.—D.]

[Thus it will be seen that in all varieties of diplopia *the image formed by the deviating eye is always deflected in a direction opposite to that in which the eye is deviated* (to the right if the eye is deflected to the left, up if the eye is deflected down, tilted to the right if the eye is tilted to the left, etc.). This is a necessary consequence of the law of projection.—D.]

[Binocular single vision is converted into *binocular double vision* when one of the two eyes leaves the correct position of fixation. This takes place most frequently in consequence of disturbances in the muscular apparatus of the eye. But the eye can also be forced mechanically into an incorrect position—e. g., by tumors in the orbit, etc. Binocular double vision can readily be produced experimentally by pushing one eye a little to one side by pressure with the finger. Finally, double vision ensues whenever the excursions of one eye are limited, as compared with those of the other by mechanical obstacles, as, for example, by symblepharon or by pterygium.

In diplopia the *distance between the double images* measured in degrees of arc corresponds precisely to the deflection of the deviating eye from the correct position, and can therefore serve as a measure for the degree of deviation. The linear distance between the double images, on the contrary, depends not only upon the degree of deviation, but also upon the distance to which the double images are projected. The greater this distance is, the farther apart the double images appear to be. When the double images stand very close to each other they overlap in part, so that only their outlines appear double. In this case the patient is often not aware that he sees double, but only complains of seeing indistinctly or complains that every object has a shadow.

647. Theory of Space Perception.—According to the doctrine of projection the visual sensations produced by looking at objects are projected out from the eye or the head by a psychic act which represents the result of the experience of the individual in question. By virtue of this projection the path which rays of light have traversed in going to the eye is, so to speak, re-traversed, and this causes the correct localization of the object producing the visual sensation.

In opposition to this idea, Hering has enunciated the proposition that visual sensations possess a priori a "spatial quality." A definite spatial concept is always associated with the stimulation of a definite retinal element; and, because of the spatial qualities belonging to them, the visual sensations form a subjective space of their own into which we look—the so-called "visual space." Any two suitably related points on the two retinæ have the property that an object which forms its image on each is associated with the same spatial concept and hence is seen by the observer in the same direction with each eye ("identical visual projection from corresponding retinal points"), so that the visual spaces of the two eyes are identical. We can conceive of this as taking place by the sliding of the two retinæ over each other in such a way that the corresponding points of the two coincide. If from such an imagined "cyclopean" eye (which would lie about at the root of the nose) we draw lines of direction out into space they would

represent approximately the lines of visual projection of the double eye. The projection line corresponding to the two foveæ centrales—called the principal projection line —is in general pointed straight ahead in the median plane, provided the visual lines are either parallel and directed straight forward or are symmetrically converged.

To other portions of the retina appertain distinct lines of projection differing from the chief line. If such portions in the two retinæ are correspondent, stimulation of them sets going a visual sensation which is localized on the plane of the point of fixation (called the reference plane). In this way the retinal images are localized so far as their height and width are concerned. Their localization so far as regards depth (fore and aft from the observer) is effected by the *physiological double images.* These develop when an object is so situated in space as to stimulate non-correspondent portions of the retinæ. The object then appears double (Fig. 317). If the image seen on the right side belongs to the right eye, we speak of the double images as being homonymous; if it belongs to the opposite eye, we speak of them as crossed. Homonymous double images are projected beyond the reference plane (Fig. 317), and crossed double images to the hither side of it; the apparent distance from the reference plane of the object thus seen double increases with the apparent distance of the double images from each other, i. e., with their *disparation.* [Usually we are not aware of the existence of physiological double images, yet from the subconscious impression they give we get our main *notions of depth and distance.* We regard A as further off than B, because when we look at A, B appears double (although we are not consciously aware of it), and vice versa; the double images in the former case being crossed, and in the latter, homonymous. The distance of a comparatively near object may also be estimated by the amount of muscular effort that we feel we are exerting in converging and accommodating on it. Finally, when moving (as in a railway train), we gauge the distance of objects which we are passing by the parallactic displacement of the nearer on the more remote. An animal with lateral vision (page 611) is particularly fitted for judging distances in this third way, having an advantage here over man with his frontal vision (Trowbridge).—D.]

The spatial relations of the objects contained in visual space, i. e., the apparent amount of their separation from each other in terms of breadth, height, and depth, we call *relative localization* (in analogy with objective orientation), in distinction from *absolute localization* (subjective orientation), by which we understand the position with regard to the observer and the distance from him of the whole complex of things seen at any one time. Relative localization depends entirely upon the position of the images on the retina, and in its turn determines the movements of the gaze that are guided by attention. When an object that is seen by indirect vision attracts the attention, the appropriate movement of the gaze is started up by the effort that the person makes to see the object more clearly. Before the movement begins, its purpose which is to bring the image of the object on the fovea centralis, is already defined. The situation of the fovea with relation to the site on the retina of the indirectly seen image determines the direction and amount of the movement.

Toward the will, acting under the guidance of spatial perceptions, the two eyes behave *like a single organ.* The object that excites a movement of the gaze, appears either to the right or left, or above or below, or closer or more remote than the object fixed; and there is set up accordingly either a movement to right or left, or a movement up or down, or an increase or decrease of the convergence. Now it is a fundamental proposition that the impulse acting to produce these movements always flows in equal intensity to the two eyes, so that in this regard the eyes may be said to behave like a single, or *cyclopean* eye.

This *association of movement* in which the sensory correspondence of the retinæ is reflected, is compulsory—a fact which asserts itself, for instance, in this that an eye which is blind or is covered shares in the movements of the eye which sees, and so much

so that the will is unable to suppress this associated movement, objectless as it is for vision.

One consequence of this law of movement is the *pathological diplopia* that occurs in paralysis of the individual eye muscles. If, for example, the external rectus of the right eye is paretic, then when the gaze is directed to the right the same impulse is sent, in spite of the paralysis, to the sound internal rectus of the left eye and the paralyzed external rectus of the right. Consequently, the paralyzed right eye will lag behind in comparison with the left, so that the images of a point will no longer fall upon corresponding spots of the retinæ. The image of the object *O* (Fig. 317), which the left eye is fixing, instead of falling on the fovea in the paralyzed eye, falls on an excentrically situated point of the retina, which is correspondent with a point having a similarly excentric situation in the left retina. Wherever this point in the left retina localizes the light stimulus that falls upon it, there, according to the law of correspondence of the retinæ, will the right eye see the object of fixation—i. e., it will not see it in the same place as the left eye does. Hence the object looks double. The pathological

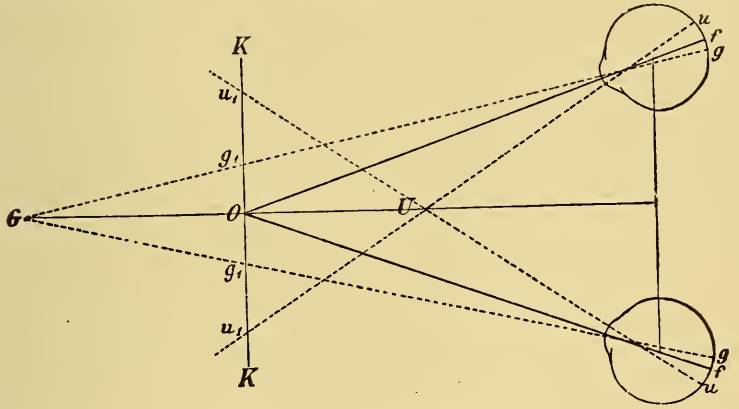


Fig. 317.—Physiological Double Vision.

The point of fixation, *O*, forms its image on the foveæ centrales, *f*, in both eyes. A more remote point, *G*, forms its image at *g*, to the nasal side of the fovea in both eyes. Hence, when projected on the reference plane, *K K*, at the distance of the point *O*, it appears double, the images being at g_1, g_1. Since the right image is seen with the right eye and vice versa, the double images are homonymous. A point, *U*, nearer than *O*, forms its image in *u*, *u*, to the temporal side of the foveæ in both eyes and therefore appears in heteronymous double images u_1, u_1. Because of these double images, therefore, if the gaze is directed at *O*, *G* looks further and *U* nearer than *O*, and that, even if the gaze does not move from *O*.

diplopia produced under these conditions is distinguished from the physiological diplopia above described by the fact that it does not, like that latter, extend simply to the objects of indirect vision, but also involves the object of fixation, whatever it may be.

648. False and True Image.—When there is binocular double vision, the two images do not look alike; one is more distinct than the other, and is hence known as the *true image*, in contradistinction to the *apparent image*. The true image is the one that corresponds to the eye that sights the object. It is therefore seen in its right place, and, moreover, is seen clearly, because it is perceived by the fovea. The apparent image belongs to the deviating eye. It is less distinct than the image of the other eye, because it is perceived by a peripheral spot of the retina; moreover, it is seen in the wrong place, so that the patient, if he tries to reach out to it, reaches to one side of it—hence apparent or false image.

649. Fusion.—Double images interfere with vision and cause confusion, so that every one tries to avoid them as far as he can. He does this by attempting to bring the eyes by suitable muscular effort into a proper position, so that the double images coalesce. This attempt at union or fusion of the double images is called the *fusion tendency.*

Frequently by virtue of it considerable obstacles opposing single vision are overcome. The following experiment shows this: We make the patient gaze at an object, *o*, and then hold before one of the eyes, for instance the right one, a prism, *P*, whose base is directed toward the temporal side (Fig. 318). The rays coming from *o* are deflected toward the base of the prism, and would strike the retina of the eye at a point to the outer side of the fovea; but in that case, as the object forms an image at the fovea, f_1, in the left eye, crossed double images would be produced. To prevent this, the right eye is turned inward until the fovea, *f*, has moved outward sufficiently far to be impinged upon by the rays which have been deflected by the prism. Hence, to avoid seeing double, an increased effort at convergence is made, so that the usual lines cross at *h* instead of at *o*. By this convergence the prism is "overcome." By placing constantly stronger and stronger prisms before the eye, we find the strongest prism which can be still overcome by *convergence*, and which therefore is a measure of the latter. [For the mechanism by which convergence is accomplished see page 721.—D.]

If the prism is placed before the eye with the base inward, the rays passing through it are deviated in, and the image of the object is thrown to the inner side of the fovea. The eye must then be turned outward in order to bring the fovea to the place of the image. In this case, accordingly, to produce single vision the prism is overcome by a divergence of the eyes. For we are able not only to make our visual lines parallel but under certain conditions even to make them a little divergent. The strongest prism which can be overcome in this way gives the measure of the *divergence*, or, as it is also called, negative convergence [see pages 721 and 722].

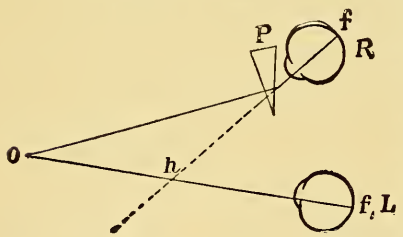


FIG. 318.—THE OVERCOMING OF A PRISM BY CONVERGENCE.

The tendency to fusion also makes itself apparent if a prism is placed before the eyes with its base up or down (Fig. 343). In this case there are formed double images with a difference of level, which have to be united by a deviation of one eye upward or downward [or rather of both eyes, one going up the other down—divergence in a vertical plane (see page 722).—D.]

Very strong prisms can be overcome by convergence, weaker ones [of 5 △ to 8 △] by divergence; and only very weak prisms (of 1 △ or 2 △) can be overcome by vertical deviation of the eyes. [For prisms and their numbering, see § 747.—D.]

The ability to perform convergence, as determined with prisms, is also called adduction, and the ability to perform divergence is called abduction. These terms had better be avoided, as they are already applied to denote the lateral excursions (side to side) of the eyes (see page 719), which follow quite different laws. This is immediately apparent when we remember that when side to side movements are to be made, the eyes can be abducted until the external margin of the cornea almost touches the external angle of the eye, while the outward movement of the eye in performing divergence is but a minimum one. [See also §§ 676, 677, and 679.]

650. Monocular Diplopia.—A condition to be rigorously differentiated from binocular diplopia is monocular diplopia. The former depends upon the fact that though there is but one image of the object cast upon each one of the two retinæ, it is not thrown upon spots that are similarly situated; but the latter is due to the formation of two images of the same object upon *one* retina. Binocular diplopia, therefore, disappears at once when one eye is shut, while monocular diplopia persists, although but one eye—that is, the one which sees double—is open. In this fact lies the most certain differential sign between the two kinds of diplopia. The cause of monocular diplopia is either an anomalous refraction of the rays of light or the presence of a double pupillary opening. The former represents one form of irregular astigmatism (see § 790), and has its seat either in the cornea or in the lens. It occurs particularly in subluxation of the lens [being then due not to astigmatism but to a sort of prismatic action]—(see page 556). In incipient cataract also monocular diplopia may develop as a result of the unequal refracting power of the different sectors of the lens, although in this case monocular polyopia (see page 527) is of much more frequent occurrence. A double pupillary opening produces diplopia when the eye is not adjusted for the distance at which the object of fixation is placed. It is most frequently found as a consequence of iridodialysis.

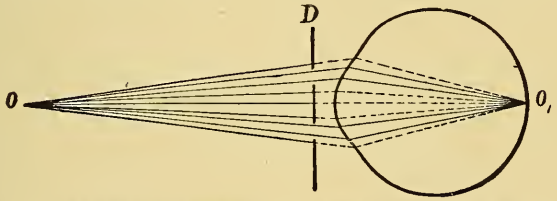


Fig. 319.—Scheiner's Experiment. (The eye is adjusted for the point *o*.)

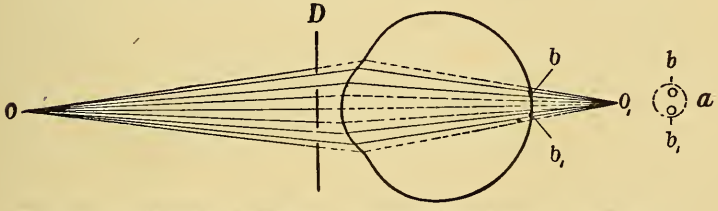


Fig. 320.—Scheiner's Experiment. (The eye is not adjusted for the point *o*.)

Monocular diplopia with double pupillary aperture (iridodialysis, perforation of the iris, division of the pupil into two parts by an opaque strand, etc.) takes place only when the eye is not properly focused; otherwise, there is single vision, even with a double pupil. This fact is accounted for by the well-known experiment of Scheiner. Two holes are made in a piece of cardboard (*D*, Fig. 319) with a needle, the distance between them being somewhat less than the diameter of the pupil, so that, when looked through, both lie at the same time in front of the pupil. Through this apparatus we look at an object—e. g., at a tightly stretched thread, *o*, at a distance of 25 cm. If the eye is focused for this distance, all the rays emanating from the object, *o*, are united

upon the retina at the point, o_1. If, now, out of the whole cone of rays only those are transmitted which pass through the two holes, these rays still unite to form an image at o_1; the only change which this image undergoes by having the diaphragm placed before it is an enfeeblement of its luminosity due to the cutting off of many rays. But if the eye is not focused for the distance of the object (Fig. 320), the apex of the cone of rays does not fall upon the retina, but (in this case) behind it at o_1. The cone of rays is cut off in front of its apex by the retina, so that the image of the point, o, is a disk (diffusion circle), a, and the point looks completely blurred. But if now only two bundles of rays out of the entire cone are admitted to the eye through the diaphragm, each one casts its own smaller diffusion circle (b and b_1); the point, o, is now seen more distinctly, it is true, but is seen double.

In myopia, especially of high degree, the complaint is sometimes made of monocular diplopia. This makes itself particularly apparent when rectilinear outlines, such as telegraph wires, the outlines of picture frames, etc., become objects of fixation, they then appearing double. Here we are probably dealing with the effect of an irregular astigmatism.

I. Paralysis of the Ocular Muscles

651. Symptoms.—1. *Limitation of Movement.*—In paralysis of an ocular muscle the excursion of the eye toward the side corresponding to the action of the muscle that is paralyzed is diminished or entirely abrogated. If, for example, the external rectus of the right eye were completely paralyzed, the right eye could be brought only to the middle line and not beyond it to the right. When the paralysis is incomplete the deficiency in motility is, of course, less considerable, and often can be made out only by comparison with the other, or sound eye. In very slight paralyses the defective motility is not marked enough to be recognized at all with certainty. In these cases we must rely for our diagnosis upon the double images produced [or the characteristic changes in screen deviation (page 745)].

The result of the limitation of mobility is a lagging behind of the eye when an associated movement is initiated within the sphere of action of the paralyzed muscle. Thus, if in paralysis of the right externus a point, o (Fig. 321), situated upon the right, should be the object of fixation, the left eye will be adjusted for it properly; but the right eye will not be turned sufficiently far to the right, and consequently its visual axis, g, will shoot off to the left of the object. The eye "squints" inward (*strabismus paralyticus*, or *luscitas* [7]). This squinting takes place only when the eye is turned in the direction of the sphere of action of the paralyzed muscle, and becomes more pronounced the farther the eye is moved toward this side; but in all directions of the gaze in which the paralyzed muscle does not have to participate, the eyes stand in their proper position. By this fact paralytic squint is distinguished from ordinary or concomitant squint, which is present in all directions in which the eye is turned, and always to the same amount.

The measure of the deflection is determined by the angle s (Fig. 321),

[7] *Strabismus*, from στρέφειν, to turn. The term *luscitas* comes from *luscus*, squinting, and is at present used exclusively for paralytic strabismus. From luscus is derived the French *louche*.

that the visual axis, g, makes with the line of direction, r, which passes from the object to the retina through the nodal point of the eye, and which gives the place of the retinal image, b. This deflection of the squinting eye is known as the *primary deviation.*

While the patient keeps on looking at the object, o, a screen, S, is placed before the left eye (Fig. 322). Now the right eye takes up the task of fixation, it being presupposed that it can be really brought far enough to the right for this purpose. If now we look at the left eye behind the screen we shall find it turned strongly inward—much more so, in fact, than the right eye had been previously. The deflection of the sound eye when covered, which is measured by the angle s_1, (Fig. 322), is called the

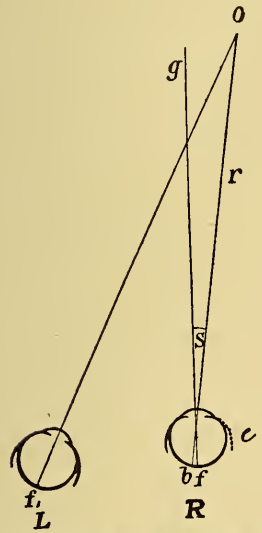


Fig. 321.—Primary Strabismic Deviation in Paralysis of the Right External Rectus.

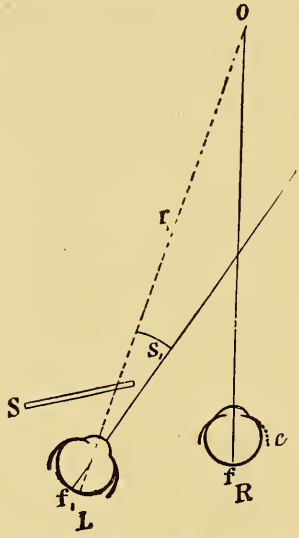


Fig. 322.—Secondary Strabismic Deviation in Paralysis of the Right External Rectus.

secondary deviation, which therefore exceeds the primary deviation in magnitude. It is accounted for as follows: When with both eyes uncovered the gaze was directed toward the right, the left internus and the right externus received the ordinary impulse for a movement to the right; but the right eye lagged behind the other in proportion as the right externus, owing to its impaired innervation, failed to answer to the impulse. If now the left eye is covered, the patient is compelled to make fixation with the right. He tries now to turn this eye to the right, by sending into the right externus a very strong impulse of innervation, although in so doing he still obtains only a very slight effect. He cannot, however, innervate thus strongly the right externus alone, but can simply send out to both eyes a very energetic impulse for a lateral movement to the right. This impulse, therefore, also affects the left internus. But in the latter the

impulse has its full effect, so that the left eye is drawn very strongly to the right (inward). Thus, while in primary deviation it is a mere question of lagging behind of the eye, secondary deviation is produced by a powerful muscular traction; therefore, the secondary deviation is greater than the primary. This point, too, is of importance in distinguishing between paralytic and concomitant squint, since in the latter the primary and secondary deviation are usually equal.

[The secondary deviation of the sound eye shows itself not only when this is screened but also when for any other reason the paretic eye takes up fixation. This happens, for example, when the patient looks so far to one side that the view of the object is cut off from the sound eye by the nose. The sound eye in that case shows a sudden marked deviation (Fig. 325). Again it often happens, especially in congenital paralysis that the paretic eye is used habitually for fixation, so that the sound eye deviates in a spasmodic way, even when both eyes can see the object. Or, fixation may alternate between the two eyes, in which case the difference between the small primary and large secondary deviation becomes at once apparent (see page 745). The amount of the primary and secondary deviation can be measured with prisms (page 746) or by the double images on the tangent plane (page 748). It must be admitted that in many cases there is much less difference between primary and secondary deviation than one would expect.—D.]

652. 2. *False Orientation.*—With the paralyzed eye the patient does not see objects in their true place. For suppose that, when the right external rectus is paralyzed, he shuts the left eye and looks with the right alone at an object situated a little to the right—i. e., within the sphere of action of the paralyzed muscle—and then is told to shut quickly his right eye, too, and thrust at the object with his index finger; as he does so, the finger will always be carried to the right of the object, whence it follows that the latter is seen too far to the right (*Von Graefe's reaching test*) [*projection test*]. [The test can also be made with the right eye open.—D.] The same phenomenon comes to light when the patient tries to walk straight toward a given point with the help of his paralyzed eye, the other being closed. He takes a wavering and zigzag course, first bending his steps too far to the right, then recognizing his mistake and correcting it; then deviating anew to the right, and so on.

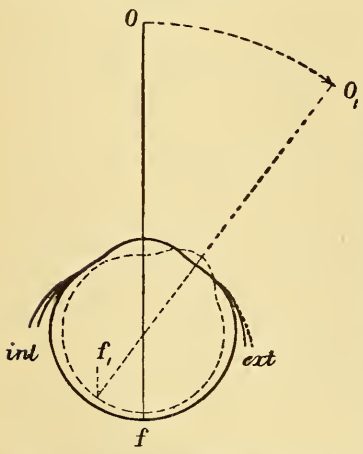


Fig. 323.—False Orientation in Paralysis of the Right Externus.

The explanation of this occurrence is similar to that which has been given for binocular diplopia (page 729). The object is falsely localized because the patient is in error in regard to the position which his eye occupies When (Fig. 323) the patient with his paralyzed right eye sights the

object, *o*, which is placed somewhat to the right of him, i. e., looks at it in such a way that it forms an image upon the fovea centralis, *f*, he can accomplish this only by the strongest possible innervation of his paralyzed externus. Now, the ideas which we have with respect to the position of our eyes depend upon our sensations with regard to the innervation of the individual muscles. The patient, therefore, is compelled to believe that the right eye is standing in the most extreme position of right lateral rotation, like the eye represented by the dotted line in Fig. 323, because he has sent an impulse for a rotation to this amount into the right externus, and he cannot know that the latter, owing to the impairment of the conduction, only partially obeys this impulse. He hence proceeds upon the assumption that the right eye is turned very strongly to the right, and that consequently its fovea is at f_1; he is, therefore, also compelled to believe that the object whose image is formed at the fovea lies opposite f_1—i. e., at o_1—and he hence sees the object too far to the right. Accordingly, objects which are sighted by the paralyzed eye are always seen too far toward that side to which the paralyzed muscle moves the eye.

653. 3. *Diplopia.*—This occurs when vision is performed with both eyes simultaneously and the visual lines do not intersect in the point of fixation; it is the consequence of false orientation of the paralyzed eye. The explanation of the way in which the double images are produced, and of the way in which they behave in the different abnormal positions of the eye, has been given on pages 728 et seq. (for Hering's explanation see page 733). The double images are the most important means that we can call to our aid in making the exact diagnosis of paralyses.

The phenomena so far mentioned, such as restriction of motility, strabismus, false orientation, and diplopia, make their appearance only when the eyes are moving in the sphere of action of the paralyzed muscle, and become more and more marked in proportion as the eyes are moved toward this side. Thus, in complete paralysis of the right externus the double images and the strabismus make their appearance the moment the eyes pass to the right of the median line [in fact, at a point some 15° or 20° to the left of the median line—D.] The more the gaze is turned in this direction, the farther apart are the double images and the more conspicuous is the strabismus. If we should have an incomplete paralysis (paresis) of the right externus before us, the double images and the strabismus would not show themselves until the eyes had been turned pretty far to the right, and in extreme cases not until the gaze was directed quite laterally (as when, for example, the patient looked straight to the right). From the direction of the eyes in which strabismus and double images first make their appearance, from the position of these double images with respect to each other, and from the way in which their distance apart increases or diminishes according to the different directions in which the eyes are looking, we

diagnosticate which of the ocular muscles is paralyzed, and whether we are dealing with a complete or an incomplete paralysis.

654. 4. *Vertigo.*—This may be excited by the diplopia, or may also occur when vision is performed with the paralyzed eye alone. The latter sees objects in their proper place so long as it occupies a direction of the gaze in which the paralyzed muscle is not called upon to exert itself. But as soon as the gaze is turned to the side representing the field of action of the paralyzed muscle, objects are located by the eye too far toward the same side, and the more so, the more the gaze is directed that way. Consequently, as the gaze passes from the region of correct to the region of false localization, objects appear to fly with constantly accelerated velocity in the direction in which the eye is moving. It is this apparent movement of the

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[Fig. 324 A.]

[Fig. 324 B.]

[A. Head-tilting in case of congenital paralysis of the superior rectus with spasm of the inferior oblique. That the tilting was really due to the muscular deviation is shown by Fig. B, which indicates the result that immediately followed a complete tenotomy of the inferior oblique.—D.]

whole outside world that determines the development of vertigo. Vertigo, therefore, sets in whenever the patient feels himself called upon to move his eyes, and hence often even in walking upon a level floor, but still more in going up and down steps, in performing complicated manipulations, in doing work, etc. It makes the patient unsteady and timid, and even excites a tendency to vomit. This kind of vertigo is known as visual vertigo, and is distinguished from other kinds by the fact that it disappears at once when the paralyzed eye is covered. Most patients hit upon this fact themselves, and in walking keep the paralyzed eye closed, either closed or covered. Another way of preserving themselves from visual vertigo lies in—

655. 5. *Head Tilting.*—A patient in whom, for example, the right externus is paralyzed, keeps the head turned to the right. If he looks forward with his head in this position, both eyes are turned somewhat to the

left, in which position the right externus does not come into play, and in which, therefore, paralysis of it does not make itself evident. And so for every variety of paralysis of the ocular muscles there is a definite position of the head, which diminishes the visual vertigo, and which is so characteristic of the paralysis that the skilled observer is able from it alone to suspect the nature of the latter.

[In paralysis of a right rotator, the patient turns his head to the right, so that in looking at objects ahead of him the eyes themselves shall be deflected to the left. Similarly in paralysis of a left rotator he turns the head to the left. In paralysis of an elevator or depressor he usually tilts the head to one shoulder, as in this way he can more readily obviate the vertical diplopia. In any case of combined vertical and lateral diplopia, tilting of the head toward the right shoulder will bring the right-hand image down. This is true whether the right-hand image belongs to the right eye (in case the diplopia is homonymous) or to the left eye (in case the diplopia is crossed). A vertical diplopia of 25° or more may be obviated in this way, the patient tipping the head toward the right or the left shoulder according as the right-hand or left-hand image is higher. The patient may combine this side-tipping with more or less lateral rotation and up or down tipping, but the side-tipping is the predominant and often the only abnormality of attitude.—D.] If with continuance of the paralysis such an attitude of the head becomes habitual, it may readily be confounded with wry-neck [particularly in congenital paralyses.—D.] If in such cases we set the head straight the upward deflection of the paralyzed eye comes to light and enables us to make the correct diagnosis.

656. Old Paralyses.—The characteristic symptoms of a paralysis are more unmixed and more pronounced the more recent it is. If the paralysis gets well after the lapse of not too long a time, the symptoms that have been produced by it disappear, and normal binocular vision is restored; if, on the other hand, the cure of the paralysis takes place only after a long time has elapsed, or does not take place at all, the symptomatic picture changes as follows: 1. The mistakes in orientation, particularly as they make their appearance in the projection test, gradually cease; the patient learns by experience that the impulses of innervation for his paralyzed eye correspond to a much slighter action than those for the sound eye, and by taking account of this fact he once more forms a correct judgment of the situation of objects. 2. The diplopia disappears because the sensory perceptions of the paralyzed eye are suppressed (exclusion).[8] 3. Contracture of the antagonists of the paralyzed muscle gradually sets in. Thus, in paralysis of the right externus, it is the right internus that becomes shortened; and so, while in a recent paralysis of the externus, the eye, when the gaze is directed straight forward, stands in the middle line, it afterward becomes drawn in more and more, and can no longer be brought up to the median position. The result of this is an increase in the paralytic strabismus, this reaching a higher degree and becoming manifested over a more extensive area than before, insomuch that it is present not only upon the side of the paralyzed

[8] [It takes a long time for this to occur; and even when there is apparent suppression diplopia can almost always be elicited by the tests.—D.]

muscle but also over the entire field of fixation. Owing to this fact, paralytic strabismus acquires a constantly greater and greater resemblance to concomitant squint, so that sometimes the distinction between the two becomes very difficult.[9] The contracture of the antagonists may even persist when the paralysis itself is cured, and may thus prevent the restoration of normal binocular vision.

657. Mode of Occurrence.—Paralysis may affect simply one muscle, or it may affect several muscles in different combinations.

1. Paralysis of *one single muscle* most usually affects either the external rectus or the superior oblique, because each one of these muscles is supplied by an independent nerve (abducens and trochlearis). All the other ocular muscles are innervated by the oculo-motor nerve, for which reason paralysis of any single one of them is of less frequent occurrence. [Isolated paralysis of the superior rectus is quite common (more frequent than trochlear paralysis), and pareses of the inferior rectus (usually very slight) are fairly common.—D.]

2. For the reason just mentioned, *simultaneous paralysis of several muscles* is found most frequently in those supplied by the oculo-motor nerve, and of these muscles some or all may be affected at once. *Complete oculo-motor paralysis* presents a characteristic picture. The upper lid hangs loosely down (ptosis), and has to be drawn up with the finger to give a view of the eyeball, which is deflected strongly outward and somewhat down, because the two muscles not paralyzed—the external rectus and the superior oblique—draw it in this direction. The pupil is dilated and immobile (paralysis of the sphincter pupillæ), and the eye is focused for the far point and cannot accommodate for near by (paralysis of the ciliary muscle). A slight degree of exophthalmus is present because three of the recti, which normally draw the eyeball backward into the orbit, have lost their tone.

Other muscles besides those innervated by the oculo-motor nerve may be affected, and the paralyses may affect not simply one but both eyes. In this way many manifold combinations are formed, of which the following are the most frequent: (*a*) All the eye muscles in one or both eyes are paralyzed, so that the lids hang loosely down, the eyes are directed straight forward, and are immovable, and there is dilatation of the pupil, with abolition of the accommodation (*ophthalmoplegia totalis*). (*b*) The paralysis affects only the exterior eye muscles, while the interior muscles of the eye (sphincter pupillæ and ciliary muscle) are intact (*ophthalmoplegia externa* [sive *exterior*]). This is more frequent than total ophthalmoplegia, and finds its explanation in the fact that the nuclei for the sphincter pupillæ and the ciliary muscle are distinct from the other nerve nuclei (Fig. 308), and hence frequently remain exempt from processes which destroy the nuclei of the other ocular muscles. For this reason ophthalmoplegia exterior in the

[9] [Indeed, many cases of concomitant squint are without doubt paralytic in origin.—D.]

majority of cases is of central (nuclear) origin. (*c*) *Ophthalmoplegia interna* [sive *interior*] forms the converse to ophthalmoplegia externa, as in it only the interior muscles of the eye are paralyzed. It can be produced artificially by means of atropine.

3. There are combined paralyses which do not affect the individual muscles, but affect associate movements. Thus the ability to look to the right or left, or to look up or down, etc., or to converge, may be lost. These are known as *conjugate paralyses* (Prévost). The most characteristic cases of this sort are those of paralysis of the lateral rotators. There may, for example, be a paralysis of the associated movements to the right. If the patient then fixes his gaze upon an object which is carried in front of him from left to right, the eyes follow it until it has got to the middle line; then both eyes stand still without being able to move farther to the right. One might suppose that he was dealing with a paralysis of the right externus combined with one of the left internus. But this idea can be readily disproved by approximating an object to the patient along the middle line. The patient converges upon the object until it is very close to him, and hence can use his left internus perfectly for purposes of convergence, while the same muscle is paralyzed in its capacity of rotator to the right. The causes of conjugate paralysis are lesions in the association centers of the nerves for the ocular muscles. [See also §§ 663 and 701.]

[**658. Diagnosis.**[10]—The diagnosis of paralysis is based mainly on three points: The restriction of movement, the deviation produced, and the double vision. In this regard the cardinal fact to be kept in mind is that all these objective evidences of paralysis (and also the subjective symptoms of vertigo and confusion) increase in proportion as the eyes are carried into the field of action of the paralyzed muscle—i. e. when they are carried into one particular cardinal direction of the gaze (see page 720). In order, therefore, to establish the diagnosis, it is essential to make the eyes move successively to the right, left, up and right, up and left, down and right, and down and left, and determine their behavior in each position.—D.]

[This is obviously so because when the eyes are carried into the field of action in which the paralyzed muscle is normally most effective, and both eyes attempt to proceed in that cardinal direction, both receive an equal nerve impulse (page 738). The normal eye responds normally and proceeds on its way as usual; the paralyzed eye responds feebly or not at all, and therefore lags behind the other. In all other directions of the gaze the restriction in movement of the affected muscle is slight or affects only its subsidary action (see page 717) and the lagging also is slight or nil. On the lagging depend the deviation and the diplopia, which consequently increase and decrease with it.—D.]

[In making all the tests, it is essential, first, that the head shall be kept steady, so that the eyes alone move; and, second, that the patient be not

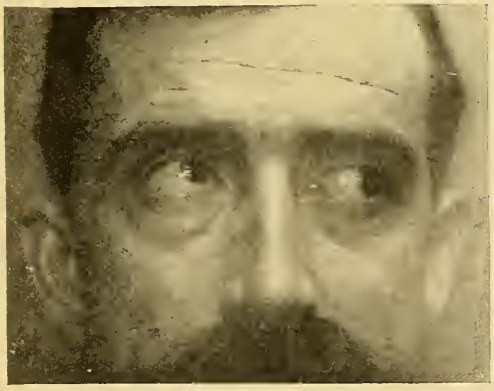
[10] [These sections (658 to 661) on diagnosis replace a number of pages in the original, in which the significance of diplopia in paralysis is considered at length along somewhat different lines. For the change the translator is solely responsible —D.]

allowed to tilt his head, as in this way we may obviate part or all of his deviation and diplopia (see page 741).—D.]

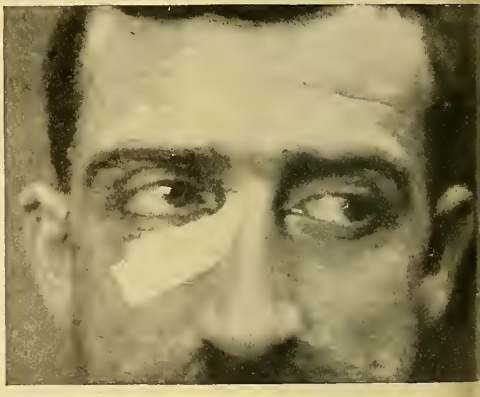
[**659.** (1) *Restriction of Movement.*—This is observed by making the eyes follow an object, e. g. a small white-headed pin, carried successively in the six cardinal directions. The paretic eye will lag when it enters the field of action of the paralyzed muscle and will lag more and more the further it is carried into this field. Hence the following table [11] holds good:

	R. lags	Paralysis of	L. lags	Paralysis of
Er.....	In.....	R. external rectus.....	Out.....	L. internal rectus.
El.....	Out.....	R. internal rectus.....	In.....	L. external rectus.
Eu & R.....	Down.....	R. superior rectus.....	Down.....	L. inferior oblique.
Eu & l.....	Down.....	R. inferior oblique.....	Down.....	L. superior rectus.
Ed & r.....	Up.....	R. inferior rectus.....	Up.....	L. superior oblique.
Ed & l.....	Up.....	R. superior oblique....	Up.....	L. inferior rectus.

The observation in this manner of the way in which the movement of the paralyzed eye is restricted constitutes the *excursion test.* But we should



A



B

[Fig. 325.—Secondary Deviation in Paralysis. (After Posey).

A, Paralysis of the left superior rectus with marked upshoot of the right eye, when the gaze is directed to the left. That the upshoot was due largely to the overaction of the right inferior oblique is demonstrated in B, which shows how the upshoot disappeared as a result of tenotomy of this muscle.—D.]

also observe the way in which the other eye behaves. For naturally the non-paralyzed eye follows the object and the other deviates, but when, either because the object is hidden from the non-paralyzed eye by the nose or for other reasons, the paralyzed eye takes up fixation, the paralyzed eye will be seen to make a sudden extreme movement (secondary deviation, spastic shoot) in the direction in which the eyes are moving. The explanation for this has already been given (page 738), namely that the paralyzed

[11 In this table Er, El, Eu & r, Ed & r, etc., denote "when both eyes are directed to the right, left, up and right, down and right," etc.—D.]

eye when called on to fix and particularly when called on to move in the given direction, being crippled has to exert an excessive effort. This calls for an excessive nerve impulse, which, according to the principle stated on page 738, is conveyed in equal measure to the other eye and makes that move excessively. The excessive movement is due mainly to overaction of the associate (see page 720) of the paralyzed muscle (see Fig. 325). For the same reason the diplopia increases when fixation is shifted from the non-paralyzed to the paralyzed eye (page 748).—D.]

[In order to examine this primary restriction and the secondary overaction systematically it is well to slant a card in front of the patient's nose in such a way that in following the test-object he has to fix first with one eye, then with the other. The results may be written in tabular form, thus:

	R. fixing	L. fixing
Eu & r	R. follows imperfectly; L. shoots higher	L. follows readily; R. lags.
Eu & l	R. normal; L. diverges slightly	L. normal; R. diverges very slightly.
Ed & r	R. and L. normal	R. and L. normal.
Ed & l	R. and L. normal	R. and L. normal.

The test thus conducted is called the *comitance test.*—D.]

[We may also measure the restriction of movement produced by paralysis by taking the field of fixation with the perimeter or tropometer (see § 676), and noting how this field is diminished in any one of the six cardinal directions. But this is rarely necessary and, moreover, does not afford as reliable indications as the other test given.—D.]

[660. (2) *Deviation.*—The deviation is best determined by alternate covering of each eye with the screen, the eyes being directed fixedly at an object (dot on a card) carried successively in the six cardinal directions. (For details of test, see § 674). We then find the eye behind the screen deviated, and this deviation increases as the eyes are carried into the field of action of the paralyzed muscle (see page 748).—D.]

[Because the paralyzed eye lags, it will deviate in a direction opposite to that in which the affected muscle would normally carry it—i. e., opposite to that in which the eye is moving. And because the non-paralyzed eye acts excessively when the paralyzed eye fixes, it will when screened deviate strongly in the direction in which the eyes are already moving. Thus in paralysis of the right external rectus, when the eyes are carried into the field of action of this muscle, i. e., to the right, the right eye will lag behind, i. e., behind the screen it will deviate to the left, or lag inward. The left eye under the same conditions will when screened deviate to the right, i. e., will shoot inward. So in case of paralysis of the right internal rectus, both eyes will deviate out behind the screen, as soon as the eyes are carried to the left. On a similar principle, in paralysis of the right superior rectus, when the eyes are carried in to the field of action of this muscle, i. e., up and to the right, the right eye will drop and the left eye will shoot up behind the screen. Moreover, since the right superior rectus is an adductor, the right eye when this muscle is paralyzed will not be adducted as well as formerly, and

hence will tend to diverge. This divergence will be particularly marked when the eyes are directed up and to the left, because it is in this direction of the gaze that the right superior rectus is most effective as an adductor. This divergence, however, is comparatively slight, and may be masked or overcome by other conditions present. Thus, if there is a condition producing a constant convergence (esophoria), this may be great enough to neutralize in all parts of the field of fixation the divergence produced by the paralysis. In that case, instead of a moderate divergent deviation (exophoria) increasing up and to the left we should find a moderate esophoria diminishing,[12] in the same direction.—D.]

[By applying a like reasoning to all cases, we get the table on page 748.—D.]

[In applying the screen test we will observe that in any given case that, as stated on page 737, the deviation of the non-paralyzed eye when covered (*secondary deviation*) is greater than that of the paralyzed eye. Thus in paralysis of the left superior rectus, the deviation of the left eye down is less than that of the right eye up under cover. In fact, the right eye in such a case, when screened or when it gets behind the nose, regularly makes a marked up-shoot (spasmodic secondary deviation—cf. Fig. 325).—D.]

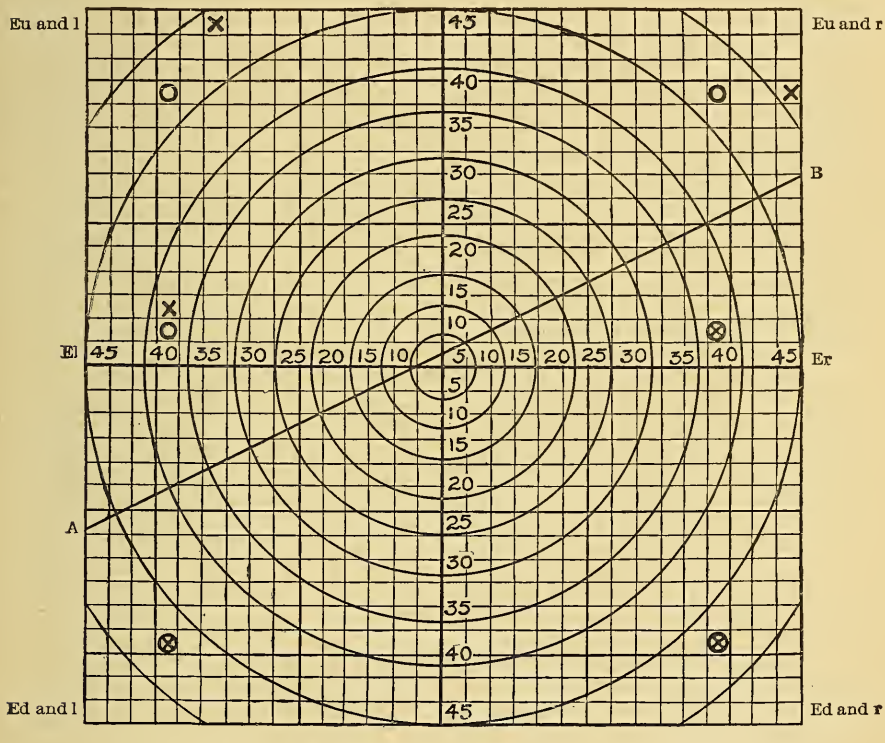
[The *amount* of the deviation may be measured by the strength of the prism required to abolish the movement of redress (see § 674). We can thus compare measurements taken at different times, and gauge the progress of the paralysis. A prism placed over the paralyzed eye measures the primary, and over the non-paralyzed eye, the secondary deviation.—D.]

661. (3) *Diplopia.*—The diplopia, being dependent on the deviation, has a character corresponding with the latter. Accordingly, paralytic deviations that produce an inward deviation are associated with an homonymous diplopia, those that produce an outward deviation are associated with a crossed diplopia, and those that produce a vertical deviation with a vertical diplopia (right or left diplopia, corresponding to a right or left hyperphoria). Moreover, the diplopia increases precisely as the deviation does. Hence all the remarks made with regard to the deviation are applicable to the diplopia as well.—D.]

[By far the best means for determining the *kind* and *amount* of diplopia in paralysis is the tangent plane, or modified Bjerrum's curtain (Fig. 34). In applying this we place the patient 75 cm. from the curtain, facing the black side. A red glass is placed before the right eye, and the head is so adjusted that, when the eyes are directed at the center pin, they are on a level with it and looking straight ahead. A small electric light is then carried over the curtain in the six cardinal directions of the gaze, and the point where diplopia occurs in each meridian is noted by thrusting in a pin. The limits of the field of single vision are thus delimited. In the region in

[12] Diminishing because it is there partially neutralized by the increasing exophoria produced by the paralysis of the superior rectus. Actually, the way in which such an anomalous esophoria diminishes may be used to confirm the diagnosis.—D.]

which diplopia occurs, the situation of the double images is similarly indicated. The patient sees two lights, a red and a white. The red image belongs to the right eye and the white image to the left. A black pin is thrust into the curtain at the site of the candle itself, and a light-colored pin at the site of the other image. This latter is evidently the false image and, if it is red, we know that the left eye is fixing; if it is white, we know that the right eye is fixing. The situation of the pins indicates the relation of the double images and their degree of separation.—D.]



[Fig. 326.—Double Images Plotted on Tangent Plane (from Posey and Spiller).

The reverse (white) side of the curtain forming the tangent plane (page 116) is here shown (seen from in front). The other (black) side is turned toward the patient, who is placed 30 inches from it, with his eyes opposite the center. A red glass is put before his right eye, and a small electric light is carried in the six cardinal directions, "Eyes right" (Er), "Eyes up and right" (Eu and r), etc., the patient following it with his eyes but not moving his head. The position of the double images is marked by pins thrust into the curtain. In the case shown (paresis of the left superior rectus) the right eye (red) image is denoted by a circle, the left eye (white) image by a cross. There is a vertical (right) diplopia of 5° and a crossed diplopia of 2°, when the eyes are directed two feet up and two feet to the left, and a simple crossed diplopia of 5° when they are directed up and to the right. Below the line AB there is single vision. It is found that the red image coincides with the actual position of the light on the screen; hence the right eye fixes, and the distance between the double images measures the primary deviation. (If the white image had coincided with the light the distance would have measured the secondary deviation.)—D.]

[In the great majority of cases the patient fixes with the eye not covered with the red glass, and, therefore, by shifting the red glass from one eye to the other, we can

often get him to alternate fixation. We can then mark by a pair of pins the situation of the false image, when the right eye and the left eye respectively fix, and thus indicate the absolute and relative amount of the *primary* and *secondary deviation* for any given direction of the gaze. The plot which is formed by the pins on the curtain and which shows the limits of the field of single vision and the amount and kind of diplopia is transferred with the aid of the diagram on the back of the curtain to a similarly marked card—see Fig. 326). This forms a permanent record, which can be compared with a similar one taken at any future time.—D.]

[A measurement of the amount of diplopia can also be made by determining the strength of *prism* required to bring the two images together. In this case the prism should be placed over the paralyzed eye. It will then indicate the amount of the *primary* deviation. If placed over the non-paralyzed eye it will indicate the amount of the *secondary* deviation. Measurement of diplopia by prisms gives somewhat uncertain results, often affording an underestimate of the deviation. The reason for this is that when a prism is applied that corrects nearly all of the diplopia the patient may overcome the rest by involuntary effort.—D.]

[The *types of diplopia* found in each variety of paralysis are shown in Figs. 327-338. The diagnosis by double images can be much better deduced, however, from the accompanying table, which also shows the types of screen deviation present.—D.]

[Table of Diplopia and Deviation in Paralysis[13]

In the accompanying table DH, DX, DR, and DL denote respectively homonymous diplopia, crossed diplopia, right diplopia (vertical diplopia with the image of the right eye below), and left diplopia (vertical diplopia with the image of the left eye below). So also S, X, RH, and LH, denote a deviation in (esophoria), out (exophoria), right hyperphoria, and left hyperphoria. >> denotes "increasing progressively."

Paralysis of		Regularly (but not invariably) associated with
R. external rectus.....	DH and S >> greatly in Er.	
L. internal rectus.....	DX and X >> " " Er.	
R. internal rectus.....	DX and X >> " " El.	
L. external rectus.....	DH and S >> " " El.	
R. superior rectus.....	DL and LH >> " "Eu & r.	DX and X >> in Eu & l.
L. inferior oblique.....	DR and RH >> " "Eu & r.	DH and S >> in Eu & l.
R. inferior oblique.....	DL and LH >> " "Eu & l.	DH and S >> in Eu & r.
L. superior rectus.....	DR and RH >> " "Eu & l.	DX and X >> in Eu & r.
R. inferior rectus.....	DR and RH >> " "Ed & r.	DX and X >> in Ed & l.
L. superior oblique.....	DL and LH >> " "Ed & r.	DH and S >> in Ed & l.
R. superior oblique.....	DR and RH >> " "Ed & l.	DH and S >> in Ed & r.
L. inferior rectus.....	DL and LH >> " "Ed & l.	DX and X >> in Ed & r.

[13] This table serves for determining combined paralyses. Thus if there is a DL (left hyperphoria) in Eu and r, diminishing to nothing as the middle line is approached and changing to a DR (right hyperphoria) in Eu and l, there is a paralysis of both superior recti; if there is a DR in Eu and l changing to DL in Ed and l, there is paralysis of the left superior and inferior recti.—D.]

Behavior of the Double Images in Paralysis of the Ocular Muscles

(The apparent image has a dotted outline.)

Left-sided Paralysis.

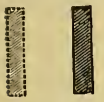


Fig. 327.

External Rectus.

Diplopia appears in looking toward the paralyzed side.

The lateral separation of the images increases as the paralyzed eye is abducted.

Right-sided Paralysis.

Fig. 328.

Internal Rectus.

Fig. 329.

Diplopia on looking toward the sound side.

The lateral separation of the images increases in adduction of the paralyzed eye.

Fig. 330.

Superior Rectus.

Fig. 331.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes [as the eye is abducted; increases as it is adducted].

Fig. 332.

Inferior Rectus.

Fig. 333.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes [as the eye is abducted; increases as it is adducted].

Fig. 334.

Superior Oblique.

Fig. 335.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and adducted.

The obliquity increases with the abduction.

The lateral distance between the images diminishes [as the eye is adducted, increases as it is abducted].

Fig. 336.

Inferior Oblique.

Fig. 337.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and adducted.

The obliquity increases with the abduction.

The lateral distance between the images increases as the eye is elevated and abducted.

Fig. 338.

[The diagnosis of paralysis by the double images can also be readily and simply accomplished by an *analysis* which shows in succession, first which group of muscles (lateral rotators, elevators, or depressors) is affected; next which pair of associates in the group; and lastly which muscle in the pair.

Thus, if we have a diplopia which is mainly lateral and which increases fast[14] in looking to the right or the left, the paralysis must affect a *lateral rotator*. If the diplopia increases to the right the paralysis affects a right rotator (right externus, left internus), if it increases to the left a left rotator (left externus, right internus). If the diplopia in either case is homonymous, it is the externus which is paralyzed, if the diplopia is crossed, it is the internus.

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking up, the paralysis affects an *elevator*. If then the vertical diplopia increases most in looking up and to the right, the muscle paralyzed must be a right-hand elevator (right superior rectus, left inferior oblique); if it increases up and to the left, the muscle paralyzed is a left-hand elevator (left superior rectus, right inferior oblique). In either case we can tell which eye and therefore which muscle is affected by remembering that the higher image belongs to the paralyzed eye (e. g., right-eye image is higher in paralysis of the right superior rectus and right inferior oblique).

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking down, the paralysis affects a *depressor*. If then the vertical diplopia increases most in looking down and to the right, the muscle paralyzed must be a right-hand depressor (right inferior rectus, left superior oblique); if it increases most in looking down and to the left, the muscle paralyzed is a left-hand depressor (left inferior rectus, right superior oblique). In either case we can tell the eye and therefore the muscle affected by remembering that the lower image belongs to the paralyzed eye.—D.]

[In any case to tell the muscle that is paralyzed we have simply to remember that the *image formed by the paralyzed eye always lies on the side toward which the diplopia increases* (i. e., is the right-hand image when the diplopia increases to the right, the upper image when the diplopia increases upward, etc.).—D.]

[And a rule to determine the kind of double vision produced by a given paralysis is,—*the image belonging to the paralyzed eye is displaced in just that direction in which the affected muscle, if intact, would naturally move the eye.* Thus, in paralysis of the right superior rectus, which moves the right eye up and to the left and tilts the vertical meridian to the left, the image of the right eye is up (higher), deflected to the left (crossed diplopia), and tilted to the left.

This rule shows us that in paralysis of the subsidiary adductors (superior rectus, inferior rectus) we expect to find a crossed diplopia, and in paralysis of the subsidiary abductors (the two obliques) we shall find an homonymous diplopia; also in paralysis of the superior rectus and superior oblique an in-tilting and in paralysis of the inferior rectus and inferior oblique an out-tilting of the false image. As before remarked, however, the lateral diplopia may be absent or reversed[14] and the tilting is often but little marked, so that neither is a safe guide in diagnosis.—D.]

[The facts with regard to the *tilting of the double images* in paralysis may be stated a little more in detail. Since the superior rectus and superior oblique rotate the vertical meridian inward, or intort the eye, the latter in case of paralysis of these muscles will be extorted, or tilted outward, and, according to the reasoning on page 730, the image formed by it will be tilted in the opposite direction, or inward. In paralysis of the

[14] [It is to be noted that an homonymous or a crossed diplopia which increases to the right or left does not necessarily mean paralysis of a lateral rotator, unless the diplopia is great and increases rapidly. An homonymous diplopia which is of slight amount and increases moderately in looking to the right may mean (*a*) paresis of *any* abductor (externus, superior oblique, inferior oblique) of the right eye, or (*b*) paresis of *any* adductor (internus, superior rectus, inferior rectus) of the right eye when combined with an esophoria sufficient to neutralize the crossed diplopia that such paresis would naturally produce. So a crossed diplopia which increases to the right may mean paresis of *any* muscle of the left eye.—D.]

superior rectus, the tilting will be observed in the upper field in paralysis of the superior oblique, in the lower. In paralysis of the inferior rectus and inferior oblique, for similar reasons the image formed by the paralyzed eye will appear tilted outward. The tilting increases in the direction of the gaze where the lateral diplopia due to the elevator or depressor paralysis also increases. These facts are shown in Figs. 331-338 and may also be stated in the following tabular form:

Muscle paralyzed	Image tilted to	Tilting most marked in
R. superior rectus	Left	Eu & l.
L. inferior oblique	Left	Eu & l.
R. inferior oblique	Right	Eu & r.
L. superior rectus	Right	Eu & r.
R. inferior rectus	Right	Ed & l.
L. superior oblique	Right	Ed & l.
R. superior oblique	Left	Ed & r.
L. inferior rectus	Left	Ed & r.

Tilting of the double images may be determined by taking the patient's judgment as to the relative position of the two images of a candle; or, better, may be determined and also measured with the Maddox rod or the clinometer (see page 771), which will also indicate surely in which eye the tilting takes place.—D.]

Frequently a patient affected with a paralysis of a depressor muscle (inferior rectus or superior oblique) is not at all aware of there being a difference in level between the double images, stating only that *one image is nearer* than the other. This is the more apt to be the case the more the plane of fixation is depressed, and is accounted for by Förster as follows: If we gaze at a rather distant point situated in the plane of the floor upon which we are standing, those points of the floor that lie nearer us form their images higher up in the retina than does the point of fixation. If, again, we fix the upper of two points which lie one beneath the other, the lower point in this instance also forms its image above the macula. When, therefore, we have upon our retina two images situated one above the other, these are open to a double interpretation; they may be produced either by two objects one of which is situated nearer than the other, or by two objects one of which is lower than the other. Now, if, owing to a paralysis of a depressor, one eye stands too high, the point which the sound eye fixes will form its image in the paralyzed eye on a portion of the retina that also is situated above the macula. Then the difference in height between images in the two retinæ may be interpreted in two different ways, just as if the two images were situated in the same retina. The patient may suppose the false image to be either lower or nearer. In Fig. 339 let the undotted figure represent the sound eye which is fixing the point O situated below the horizontal plane, i. e., is so directed that this point forms its image on the fovea, f, of the eye. The paralyzed eye represented by the dotted figure has not shared in the downward movement; its line of sight ϕx remains directed straight forward. Hence in this eye the point O forms its image above the fovea, ϕ, that is at β. Accordingly, this eye projects the point too far down and as much so as β is too far up, that is in the direction *by*. (According to Hering the paralyzed eye must see the point O at the spot where the point on the retina of the sound eye which corresponds to the point β occupied by the image of O would localize an image. This corresponding point is b, which is situated as much above the fovea f of the sound eye as β is above the fovea ϕ of the paralyzed eye. The point O therefore is seen along the directing line *by*. Cf. page 733.) Now the point O may be regarded as located at different places along the directing line *by*, thus either at m so that the false image lies below the true

image, or at n, so that it stands on the same level but closer than the true image, or somewhere between these two points, for example at r, in which case the false image appears somewhat lower and at the same time somewhat closer than the true image. That kind of projection which makes the false image look closer is actually forced upon the patient if the test object is set on a table or on the floor. The paralyzed eye ought then to see the candle flame beneath the table or beneath the floor. But this so contradicts experience that the patient involuntarily chooses that kind of projection by which the image of the higher eye appears on the table or floor but closer to the eye. [By varying the conditions of the experiment, e. g., by placing the candle on a descending flight of stairs, the image which should be lower may be made to appear not only closer but also higher than the other (Sachs).—D.]

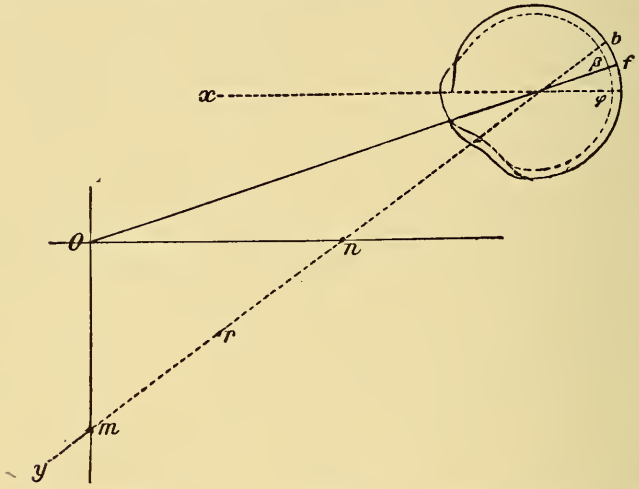


Fig. 339.—Apparent Approximation of the False Image in Paralysis of a Depressor.

662. Complex Cases.—The diagnosis as to which muscle is paralyzed often presents considerable difficulties even to the adept, if the case is complicated. This occurs—

1. When several paralyses are combined, particularly in both eyes, and the paralyses are partly complete, partly incomplete.

2. When a disturbance of muscular equilibrium under the form of latent convergence or divergence (esophoria or exophoria) was previously present. Such a disturbance is converted from a latent into a manifest one when the paralysis sets in, as, owing to the latter, binocular vision becomes impossible in spite of the tendency toward fusion.

3. When the two eyes have an unequal visual power, and the paralysis affects the better eye. The latter then is used to perform fixation with,[15] and the non-paralyzed eye is in a condition of secondary deviation. In such a case it is easy for the sound eye to be regarded as the paralyzed one.

4. When, in old paralyses, a contracture of the antagonists has taken place.

The difficulties of diagnosis are often increased by lack of intelligence or by insufficient attention on the part of the patient, in consequence of which it is impossible to determine with precision the position of the double images. It may also be impossible

[15] [This may even occur when the paralyzed eye does not see as well as the other.—D.]

to determine their position when, as in old paralyses, there is a tendency toward suppression of the double images. Then it often requires long and painstaking efforts to get the patient to perceive the remote and indistinct false image.

[Even these complex cases can usually be unravelled, if care is taken to use all the methods of diagnosis (excursion comitance, screen and diplopia tests) in all the cardinal directions of the gaze. In occasional doubtful cases of slight paralysis the use of the Maddox rod or the clinometer to determine which eye is pathologically tilted and which, therefore, is affected by the paralysis, may be of service.—D.]

663. Etiology; Site of Causal Lesion.—Paralyses of the ocular muscles are the result of a lesion which may be situated anywhere in the course of the nerve tract, from its very beginning in the cerebral cortex to its termination in the muscle itself. According to the site of the lesion, paralyses are distinguished into intracranial and orbital.

In *intracranial* paralyses the focus of disease lies within the cranial cavity. It may affect the centers of highest rank which lie in the cortex of the brain (cortical paralysis), or the association centers, or, lastly, the centers of lowest rank—i. e., the nerve nuclei upon the floor of the fourth ventricle (nuclear paralysis). The bands of fibers, likewise, that connect these centers may be affected, as may also be those fibers that run from the nuclei to the surface of the brain and unite there to form the nerve trunks (fascicular paralysis); and the nerve trunks themselves may be affected in their course along the base of the skull (basal paralysis).

Orbital paralyses are those in which the lesion is seated in the nerve trunk and its branches, commencing from the entrance of the nerve into the orbit through the superior orbital fissure, or those in which the muscle itself is affected.

To diagnosticate the site of the lesion we must take account of the character of the paralysis, and particularly of those accompanying symptoms that point to an intracranial or to an orbital lesion.

1. Lesions of the *centers of higher rank*, situated above the nerve nuclei (that is, the cerebral cortex, the association centers, and the fibers connecting these parts with one another and with the nuclei—regions which are all comprised under the name of intracerebral tracts), never cause paralyses of individual ocular muscles. If, therefore, isolated paralyses are present, lesions of as high a situation as this can be excluded. The only exception is *ptosis*, as this sometimes is met with as an isolated phenomenon in cortical affections. Otherwise lesions of the higher centers always cause *conjugate paralyses*. The eyes are unable to turn in some special direction, or they cannot be made to converge [or diverge]. In the given case the eyes are found not infrequently to be drawn toward the opposite side by a spasmodic contraction of the antagonists. Thus, for example, in paralysis of the lateral rotators to the right, not only is it impossible to turn the eyes to the right, but it may be that both eyes are turned continuously and strongly to the left (conjugate deviation). Conjugate paralyses, with or without deviation of the eyes to the opposite side, occur in diseases of the crura cerebelli ad pontem, of the pons, of the corpora quadrigemina, of the great ganglia of the brain, particularly of the thalamus opticus, and of the cortex. [See also page 797 and, for paralysis of divergence and convergence, pages 781 and 784.]

2. Lesions of the *nuclei* on the floor of the ventricle (nuclear paralyses) produce for the most part paralyses of several ocular muscles. In this way there is developed what is known as *central ophthalmoplegia*. As a rule this is bilateral, for since fibers go from each oculo-motor nucleus to the muscles of both eyes, a lesion of the nucleus even though unilateral must also make its appearance in the muscles of both eyes. The ptosis, in comparison with the complete paralysis of the other muscles, is often conspicuously slight. In most cases the interior muscles of the eye (those of the pupil and accommodation) remain exempt from the paralysis—i. e., it is an ophthalmoplegia externa. In the latter, the lesion is generally seated in the nerve nuclei. Ophthalmoplegia externa does, indeed, occur with basal lesions, but, of course, it will rarely happen that a lesion which affects the nerve trunk itself should out of all the fibers spare just those that are designed for the interior muscles of the eye. [Yet an ophthalmoplegia exterior of basal site seems to be not so very rare. It may also be fascicular in origin. —D.] If there is an ophthalmoplegia totalis—that is, one in which all the muscles are paralyzed without exception—the site of the lesion may vary. In bilateral ophthalmoplegia we may be dealing with a nuclear paralysis, while an unilateral ophthalmoplegia totalis commonly originates in a lesion of the trunk of the nerve at the base of the brain, or even within the superior orbital fissure.

A primary affection of the gray substance of the nuclei of the nerves of the ocular muscles lies at the bottom of most cases of ophthalmoplegia. In its nature this affection is analogous to that which in bulbar paralysis attacks the motor nuclei situated farther down (the facial, glosso-pharyngeal, hypoglossal, and spinal accessory nuclei). Hence, the ophthalmoplegia may be combined with a bulbar paralysis.

Ophthalmoplegia occurs either in an acute or a chronic form. *Acute* ophthalmoplegia develops within a few days and is combined not infrequently with drowsiness. It is observed after poisoning (by alcohol or lead, botulismus, carbon monoxide gas) and after acute infectious diseases (diphtheria, influenza, measles, etc.), in which case also it is probably a toxic affect that is in question. It occurs, moreover, under the form of a disease analogous to the poliomyelitis of children. In all these cases there is an acute inflammation in the region of the nuclei (Wernicke's polioencephalitis superior). Such cases may go on to recovery, but may also end in death by transfer of the process to the deeper motor nuclei of the medulla oblongata with consecutive respiratory paralysis. In *chronic ophthalmoplegia* the paralysis slowly attacks one muscle after another. The anatomical change that lies at the bottom of it is not an inflammation, but a gradual degeneration and atrophy of the gray substance. The most frequent cause is syphilis, and chronic ophthalmoplegia also occurs in tabes, progressive paralysis, disseminated sclerosis, myasthenic paralysis, and Basedow's disease. Chronic ophthalmoplegia is as a rule incurable. There are also cases of congenital ophthalmoplegia.

Ophthalmoplegia interna (paralysis of the sphincter iridis and the ciliary muscle) is a frequent sign of cerebral lues, and also occurs in the early stage of tabes and paresis. Cases that develop acutely are generally referable to poisoning (by substances resembling atropine in action and by sausage poison).

Paralyses of *individual exterior muscles* also may arise as a result of lesion of the nerve nuclei. In this category belong, above all, the paralyses which appear in the beginning of tabes dorsalis, and, although somewhat less frequently, in disseminated sclerosis, and which in most cases are of nuclear origin. Tabetic paralyses often disappear in a surprisingly short time, in spite of the progress of the causal disease. But still they are apt to recur, and in many cases they remain permanently. By a nuclear lesion the abducens can be paralyzed at the same time as the facial, since the nuclei of these two nerves lie close together.

3. *Fascicular paralysis* due to lesion of the fibers between their point of departure from the nerve nuclei and their emergence at the base of the brain may be diag-

nosticated if there is paralysis of the oculo-motor nerve of one side with simultaneous paralysis of the extremities of the opposite side (crossed paralysis). In this case, then, a focus of disease must be assumed to exist in the lower part of the pedunculus cerebri (*a*, Fig. 307). Such a focus of disease causes injury both to the fibers of the oculo-motor nerve as they pass through the peduncle, so that the oculo-motor nerve of the same side is paralyzed, and to the pyramidal tract; but as the latter decussates below this point, the extremities are paralyzed on the side opposite to the lesion. Such a paralysis, however, might also be produced by a focus of disease at the base of the brain, if the disease were situated so near the peduncle as to cause injury to it. In analogous fashion a crossed paralysis of the extremities and of the abducens (and also of the facial) argues the existence of a focus of disease in the posterior part of the pons, or in the portions of the base of the brain adjoining it (*b*, Fig. 307).

4. Lesions at the *base of the brain* may likewise affect one nerve or several, and not infrequently affect both sides at once. The facts which with more or less probability lead us to infer the existence of a basal paralysis are: (*a*) When a whole series of cerebral nerves upon one side, such as those supplying the ocular muscles, the facial, auditory, the trigeminal, the optic, and the olfactory nerves, are paralyzed one after another. (*b*) When the affection of the trigeminus begins under the guise of a neuralgia, the latter not being observed in central paralyses. (*c*) When disturbances of sight of a certain sort are present. Among these are blindness of one eye without implication of the other, a condition which proves a lesion of the intracranial [or intra-orbital] segment of one optic nerve; also temporal hemiopia which indicates a lesion of the chiasm. Homonymous hemiopia occurs both in affections at the base of the skull through implication of the optic tract and also in lesions situated higher up. Hemiopia from involvement of the tract is rare and should be assumed to exist only when there are other factors that argue the presence of a basal affection. A tract hemiopia would also be inferred if there was a hemiopic pupillary reaction (page 614). The disturbance of vision can be made use of for diagnosticating the site of the lesion only when by ophthalmoscopic examination we can exclude the possibility that the disturbance of vision is located in the eye itself (e. g., as when due to papillitis or primary atrophy of the optic nerve).

Basal paralyses of the eye muscles often result from fracture of the base of the skull. The abducens is particularly often paralyzed, as it runs close by the apex of the pyramid of the petrous bone and is readily injured by it (Panas). Because also of its closeness to the petrous bone the abducens is often implicated in otitic processes. Furthermore, transient abducens paralysis often occurs after lumbar anæsthesia.

To the paralyses of basal origin belong, too, most of the cases of *periodic paralysis* of the eye muscles. Such a paralysis most frequently affects the oculo-motorius [occasionally the abducens]. The attacks are ushered in by headache, which is often associated with vomiting [ophthalmoplegic migraine]. After these symptoms have lasted for some days, the paralysis sets in. After some days or weeks this either disappears entirely or leaves a paresis of the muscles that lasts till the next attack. The disease often begins in childhood, and ends either in recovery or in permanent paralysis. Some of these cases are of a purely functional nature (hysterical), in others there is a basal lesion (a circumscribed exudate or small new growths that press on the nerve).

5. The diagnosis of an *orbital* paralysis must be made from the accompanying symptoms, when these are indicative of an affection within the orbit. Among such symptoms are pain in the orbit, either spontaneous or excited by pressure upon the eyeball or upon the margin of the orbit, a tumor deep in discoverable by palpation, protrusion of the eyeball, unilateral optic neuritis due to pressure of the optic nerve, and finally the evidences of antecedent trauma affecting the orbit. (Cf. page 624.)

664. Nature of Causal Lesion.—As regards its nature, the lesion may develop as a primary affection in the nerves or in their areas of origin, these being attacked by inflammation or by simple degeneration. Much more frequently, however, these structures suffer indirectly as a result of disease in their vicinity, such as exudates (especially in the meninges), thickenings of the periosteum, neoplasms, hæmorrhages, injuries, etc., by which the nerves or their nuclei are thrown into a condition of inflammation, are compressed, or are in some other way subjected to injury. Among the vascular changes which are to be enumerated as causes producing lesions of the nerves supplying the ocular muscles are arteriosclerosis, aneurysm, occlusion and rupture of the vessels.

The *cause* of the lesion in the intracranial paralyses is most frequently syphilis in its later stages. This causes damage to the eye muscles either directly or by the roundabout way of tabes and progressive paralysis. Among the diseases of the central nervous system requiring mention are disseminated sclerosis, vascular changes with their consequences [embolism, hæmorrhages], injuries, inflammations, and tumors. Among acute infectious diseases, diphtheria is the most frequent cause of paralyses of the eye muscles, and among disorders of metabolism, diabetes.

The causes of orbital paralyses may be tumors, injuries, or inflammations of the orbit or its vicinity, and as regards the latter the transfer of inflammation from the accessory sinuses of the nose is particularly to be thought of (see §§ 555 and 737).

In the majority of these paralyses, which from the accompanying symptoms we must regard as peripheral, none of the causes above mentioned is discoverable. Since these paralyses which as a rule take a favorable course, are frequently referred by the patients to a cold, they bear the name of rheumatic paralyses.

Myasthenia often begins in the eye muscles, usually under the form of ptosis. In this case the paralysis may pass over to the other eye muscles [besides the levator], but the interior muscles always remain exempt. The paralyses are characterized by the fact that they are made to increase rapidly by exhaustion. The diagnosis is made certain if at the same time there is feebleness of the muscles of the face, neck, and masticatory apparatus.

Paralyses of the ocular muscles may be of *congenital* occurrence. Mention has already been made of congenital ophthalmoplegia. The most frequent congenital paralyses are those of the abducens [occurring mainly in the form of the syndrome described below]. It is a remarkable fact that in these, contrary to what takes place in the acquired paralyses, contracture of the antagonists does not set in; both eyes have a perfectly proper position as long as the gaze is not directed toward the side of the paralyzed muscle. An inability to turn the eye upward has been observed occurring coincidently with congenital ptosis. Autopsies have shown that in this case the superior rectus was absent. [Congenital paresis of the superior rectus is frequent and occurs often without any coincident ptosis. In such cases the patient often fixes with the paretic eye, the other showing a marked secondary deviation upward (upshoot) when adducted (Fig. 325). Paresis of the inferior rectus is also common, al-

though it is often very slight. Another less frequent congenital deviation occurs as a syndrome in which the external rectus is nearly or quite paralyzed, the internal rectus is paretic, and the eye when turned inward retracts more or less strongly into the orbit and at the same time turns up (sometimes down) while the palpebral fissure closes (false ptosis). Many cases of so-called congenital paralysis are really due to absence, abnormal insertion, or other structural anomalies of the muscles themselves. In cases of congenital paralysis diplopia can usually be elicited, and in any event true suppression seems to be very rare. Congenital paralyses are frequently associated with a faulty position of the head, which may be noticeable early in infancy and in marked cases may simulate torticollis (see Fig. 324). They not infrequently form the starting point of regular convergent or divergent squint, which develops during the first three or four years of life. Such a squint, which is produced by the superadding of an acquired lateral deviation to a congenital deviation, usually vertical, is distinguished from ordinary squint by not being amenable to orthoptic or refractive treatment. Such cases, in fact, if they demand relief at all, require operation.—D.]

665. Course and Treatment.—The paralyses either set in suddenly or develop in an insidious manner. Sometimes relapses occur. The course of the paralyses is always chronic. Even in the most favorable cases six weeks and more are required for a cure, and many paralyses are absolutely incurable. Whether this is so or not depends mainly upon the cause which lies at the bottom of the paralyses, and which, therefore, must first of all be taken into consideration in making the prognosis. Another means for determining the latter is afforded by the duration of the paralysis, since old paralyses, on account of the secondary changes that set in (atrophy of the paralyzed muscle and contracture of its antagonist), no longer hold out any prospect of a cure.

Treatment has first of all to take account of the causal indication. In this respect syphilitic and rheumatic paralyses afford the best prognosis. In the former an energetic antisyphilitic treatment with iodine and mercury is indicated. In the latter we give salicyl compounds, and employ diaphoresis. For symptomatic treatment the local application of electricity is frequently employed (see page 57).

Besides the treatment of the paralysis itself, it seems also requisite, until the cure has been accomplished, to relieve the patient from the annoyance which the diplopia and the vertigo entail. When we are dealing with very slight paralyses we can unite the double images by means of prisms set in a suitable position; in this case the patient is made to wear the prisms under the form of glasses. [Prisms are rarely useful, because in paralysis the diplopia changes in amount each time the eye is moved, so that at one moment is it over-corrected, at another under-corrected by the prism.—D.] In more marked paralyses prisms do not suffice to compensate for the incorrect position of the eyes. Then there is no other way of relieving diplopia than to bandage the paralyzed eye, or, better still, to apply glasses which have an opaque plate for the paralyzed eye.

In old paralyses in which contracture of the antagonists has set in, we

can get a result only by operative treatment. This consists in section of the contractured muscle with simultaneous advancement of the muscle that is paralyzed (see §§ 882 and 887). By this means the deflected eye is brought into the proper position; and if the paralyzed muscle still has a certain contractile power, this muscle will be put under more favorable mechanical conditions for working. [In other cases, tenotomy of the associate is useful. See § 890.—D.]

II. Spasm of the Ocular Muscles

[**666. Symptoms.**—Spasm of any muscle of one eye will cause that eye to shoot ahead of the other when both are moving in some special direction. The symptoms of such spasm are—

1. *Excessive Movement.*—The affected eye shoots beyond the other when the eyes are carried in a certain direction, and its total range of excursion in that direction is greater than that of the fellow-eye.

2. The *deviation* thus produced, which can be made out by the screen and other tests, is greater if the sound eye is used for fixation than if the affected eye fixes,—i. e., the *primary is greater than the secondary* deviation. (Cf. remarks on p. 737.)

3. *Diplopia*, increasing as the eyes are carried in the direction of the field of action of the overacting muscle. This diplopia is precisely the same in character and in behavior as that which would be produced by a paralysis of the associate muscle in the fellow-eye,—i. e., the conditions will be as shown in the following table.

Spasm of right	Corresponds to paralysis of left	In either case			
		If L. eye fixes R. deviates greatly	If R. eye fixes L. deviates somewhat	Diplopia is	Deviation and diplopia increase as eyes are carried
External rectus...	Internal rectus...	Out....	Out....	Crossed.....	To R.
Internal rectus....	External rectus...	In.....	In.....	Homonymous.	To L.
Superior rectus....	Inferior oblique...	Up....	Down..	Right [16].....	Up and to R.
Inferior oblique...	Superior rectus...	Up....	Down..	Right [16].....	Up and to L.
Inferior rectus....	Superior oblique..	Down..	Up....	Left [17].....	Down and to R.
Superior oblique...	Inferior rectus....	Down..	Up....	Left [17].....	Down and to L.

Note.—The conditions obtaining in spasm of the left eye (paralysis of the right) may be deduced by substituting right for left and vice versa.

4. *False Projection.*—If the projection test is made in the manner indicated on page 738 by having the patient close the good eye and try to put his finger on an object situated within the field of action of the affected muscle it will generally be found that in a case of spasm the patient undershoots his mark, just as in paralyses he overshoots it. Thus, a patient with a spasm of the right abducens who is trying to touch an object on his right will put his finger to the left of it. The reason for this is that the patient underestimates the distance that the eye travels, since he is aware only of the effort that he is putting forth in order to look at the object, and with an overacting muscle this effort is less than normal.

5. An *apparent movement* of objects looked at may be produced by the false projection, and this movement together with the diplopia may cause *vertigo*.—D.]

[16] I. e., vertical diplopia with the image of the right eye below.—D.]
[17] I. e., vertical diplopia with the image of the left eye below.—D.]

[The appearances presented by a spasm of a muscle in one eye closely simulate those of a paralysis of the associate muscle in the other eye. Indeed the diagnosis between the two may be difficult, but in general can be made from the following:—

Points in Common

One eye moves faster and further than the other when both are carried in some one particular direction; and this discrepancy between the position of the eyes and also the diplopia, false projection, and vertigo become more and more pronounced, the further the eyes are carried in that direction.

SPASM.	PARALYSIS.
The absolute excursion of the faster moving eye in the given direction is greater than normal; that of the other eye is normal.	The absolute excursion of the faster moving eye is normal in the given direction; that of the other eye is subnormal.
The total excursion of the faster moving eye in the given direction and in the direction opposite is greater than normal—i. e., the field of fixation is excessively large in one of its diameters.	The total excursion of the faster moving eye is normal, that of the other eye is subnormal—i. e., the field of fixation of the latter is contracted in one of its diameters.
Fixation is usually performed by the slower moving eye.	Fixation usually performed by the faster moving eye.
The amount of deviation may show great and sudden changes from time to time.	The amount of deviation remains constant or changes slowly and progressively.
If false projection is present, it occurs when the faster moving eye is used for fixation (the other being closed), and the patient undershoots the mark he is trying to touch.	False projection occurs when the slower moving eye is used for fixation, and the patient overshoots the mark he is trying to touch.

Furthermore, if an elevator or depressor is affected the eye which by the Maddox rod or clinometer shows an abnormal torsion is the affected eye.—D.]

667. Etiology.—Over-action of an ocular muscle may occur either because the muscle itself is excessively strong and over-developed (*structural over-action*), or because the tendon is inserted so close to the cornea that it exerts an undue leverage on the eye (*insertional over-action*). A marked example of insertional over-action is that produced by the operation of advancement.—D.]

[*True spasm* is that form of over-action produced by excessive innervation. It may be either primary or secondary.

Primary spasm of the ocular muscles is rare. It may be produced by meningitis or other irritative affections of the brain, by reflex irritation from decayed teeth, by heightened pressure in the labyrinth, and by tetanus. Sometimes it occurs without known cause. The spasm produced by hysteria or occurring in connection with convulsive attacks does not usually affect the individual muscles but some conjugate movement of the eyes especially convergence (convergence-spasm, see page 781) or a parallel movement (conjugate spasm, see page 796)

Secondary spasm is common. It comprises—

1. *Secondary spasmodic deviation of the sound eye when the paretic eye fixes* (see page 744). This deviation (see Table on page 758) always appears under the guise of a spasm of the associate to the paralyzed muscle.

2. *Spasm of a synergic muscle in the same eye.* Thus in paralysis of the right superior rectus there may be a sort of compensatory spasm of the right inferior oblique. The resulting condition is quite like that produced by simultaneous paralysis of both superior recti, and differs only in that the diplopia in the left upper field fluctuates considerably and irregularly from one time to another.

So, too, in an abducens paralysis we sometimes find an associated spasm of the other abductors (the two obliques) in the same eye. This causes the eye to move by zig-zag jumps a little further out than the externus alone can carry it. If the eye is above or below the horizontal plane, the amount of additional movement thus obtained may be considerable.

3. More or less irregular and varying *spasm in the opponents of the paralyzed muscle.*

4. *Permanent spastic contraction* (*contracture*) *of the opponent* to the paralyzed muscle (see page 741). In this case the paralysis itself may disappear and the contracture remain (see page 742). When this happens the picture presented will be that of spasm, pure and simple.—D.]

[III. Heterophoria and Comitant Squint[18]

668. Kinds of Deviation.—Normally a person looking with both eyes fixates the same object with both. If he does so under all conditions—even for example, when the image in one eye is artificially made different from that of the other or is suppressed by covering one eye—he is said to have *orthophoria*. This is the ideal or natural condition.

Opposed to orthophoria are—

(A) *Heterophoria.* Both eyes fixate the same object when both are uncovered, but each eye will deviate, as soon as it is covered.

(B) *Squint* (*strabismus, heterotropia*). Only one eye fixates at a time, the other deviating even when both eyes are open.

A special variety of squint is produced by paralysis of the eye muscles. This on account of its peculiar characters has received special consideration (see page 736).

What orthophoria signifies is rendered clear by the following experiment: We cause the patient to fix with both eyes an object at a distance of 6 metres from him. Then we push a sheet of paper before one eye and watch behind the paper the eye thus covered. We shall find that the eye remains correctly adjusted for the object, although it no longer sees it. It remains steadily in the position of fixation because this is the position of equilibrium for the eye. This position is the resultant of the varying amounts of innervation which are supplied to the individual muscles and which are distributed among them in proper proportions (see page 718).

Disturbances of muscular equilibrium (*muscular imbalance*, including

[18] [In the sections that follow to the end of the chapter the matter, distinct in the former editions, contributed by the translator has been consolidated with the original in order to prevent repetition and overlapping. Owing to the way in which the material is dovetailed the use of brackets to indicate added matter would cause confusion. These, therefore, have been omitted, and it will be understood without them that for the statements contained in the ensuing sections the translator is responsible.—D.]

heterophoria and squint) are recognized by the same experiment. Suppose, for example, we are dealing with *heterophoria*. In this case, according to definition, the patient when he is looking with both eyes at the object will fixate it with both—i. e., there will be no deviation. Suppose now a screen is held before one eye. This will then deviate behind the screen in some way—say outward. When then the screen is withdrawn, the visual axis of this eye is no longer directed at the object, but the eye has an outward squint. It hence has to be brought back to the position of fixation by a movement inward (movement of adduction). Hence, on withdrawing the screen we observe a movement of the eye in a direction precisely opposite to that of its deviation behind the screen (*movement of readjustment* or *redress*). This latter movement is generally easier to make out than the deviation of the eye behind the screen, and hence is currently employed as a means of recognizing the latter. If, on the withdrawal of the screen, the eye makes a movement of redress inward, it has been deviating out behind the screen, and vice versa. The phenomena that present themselves in this experiment are accounted for as follows: In the example selected, in which the eye deviates out behind the screen, the two eyes during the act of fixation were not in muscular equilibrium, but tended to diverge. Yet, so long as vision was performed with both eyes, there was correct fixation because otherwise there would have been double vision. Now, there is a great antipathy toward double images and a correspondingly strong effort to secure single vision (fusion tendency; see page 734). Hence, an excessive convergence innervation is brought into action in order to oppose the tendency to divergence. But as soon as one eye is covered, diplopia can no longer take place; there is now no object in maintaining an excessive effort to perform convergence, and the eye consequently rolls outward. The position of equilibrium for this eye is therefore a pathological one—namely, a position of divergence to a certain amount. As soon as the screen is withdrawn again, double images make their appearance, which, however, are speedily united by the return of the deviating eye to its normal position once more.

Because of the fact that under ordinary conditions the deviation in heterophoria is not apparent, since it is compensated for by a corresponding output of innervation, the condition is called a *latent* disturbance of equilibrium (or latent deviation, or dyamic squint—Von Graefe.)

In *squint* (including paretic squint), a similar deviation occurs not only when one eye is covered, but also when both eyes are open. In squint, therefore, we are dealing with a *manifest* deviation (manifest squint).

Mixed conditions, e. g., orthophoria for distance and heterophoria for near points, heterophoria for distance and squint for near, or the reverse, often occur (§§ 682-685).

669. Directions of Deviations.—Deviations are also named according to the direction of the deviating eye. Thus:

Heterophoria.—Both eyes fix when both are uncovered, but behind screen—

Either eye turns in.....Esophoria
Either eye turns out.....Exophoria
R. eye turns up and L. down [19].....R. hyperphoria
L. eye turns up and R. down [19].....L. hyperphoria

Squint.—When both eyes are uncovered one fixes and the other turns—

In.....Esotropia (convergent squint, strabismus convergens), R. or L.[20]
Out....Exotropia (divergent squint, strabismus divergens), R. or L.[20]
Up.....Hypertropia (upward squint, strabismus sursumvergens), R. or L.[20]
Down..Hypotropia (downward squint, strabismus deorsumvergens), R. or L.[20]

Behind screen—

Either eye deviates in.....Esotropia (R. or L.)[20]
Either eye deviates out.....Exotropia (R. or L.)[20]
R. eye turns up and L. down [21]....R. hypertropia or L. hypotropia[21]
L. eye turns up and R. down [21]....L. hypertropia or R. hypotropia [21]

Squint may be either monocular or alternating. In *monocular* squint one eye, either the right or the left, always deviates and the other always fixes. In this case the squinting eye is commonly but not necessarily amblyopic. Of *alternating squint* three varieties are distinguished. In the first, both eyes have good vision but unequal refraction. For instance, one eye is far-sighted and the other near-sighted, so that the former sees clearly at a distance, the other near by, but both eyes can never see distinctly at once. In this case the far-sighted eye will fixate in looking at a distance, the near-sighted eye when looking near by, and in either event the eye not used squints; the image in this eye not being distinct, its suppression is readily accomplished. Alternating strabismus [of this sort] is usually divergent, rarely convergent. Another kind of alternating squint is found exclusively in strabismus convergens. It consists in each eye's being used for that half of the common field of fixation which is on the opposite side from it. An object which is carried from the patient's left to his right in front of the eyes is first fixed by the right eye, while the left eye squints past the object to the right, but as soon as the object has passed to the right of the middle line, the left eye, by a movement of both eyes back to the left, takes up the work of following the object the rest of the way. Thus each eye avoids the necessity of making a strong movement of abduction. The third and commonest kind of alternating squint is that in which the patient, sighting an object straight ahead of him, and either far or near, fixes with either eye indifferently and sometimes with either at will. In this case the two eyes are usually, though not always, equal in refraction and vision. This kind of alternation occurs both in convergent and in divergent strabismus, and it is probable that in their beginning very many cases of squint are alternating. In all types of alternating strabismus since the two eyes are used alternately, the sight of both remains good.

670. Types of Deviations.—We distinguish the following types:

A. Non-comitant deviations, or those which increase in amount when the eyes are carried in some definite direction of the gaze and particularly when they are carried to the right or left. These deviations have already

[19] In some anomalous cases instead of one eye's going up and the other going down, when screened, each eye goes up or each down (anophoria, catophoria).
[20] According to the eye which habitually squints.
[21] In some very rare cases each eye goes up or each down behind the screen (anotropia, catotropia)

been considered under the heads of Paralysis and Spasm of the Ocular Muscles (pages 736 and 758).

B. Comitant deviations, which do not increase in amount as the eyes are shifted from one side to the other. These include—

1. *Periodic* deviations, which increase as the eyes converge or diverge, thus being either greater for near than for far distance (directly periodic) or vice versa (inversely periodic).

2. *Continuous* deviations, which are the same in amount for distance and near.

Deviations are also classed as intermittent and constant. An *intermittent* (which should not be confounded with a periodic) deviation is one which under the same conditions and with the object at the same distance from the eyes is sometimes present and sometimes not. A *constant* deviation is one which under the same conditions is always present.

671. Vision in Squint.—As a result of one of the eyes' being in an incorrect position, a man with a squint should see double. But this is really the case only at the commencement of strabismus. The diplopia soon disappears, and afterward can be evoked only by the use of all sorts of artifices, or can not be evoked at all. This, too, is a feature distinguishing strabismus from paralysis, in which the diplopia is such an annoying symptom. A man with strabismus fails to see double, because he learns to withdraw his attention from the impression conveyed by the squinting eye; he "excludes" or "suppresses" the image with this eye. The act of *exclusion* is a psychical act; the squinting eye really does see, but the visual perceptions set up by it do not excite attention—just as many men are able in looking through a microscope or telescope with one eye, to leave the other open, and yet not see with it. As a result of this act of exclusion, a man with squint has mere monocular vision; he does not, therefore, have stereoscopic sight in the proper sense of the word.

The *visual acuity* of the squinting eye is diminished as compared with the other or sound eye (except in alternating squint). In many cases probably a certain degree of enfeeblement of sight exists even before the inception of the strabismus, and, in fact, constitutes one of the reasons for the development of the latter. The enfeeblement of sight, however, becomes greater and greater the longer the strabismus lasts, since an amblyopia ex anopsia develops on account of the exclusion of the eye from the act of vision (see page 633). This amblyopia finally reaches such a pitch that reading becomes impossible, and the sight may even be reduced to the ability to count fingers at a short distance. Such an eye has unlearned its ability to perform fixation; when the sound eye is covered the squinting eye remains rigidly fixed in its false position or wanders uncertainly or may actually squint more strongly than before.

There is no doubt but that in the beginning of strabismus there is *diplopia* quite as much as there is in paralysis of an ocular muscle. But since in convergent strabismus the development of squint takes place in childhood, we hear nothing about diplopia, and by the time that the children are old enough to give a trustworthy account, they have already learned to perform exclusion. Then diplopia can usually be produced only by artificial means—e. g., by putting colored glasses before the eye or by prisms through which the image in the squinting eye is brought nearer to the fovea centralis. But when the squint does not develop until later in life, as is the exception in convergent, but the rule in divergent strabismus, diplopia is then one of the regular symptoms. Sometimes it is so disagreeable as to furnish the main reason for the patient's visit to the physician.

While ordinarily no diplopia exists in old cases of strabismus, it is just in these cases that it is very apt to develop after tenotomy. This is accounted for as follows: The process of exclusion affects not only the squinting eye, but also in part the one that does not squint. In the squinting eye, as long as the eye was squinting inward, the image of the object that the other eye is gazing at falls on the portions of the retina situated to the inner side of the fovea (Fig. 313). This region of the retina had accordingly accustomed itself to abstract its attention from the impressions received. But this is not the case with the fovea of this eye, which does perceive the images falling on it; while, on the other hand, these latter images are excluded by the corresponding portion of the retina (lying to the inner side of the fovea) in the sound eye. This condition of things we call *regional exclusion*. If, now, after tenotomy the eyes stand nearly or quite in the proper position, the image of the object of fixation falls in both eyes on the fovea or its vicinity—that is, on portions of the retina which have not been exercised in exclusion. Hence the patient complains of diplopia. In this case the diplopia frequently does not correspond to the relative position of the eyes. There may, for instance, still be a slight degree of pathological convergence, and yet the double images may be crossed, as if the visual lines of the two eyes diverged (*paradoxical double images*). The usual explanation for this phenomenon is that the squinting eye has gradually learned to project its images exteriorly in accordance with its own faulty position. Just as in the sound eye the starting point for orientation is the fovea, in the squinting eye the starting point is that part of the retina, lying to the nasal side of the fovea, upon which fall the images of the objects that the sound eye gazes at directly; i. e., it is the spot which, when the eye was in a squinting position, was identical with the fovea of the other eye. If, then, the squinting eye is suddenly brought back to the correct position, this portion of its retina, that thus formerly corresponded to the fovea of the other eye, is carried still further inward, as in other cases the actual fovea is carried in divergence of the eyes, and now the eye projects erroneously, just as it does in a case of divergence due to paralysis (page 739). Many of the cases, however, can not be explained in this way. The diplopia following a squint operation as a general thing soon disappears, but in very rare cases it obstinately persists and becomes extremely troublesome to the patient. Paradoxical diplopia may also occur in squint without operation.

If a man with squint does see with both eyes, he still resembles a one-eyed man in this, that like him he has deficient perception of depth and deficient or wanting *stereoscopic vision*. Those who squint do not notice this defect themselves, because they have learned by practice to form from attendant circumstances conclusions as to dimensions of depth, even though they do not appreciate them directly, and hence, so far as perception of depth is concerned, are not inferior to persons who have binocular vision. To show that the perception of depth in those who squint is nevertheless inferior to that of people with binocular vision requires pretty refined tests such as examination with the stereoscope or by means of Hering's test with falling bodies (see page 728). Moreover, the vision of those who squint is distinguished from that of

one-eyed people by the greater extent of the *field of vision*. The field of vision of one-eyed persons as compared with the normal field of binocular vision is limited toward the nasal side. In default of the right eye, for example, the field of vision represented in Fig. 271 would be minus the shaded portion, *R*, upon the right side. But this would not be the case with one who squints with his right eye. The exclusion of the right eye from the visual act is done only to avoid diplopia, and hence is limited to those objects which throw their images in both eyes at once—that is, to those which are found in the portion of the visual field common to both eyes (the portion left white in Fig. 271). The case is otherwise when the object passes into the temporal portion of the visual field of the squinting eye (into the right shaded portion in Fig. 271), where it can no longer by seen by the other eye because hidden from it by the nose. Then the image of the object is not suppressed by the squinting eye. Hence, the field of binocular vision of a man with squint is about as large as that of a normal man.[22]

The temporally situated portion of the visual field, for which there is no suppression of the images, corresponds to the innermost portions of the retina of the squinting eye. This, therefore, remains in constant practice and retains relatively good sight, while the sight in the other portions of the retina keeps falling off more and more. Hence in old cases of squint, we find on covering the healthy eye that the squinting eye no longer fixates, but, on the contrary, turns even more strongly in, so as to direct toward the object the innermost section of the retina with which, relatively speaking, it sees best.

Views are by no means in accord with regard to *amblyopia ex anopsia*—many holding that there is no such thing and that the amblyopia of a squinting eye is always congenital and is the cause, not the result of the squint. On the whole, however, the testimony seems in favor of the view enunciated.

672. Tests.—Our main tests and what they show us are as follows:

I. Tests of monocular fixation, showing whether each eye by itself can perform fixation.

II. Tests of binocular fixation, showing whether the eyes always or usually perform binocular fixation or whether one deviates.

III. Tests of binocular vision, showing whether the patient has or has not simultaneous vision with the two eyes, and, if he has, whether he has binocular single vision, or else either diplopia or a tendency to diplopia, and how much tendency he has to fuse the double images.

IV. Tests of monocular movement, showing whether each eye by itself can move freely in all directions.

V. Tests of the co-ordinate movements of the two eyes and, particularly of:

(a) Parallel movements.

(b) Movements of convergence.

(c) Movements of divergence.

The best *test object* is either a small flame on a dead-black background or a black spot on a large white surface. The spot should be 3 mm. wide for the distance tests and 1 mm. wide for the near tests.

In many of our tests we measure the deviation by correcting it with *prisms*. In so doing the apex of the prism is always placed in the direction

[22] As a matter of fact it is somewhat smaller in strabismus convergens, because, owing to the convergent position of the eyes, the visual fields overlap more than usual; and for an analogous reason in strabismus divergens it is larger than usual.

in which the eye deviates, i. e., toward the temple in exophoria or divergent squint, toward the nose in esophoria or convergent squint, up before the right eye or down before the left in right hyperphoria, and in the opposite direction in left hyperphoria.

In applying prisms we must remember that the amount of deviation that they produce and hence the amount of deviation that they measure (a) vary unequally with the strength of the prism; (b) vary greatly according to the way in which the prism is held. If the prism is held in the usual position before the eyes, i. e., with the plane of its base parallel with the line of sight, the following relation holds good

Prism angle (Δ)	Actual deviation (°) (to nearest half degree)	Prism angle (Δ)	Actual deviation
1	0.5	45	29
5	2.5	50	34.5
10	5.5	52	37.5
16	9	54	40.5
20	11	56	45
25	14	58	50.5
30	18	59	56
35	21	59.2	Total reflection.
40	25		

A slight variation in the position of the stronger prisms will make a great difference in its deflection power.

673. Test of Monocular Fixation.—1. *Fixation test.*—We make the patient cover one eye and then direct his other eye sharply at some well-defined object, like a pencil or a light, held straight in front of him. Inspection then will show, whether his fixation is central or eccentric, or whether, as in cases of a high grade amblyopia, he cannot fix at all.

674. Tests of Binocular Fixation.—1. *Inspection.* We note by this any obvious deviation of the eyes, and whether this deviation is constant or intermittent, and monocular or alternating.

While ordinarily the presence of a squint is obvious from inspection, a slight strabismus may escape notice. The diagnosis, however, will be made at once from the screen test (page 767).

Just as a slight strabismus may remain unnoticed, so also it can happen that we may believe that we are dealing with a strabismus when none exists. An *apparent strabismus* of this sort is most frequently simulated in the following way: The visual axis which joins the object of fixation to the fovea centralis does not in most eyes pass through the apex of the cornea, but the latter lies somewhat to the outside, or rarely to the inside of the axis.[23] If, then, the visual axes are parallel for distant vision, the corneal apices in the first case are divergent, in the second case convergent. If this deviation attains a pretty high degree, it becomes noticeable and simulates strabismus. Here, again, the screen test leads to the correct diagnosis; for when each eye

[23] [The angle between the real and the apparent directions of the eye in this case has been variously termed angle α, angle β, angle γ and angle κ, according to the way in which it has been defined. (See page 769.)—D.]

is covered alternately it is apparent that the eye which is not covered remains steadfast in its position, and hence performs fixation properly.

With many persons it happens that during fixation the eyes are properly placed, but, when they are looking about without thinking of anything in particular, one of the eyes squints a little outward. In such cases we are generally dealing with myopes with latent divergence (exophoria). In most this condition remains the same all through life, while in some few a constant divergent squint develops from it.

2. *Screen Test.*—The patient is directed to look sharply at the test object, and then a card is placed before the right eye and passed quickly to and fro from one eye to the other.

If there is *orthophoria,* neither eye will deviate when covered, and each consequently will remain steady when the screen is removed. If, however, there is *squint* or *heterophoria* of more than a degree or two, each eye when covered will deviate, and when uncovered will turn back into the fixing position. Thus, if there is either an esophoria or convergent squint each eye in turn will deviate in or towards the nose when covered, and will swing out again when uncovered.

In squint, whether non-comitant (paralytic) or comitant, the deviation of the squinting eye behind the screen is called the *primary* deviation; that of the non-squinting eye is the *secondary* deviation. In paralytic squint the secondary deviation exceeds the primary (page 737); in comitant squint, usually, but not always it equals it.

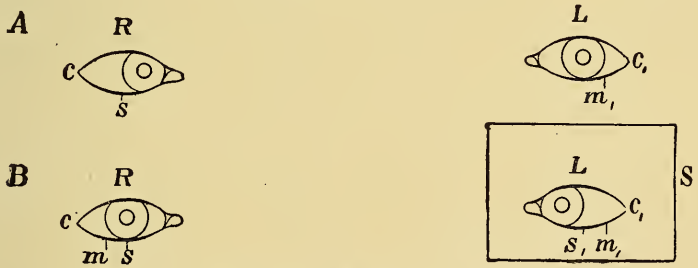


Fig. 340.—Primary and Secondary Deviation in Squint.

A, eyes uncovered. The right eye, R, squints in so that its cornea is distant *cs* from the outer canthus *C*. The left eye, *L*, is straight. Its distance from the outer canthus is c_1m_1.

B, left eye covered. This eye now deviates in by an amount s_1m_1,=the secondary deviation. The right eye is now straight, having turned out by an amount *sm*=the primary deviation.

The *amount* of the deviation can be roughly determined by inspection, but is best measured accurately by placing a prism before the eye (apex in for esophoria and convergent squint, apex out for exophoria and divergent squint, apex up or down for hyperphoria), and increasing the strength of the prism until the screen-deviation is first abolished and then converted into a movement in the opposite direction. The first prism that causes this over-correction, less 2Δ, represents the amount of the deviation.

In paralysis and ordinary squint, the prism which abolishes the movement when placed before the deviating eye, measures the primary deviation;

that which abolishes the movement when placed before the sound eye measures the secondary deviation.

Having thus ascertained that a deviation is present and how much it is, we next inquire *if it is a squint or a heterophoria.*

To determine this, we make the patient look fixedly at the test-object with both eyes open, and then alternately cover and uncover one eye,—say the right,—leaving the other all the time uncovered (*method of binocular uncovering*). By thus covering the right eye we compel the left eye to fix, if it is not already doing so, and we allow the right eye to deviate, if it has any tendency to do so or if it is not already deviating.

In this case three conditions may obtain:

(*a*) There may be *heterophoria.* In this event the left eye which is fixing already will continue fixing when the right eye is covered, and the latter which was fixing before being covered will now deviate. Then, when the cover is taken off again, the right eye will swing back into place and the left will still remain fixing,—i. e., both on applying and removing the screen, but one eye will move, and that the one that is being covered.

(*b*) There may be a *squint of the right eye.* In this case the left eye which was fixing before the right was covered will continue fixing afterward, and the right eye which was deviating before it was covered will remain deviating afterward. Then when the right eye is uncovered again, the left eye will still remain fixing (because it is the eye that habitually fixes) and the right will still remain deviating (because it is the eye that habitually deviates). That is, both on covering and uncovering, neither eye moves, provided the cover is put over the squinting eye.

(c) There may be a *squint of the left eye.* Then the left eye, which was deviating before the right eye was covered, will now have to move into place in order to fix. As it moves into place, the right eye, which is behind the screen, will move out of place. Then, when the screen is removed, the right eye, which has thus become deviated, will move back into the fixing position, since it is the right eye which ordinarily fixes when both eyes are open, and the left eye will move out of the fixing position and back to its ordinary position of deviation. That is both on covering and uncovering, both eyes will move, provided in the case of a squint the screen is put before the fixing eye.

If repeated applications of the test show that sometimes the right eye squints, sometimes the left, then we are evidently dealing with an *alternating strabismus.*

3. *Perimeter* and similar tests (Priestley Smith's tape test, Hirschberg's test). In *Hirschberg's test* the patient is made to look at a lighted candle placed one foot in front of his eyes. The observer stationing himself behind the candle notes the position of its reflection in the cornea of the squinting eye. If it is at the margin of the cornea

we assume that there is a squint of six mm. (i. e., about 45° of arc); if it is half way between the centre of the cornea and the margin it is three mm., etc.

In the *perimeter* test, the squinting eye (L, Fig. 341) is placed at the center of a perimetric arc, and the good eye, *R*, is directed to look at a light, *G*, twenty feet distant and in line with the squinting eye and the zero point, *o*, of the arc. The observer carries another light, *K*, along the arc of the perimeter (keeping his own eye *A*, right behind the light), and watches the reflection of the latter as it sweeps across the cornea of the squinting eye. When the reflection occupies the center of the cornea the position of the light on the perimetric arc indicates the angle of squint.

Similar in principle is *Priestley Smith's tape method*, except that the good eye is made to follow an object along a tangent scale, until the reflection of an ophthalmoscopic mirror, through the peephole of which the observer is looking occupies the center of the cornea of the squinting eye.

In all these methods allowance must be made for the amount of the angle gamma.[24]

No one of these methods at all approaches the screen test either in accuracy or in convenience. They do not show, or at least they show very imperfectly, the character of the squint (whether alternating or constant, periodic or continuous). Nor can they measure a vertical and a lateral deviation simultaneously, as the screen test does. Nor can they measure well, as it does, low degrees of squint, and they cannot measure heterophoria at all. Nor can they show, as it does, the varying amount of the deviation in different directions of the gaze.

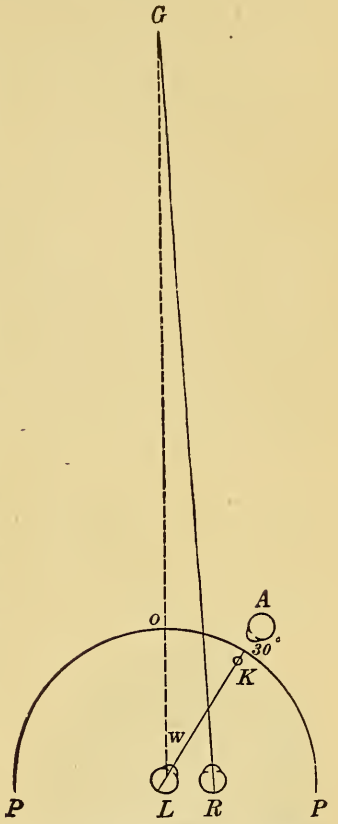


Fig. 341.—Measurement of Strabismic Angle with a Perimeter.

675. Tests of Binocular Vision.—1. *Parallax Test.*—As we are passing the card from eye to eye in making the screen test, we ask the patient whether the object he is looking at seems to move or not. If it does, it means that he actually sees the object double, only instead of seeing the two images simultaneously he sees one after the other. Thus, if on uncovering the right eye the object seems to move to the right it means that the right-eye image is to the right of the left—i. e., he has homonymous diplopia (esophoria). If, under the same conditions, the object had seemed to move to the left he would really have crossed diplopia (exophoria). If it had moved down he would have had a right hyperphoria; if up, a left hyperphoria.

In order to be sure that the patient is actually fixing the object and not

[24] The angle γ is determined sufficiently well by placing the eye at the center of a perimetric arc and making it fix the zero point of the perimetric scale. A light is then carried along the arc until its reflection occupies the center of the cornea. The position of the light on the perimetric arc shows the amount of the angle gamma.

looking beyond it, so that he sees it projected against a background more remote, the test object should be on the same plane as the objects immediately surrounding it.

The *amount* of the parallax is measured accurately by the prism which, placed before the eyes, will cause its abolition. Thus, an homonymous parallax which is completely abolished by a prism of 15Δ base out, indicates an esophoria of 15Δ, etc.

The test is very precise, deflections of a quarter of a degree being measurable in this way. It is also accurate. It has further the advantage that vertical and lateral deflections, simultaneously present, can be simultaneously measured by a combined lateral and vertical prism. But its greatest value lies in the fact that it is a subjective test which is made at the same time and under the same conditions as the objective screen test. If there is any real discrepancy between the subjective projection of images and the objective position of the eyes—in other words, if there is a faulty projection—it ought to show in a notable and constant difference between the findings of the two associated tests, the parallax and the screen. This, indeed, is occasionally, but only occasionally, the case.

2. *Red Glass.*—If there is much tendency to diplopia the mere interposition of a red glass before one eye will evoke it. In this case the test object should be a light, of which, then, two images will be seen—one red, the other white. This proves that the patient has *binocular vision with diplopia.* If, with the red glass before one eye, the patient sees a single light which is white tinged with red, he surely has *no diplopia, and has binocular single vision.* If he sees a single light which is pure white or pure red he is suppressing the image in one eye (see page 763). In this case he has *no diplopia and has only monocular vision.*

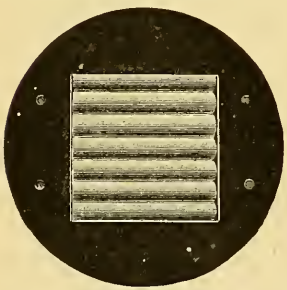


Fig. 342.—Maddox Rod. Instead of a single thick glass rod there is now used a series of thin rods usually made of red glass, placed side by side in a frame. If the rods are held horizontally the red line of light produced by them is vertical and vice versa.

The *amount* of deflection, and hence the amount of deviation, may be determined either by ascertaining the linear distance between the double images (allowance being made for the distance of the object from the patient), or by finding out what prism will be required to overcome the diplopia.

3. *Maddox Rod.*—The Maddox rod consists of one or more little cylinders of glass set in a frame. (See Fig. 342.) If the instrument is held as shown in the figure and placed before the right eye, the patient, looking at the light, will see with this eye the light transformed into a luminous vertical streak. This streak is so different in appearance from the flame that the patient usually has no tendency to fuse it with the latter as he fuses ordinary double images. He hence allows a deviating eye to assume its natural position, as it would do behind the screen. If he has neither esophoria nor exophoria the streak will appear to run through the flame which

in its unaltered form he sees with the left eye. If he has esophoria it will appear to the right of the flame; if he has exophoria, to the left of it.

To ascertain whether hyperphoria is present or not, the rod is turned at right angles to the position shown in the cut and again placed before the right eye. If the patient has no hyperphoria, he will see a horizontal line bisecting the light. If, however, he has right hyperphoria the horizontal line will appear below the light, and if he has left hyperphoria the line will appear above the light.

The *amount of deviation* may be measured by the linear distance of the streak from the light or by the amount of prism that will be required in order to make the streak coincide with the light.

The Maddox rod can also be used to indicate *whether there is any torsion* of the eye—i. e., any tilting of its vertical meridian to the right or left. If there is, the streak of light will appear tilted when the rod is set vertical, and the number of degrees through which rod has to be rotated to make the line of light appear vertical, will indicate the amount of the torsion. Torsion can also be measured by Stevens's clinometer.

4. *Phorometer.*—The basis of this is *Von Graefe's equilibrium test.*

This starts from the fact that a disturbance of equilibrium becomes manifest as soon as we render binocular single vision impossible. To accomplish this, we place before one eye a prism with its base down or up and of such strength that it can not be overcome by an effort of the eyes acting to place them on different levels (see page 734). For example, we place the prism, *P* (Fig. 343 A), whose refracting angle amounts to 10°, with its base down before the left eye, and tell the person under examination to fix his gaze upon an object, *o*. The left eye now sees the object, *o*, not at its proper place, but at o_1; and hence with both eyes together there are seen double images on different levels. If there is muscular equilibrium, so that the eyes converge properly at *o*, the two images stand vertically over one another (*L* and *R*, Fig. 343 B). But if there is a disturbance of equilibrium, and consequently an excessive or a deficient convergence, a lateral separation of the images is superadded to the difference of level. In fact, the effort to compensate for the disturbance of equilibrium by appropriate innervation now disappears, since the double images could not be seen as one in any case on account of the difference of level. Suppose, for instance, that there is an exophoria. Then the left eye deviates out behind the prism. The point *o* consequently casts an image to the outer side (left) of the fovea, and is therefore seen too far to the right (crossed double images, Fig. 314). Hence, the upper dot which belongs to the left eye no longer stands vertically above the lower, but to the right of it (Fig. 343 C). If now a second prism, the base of which looks inward, is placed before the prism which has its base down, the rays coming from the dot are deflected by this second prism

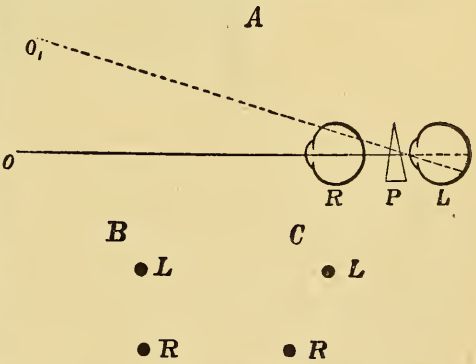
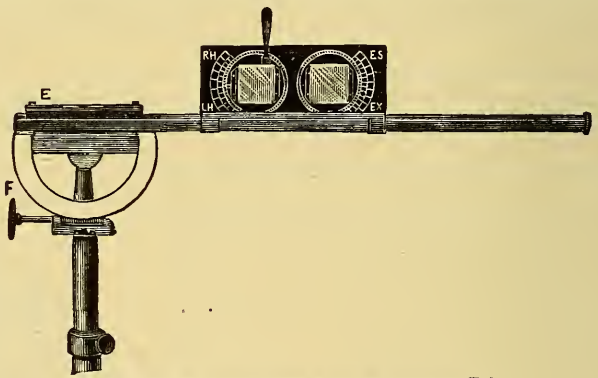


Fig. 343.—Von Graefe's Equilibrium Test.

inward toward the vertical meridian of the retina, and the upper image moves to a point more nearly above the other. By trying stronger and stronger prisms we can at length find one which brings the two images exactly over each other in a vertical line. This corrects the lateral deviation of the eyes, and hence gives the numerical expression for the heterophoria.

The phorometer (Fig. 344) consists essentially of two 6Δ prisms, geared so that they can be rotated simultaneously but in opposite directions. The prisms are first set so that their bases both look inward; the patient then looking through the prisms will see two images of the test object, that of the right eye being on the right side. If he has no hyperphoria the images will be on the same level. If he has right hyperphoria the right image will be lower; if he has left hyperphoria it will be higher. In case the images are not on a level the prisms are rotated until they are; then the amount of hyperphoria can be read off from the index.

To measure for lateral deviations the prisms are rotated until the right



[Fig. 344.—Stevens's Phorometer.—D.]

hand one points down, the left up. Then, if there is no esophoria or exophoria, the images will appear directly over each other. If, however, there is esophoria the upper image will appear on the left; if there is exophoria it will appear on the right. In either of the latter events the prisms are rotated until the images do stand over each other, when the amount of the esophoria or exophoria, as the case may be, can be read off from the index.

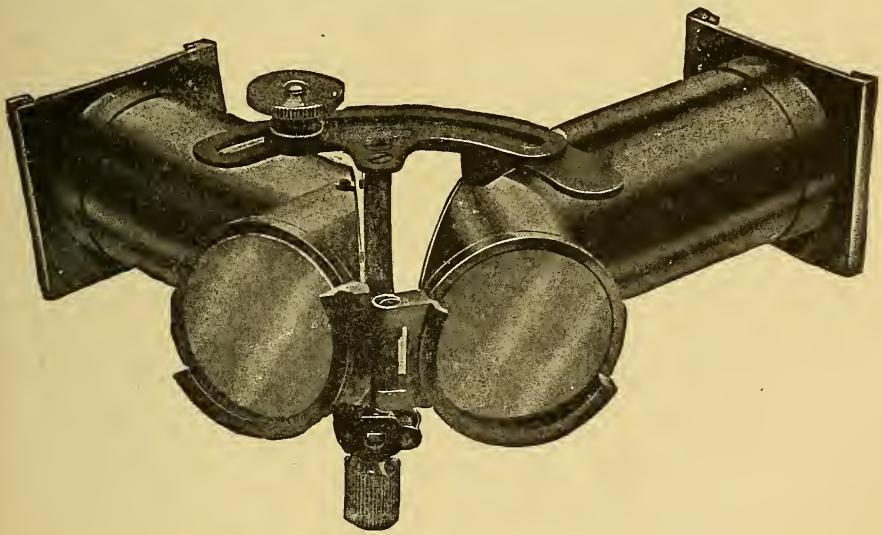
5. *Bar-reading.*—If the patient is directed to read and holds a pencil some three inches in front of the page, between it and his eyes, he will still if he has binocular vision, be able to read every word, because the letters that are hidden from the left eye will be seen by the right, and vice versa. If he has no binocular vision, the pencil obviously will cut out part of what he is reading. Hence, this simple test enables us to tell whether he is using one or both eyes.

6. *Stereoscope and Amblyoscope.*—A patient who has binocular vision will be able to see two dissimilar images placed in the stereoscope and to fuse them into a single image. This is impossible for one who has only monoc-

ular vision. Different forms of stereoscope have been devised, both for determining the presence or absence of binocular vision and also for training binocular vision and the fusion faculty. The same object is accomplished with the amblyoscope (Fig. 345).

The stereoscope and especially the amblyoscope will show both the patient's ability to perform fusion and to secure stereoscopic vision (stereopsis). In some cases, especially of long-standing squint, both fusion ability and stereopsis are entirely absent. In others they are present to a varying degree, and then can often be cultivated—the more so, the younger the patient. By such cultivation, we may secure a cure in some cases of squint, and in others may make a cure by operation more likely.

Another test (7) of stereoscopic vision is *Hering's test* (see page 728).



[Fig. 345.—Worth's Amblyoscope, Modified by Black.

The amblyoscope consists of two tubes, bent and joined so as to admit of being converged and diverged through a considerable angle. In the Black modification here shown, one of the tubes can also be raised or lowered, an arrangement advantageous when the two eyes are not on the same level. A graduated arc may also be added, which shows the amount of the deviation or of the convergence and divergence employed. The further end of each tube carries a picture mounted on a translucent plate of celluloid. A mirror at the bent portion of the tube enables the patient looking through the eyepiece to see the picture with the corresponding eye. If he has binocular vision he will see two pictures, one with each eye, and by swinging the tubes in or out he will get these two pictures together and fuse them. By the use of simple devices the image formed by the better eye can be dimmed so that there is less tendency to suppression by the other.—D.]

All the tests given in this section are dependent for their success on the presence of at least a certain amount of binocular vision. When this is absent, as is the case in *suppression* (see page 763), these tests cannot be applied. This is true of most cases of comitant squint. We are then thrown back on the other tests, particularly the screen test. But in many cases of comitant squint the suppression is only partial, and in these some of the tests of binocular vision, particularly the amblyoscope, are applicable.

676. Tests of Monocular Movement.—These tests are made to determine whether either eye by itself can move properly—irrespective of whether

it can keep pace with its fellow or not. By determining the limits of movement in every direction we delimit what is called the *field of monocular fixation.*

The limits of the monocular excursions in each meridian, i. e., the limits of the field of fixation are variously given by different observers and probably vary within somewhat widely even in normal subjects. According to Landolt, with whom the translator is in substantial agreement, the field of fixation extends not less than 47° in all directions, except above, where it is generally somewhat less. Outward and inward it is usually somewhat more than this (about 50°), and below it often extends to 55°. Permanent restriction within 47° in any direction except above must be regarded as probably pathological (Landolt). Quite certainly pathological would be a rotation upward of less than 40°, one downward of less than 50°, and one in or out of less than 45°.

The tests are:

1. *Inspection.* The eye is made to follow a well defined object and the observer notes when the eye begins to flag or waver. This, though a rough test, is sufficiently accurate in most cases.

2. Measurement of the excursion on a *tangent scale* or a *perimeter.* Here the patient is made to follow a sharply defined object (e. g., two closely set parallel lines on a bit of cardboard) which blurs the moment fixation wavers. The object is carried out from the center, the eye following it. The exact point at which the object blurs is noted, and the corresponding amount of angular rotation read off from the scale.

3. Stevens's *tropometer.*

In measurements made by any of these methods it is absolutely essential that the head should be fixed, and the eye alone move.

It is also important in most cases that measurements be taken in each one of the six cardinal directions of the gaze.

Another monocular test that indicates whether an eye performs rotation properly or not is:

4. The *projection test* (page 738). For, if the patient, when looking with but one eye projects faultily—i. e., if he overshoots or undershoots the mark—as he makes the attempt to point at an object lying to one side or the other, he is evidently trying to move the eye with a muscle that is abnormally innervated. If he overshoots the mark, the muscle that moves the eye in that direction is too weak, if he undershoots it, the muscle is too strong.

One other monocular test is:

5. The *clinometer* or Maddox rod. While these may be employed to test torsion in binocular vision (see page 771), they may also be employed to show deflections of the corneal meridian in one eye even when the other is covered or blind. Occasionally in doubtful cases we can by thus proving the presence of torsion in one eye show that this eye is the one affected with paralysis or spasm (cf. page 759).

In general, the tests of monocular movement (tropometer, etc.) are of subordinate importance; first, because they do not show what we mainly wish to know, i. e., how the two eyes work together; and, second, because they are very variable and often fail to

reveal an anomaly that is present. I have repeatedly seen cases in which one eye, when tested by itself, apparently moved well in all directions and yet showed a considerable and increasing deflection when the attempt was made to move it in conjunction with its fellow. Again, when we examine the eye in different days we may find wide variations in the range of its excursion, showing that the patient sometimes puts forth his full strength, sometimes does not. In binocular movements whether he puts forth his full strength or not is of little importance, since if he fails with one eye he will equally fail with the other, and any disparity in movement between the two will still be apparent.

677. Tests of Binocular Movement.—These comprise—

A. *Tests of Conjugate (Parallel) Movement of the Two Eyes.*—An essential requirement in making these tests is that the eyes shall be carried in each of the six cardinal directions of the gaze (see page 720). The tests comprise—

1. *Excursion test* (see page 744).
2. *Comitance test* (see page 745).
3. Determination of the *screen deviation* in the six cardinal directions (page 745).

These three tests are fixation tests determining the binocular excursions, the field of binocular fixation, and the deviation in different directions of the gaze.

They are independent of binocular vision and can therefore be employed when there is suppression. The subjective counterpart of these objective tests, but which can be employed only when there is binocular vision, is the determination of

4. The *field of double vision* with the tangent plane (see page 746).

B. *Tests of Convergence.*—These comprise:

1. A comparison of the deviation for *distance* and that for *near*, as determined by the screen, parallax, and phorometer. A deviation which increases notably in near vision, i. e., a directly periodic deviation, argues in general an anomaly of convergence.

This comparison is of the utmost importance and should never be neglected in the routine examination of a case of motor anomaly.

2. *Convergence near-point.*—This is determined by carrying a fine object up to the eyes and urging the patient to converge on it as much as possible. The normal convergence near-point should not be over 5.5 cm. from the eyes except in presbyopia when it may be 8 or even 9 cm. A near point, persistently remoter than this, means a convergence-insufficiency or an actual divergent squint.

In certain cases of divergent squint in which the patient can never actually converge the eyes so as to secure binocular fixation, he will yet make the effort to converge

and may do so down to a comparatively near point, the eyes visibly coming together, although not perfectly, until the object is brought to perhaps four or five inches from the eyes, when the latter suddenly give up the effort and diverge widely. The patient is then said to have a *relative* though not an absolute near point of convergence.

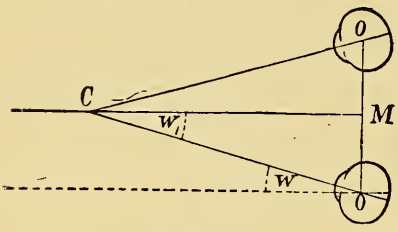


Fig. 346.

It is best to measure the near point of convergence (*C*, Fig. 346) from the line *oo* connecting the centers of rotation of the two eyes, i. e., measure the distance *CM*. This is done by measuring the distance of *C* from a plane (spectacle frame) placed 11.5 mm. in front of the eyes. If we add 25 mm. to this measurement we shall have the distance *CM*. Using the abbreviation *Pc* (Maddox) to denote the near point of convergence, *PcB* may be used to indicate the distance of *Pc* from the base line.

We may wish to determine the actual angle that the visual axes, *OC* make in converging on an object *C*, whether that is at the near point of convergence or not. This is readily ascertained. If we determine by the method given on page 76 the distance *oo*, between the centers of rotation of the two eyes, and measure *CM* by the method given above, then for all distances of *C* from 5 to 22 cm. the relation

$$oCo = \frac{OO \times 50}{CM} + 3^0$$

will hold good (*oo* and *CM* being measured in mm.). Thus if we find *oo* to be 60 mm. and $CM = 120$ mm., then $oCo = \frac{60 \times 50}{120} + 3 = 28°$. This is evidently double the angle W^1, by which each eye is converging, i. e., double the convergence-adduction of that eye (see page 722).

3. *Prism-convergence* is determined by placing before the eyes successively stronger prisms, base out, until the patient can no longer unite the double images.

It is usually determined for distance only, although it may also be determined for near points, especially when we are trying to ascertain the effect of prism exercise in relieving an exophoria.

The prism convergence can usually be run up to 50Δ by practice. In the first trials the results vary greatly and the variations are evidently not due to any real variations in the muscular power. Hence the test is of significance only when on repeated trials the patient can not be made to overcome more than 12 or at most 15Δ and when even that small degree of convergence is made with effort and maintained with difficulty. In such cases the convergence near-point pretty nearly always will be found to be remote.

4. Ability to *overcome crossed diplopia*, however caused, or ability to maintain fusion with the *amblyoscope* when the tubes are converged.

C. *Tests of Divergence.*—These comprise:

1. Comparison of the *deviation found for distance* and for *near points*. A deviation, which, as determined by the screen, par-

allax, or phorometer, is essentially greater for distance than for near, i. e., one which is inversely periodic, argues a divergence anomaly or possibly an anomaly of the external recti.

2. *Prism-divergence* is the ability to overcome prisms, base in, while looking at a distant object. This ability, which varies between 3 and 8 (usually between 4 and 7) Δ is a quantity, usually constant on repeated trials and seems to be a true measure of the diverging power. It is an important test.

3 Ability to *overcome homonymous diplopia*, however caused, or ability to maintain fusion with the *amblyoscope* when the tubes are diverged.

678. How the Tests are made in Practice.—Usually not all the tests need be made, some being reserved for cases of special complexity. The following very complete routine suffices for all but the most unusual cases.

A. We first determine the *relations for distance* by placing the test object far off,—if possible, twenty feet or more,—from the eyes and then measuring the amount of deviation, vertical and lateral, by

(1) The *screen* and (2) the *parallax* simultaneously. By the screen we also determine whether the deviation is a squint (monocular or alternating) or a heterophoria (see page 768).

(3) The *Maddox rod*, or (4) the *phorometer*.

B. We measure the *deviation for near points* (with the test object held at about 25 cm.) by

(5) the *screen* and (6) the *parallax* simultaneously and may supplement this with (7) the *phorometer*, though this is rarely necessary. In making the screen test, we ascertain by it whether the deviation is a squint or a heterophoria.

C. We determine:

(8) The *convergence near-point.*

(9) The *prism-divergence* (best done just after test 4).

(10) The *prism-convergence* (may be omitted if 8 is normal).

(11) The *excursion test.*

(12) The *field of binocular single vision.*

(13) The *comitance* test, supplemented if necessary by (14) the *screen deviation* in the six cardinal directions of the gaze.

679. What the Tests Show in Normal Cases.—The eye muscles may be regarded as normal if

(*a*) The screen test shows that there is no squint, and the other tests enumerated under A and B show that there is only a minimal amount of heterophoria (less than 2Δ of esophoria or exophoria and less than ½Δ of hyperphoria) for distance and 0–3Δ of exophoria for near.

(*b*) The convergence near-point is less than 7 cm. from the base line connecting the centers of the two eyes.

(*c*) Prism-divergence is not less than 4Δ and not over 8Δ.

(*d*) Prism-convergence after a few trials can be run up to 15Δ or 20Δ or more.

(*e*) The excursions of the eyes are normal in extent (see page 774) and comitant and the field of binocular single vision is normal—i. e., the patient having a red glass before one eye and following a light that is carried in all directions sees everywhere a single parti-colored light, each eye moves equally with its fellow without lagging, and there is no noticeable screen deviation anywhere.

In certain cases the diplopia tests (parallax, Maddox rod, phorometer) do not agree with the screen test, so that we have, for example, a well-marked divergent squint with an homonymous diplopia, etc. This is due to a peculiar kind of *false projection*, acquired usually in very early life. It sometimes causes a troublesome anomalous diplopia which develops after a squint operation (see page 764).

If there is suppression of the image in one eye, such as regularly takes place in long-established squint (see page 763), all the *tests for binocular vision* (parallax, phorometer, red glass, stereoscope, amblyoscope) will fail. Per contra, the failure of these tests in any case argues the existence of suppression. In this event we must fall back on the screen test, and the determination of the field of monocular and binocular fixation and of the convergence near-point.

680. Etiological Classification of Deviations.—A deviation of the eyes may be caused by—

A. An anomaly of structure, insertion, or innervation of one or more of the *ocular muscles* (muscular paralysis and spasm, using these terms in their widest sense).

B. Anomalies of the *co-ordinate movements of the eyes* (the muscles per se being normal). These include—

1. Anomalies of *convergence* and *divergence*, either in the sense of excess or insufficiency.

2. Anomalies of *parallel movements* comprising conjugate paralysis and spasm (see page 796) and nystagmus (see page 798).

681. Differential Diagnosis of Deviations.—The diagnosis of a *paralysis* or insufficiency of the individual muscles is made from the principles laid down in pages 743 et seq. The tests used are the screen test (primary and secondary deviations), and the determination of the field of binocular fixation and of binocular single vision; sometimes also the field of monocular fixation (see page 774).

These deviations may vary all the way from a very slight insufficiency to most pronounced paralysis. Their characteristic feature is that the deviation is *non-comitant*.

The diagnosis of a *divergence* or *convergence anomaly* is based on the following considerations:

When we look to the right we converge or diverge our eyes with the same facility as when we look to the left, and with nearly the same facility as when we look straight ahead. The deviation produced by a convergence or divergence anomaly, therefore, will remain about the same, no matter how the eyes are turned, provided that the object of fixation is kept at the same distance from the eyes. But since generally we use our convergence strongly when looking at near objects and do not use it when looking at distance, the effect of a convergence anomaly in producing deviations will be much greater in near than in distant vision. The opposite will be the case with a divergence anomaly. Convergence and divergence anomalies then are comitant in that the deviation changes but little as the eyes are carried to the right or left, and are periodic in that the deviation due to a convergence anomaly increases and that due to a divergence anomaly decreases, as the object of fixation approaches the eyes.

Nearly all cases of periodic comitant heterophoria and squint are produced by an anomaly of convergence or divergence. Nearly all cases of continuous comitant squint are produced by an anomaly of convergence combined with one of divergence.

Convergence-excess will produce an active, and divergence-insufficiency a passive, convergence of the visual lines (esophoria or esotropia). Convergence-insufficiency and divergence-excess will produce exophoria and exotropia. Ordinary comitant convergent squint (esotropia) is due to a convergence-excess combined with a divergence-insufficiency, and ordinary divergent squint (exotropia) to divergence-excess combined with convergence-insufficiency.

682. Esophoria and Convergent Squint.—The varieties of esophoria and convergent squint are:

A. Non-comitant (Paretic) Esophoria.—The characteristic mark is that the esophoria increases progressively as the eyes are carried either to th right or to the left. It is due to insufficiency of an abductor (externus or an oblique) or over-action of an adductor (internus, or superior or inferior rectus). May also be due to insufficiency of an adductor, if combined with a comitant esophoria (see below) great enough to overcome the exophoria due to the insufficiency. Thus an esophoria increasing to the right may be caused by insufficiency of the right internus (which would produce an exophoria diminishing to the right) combined with an esophoria of constant amount due to a convergence-excess.

In any case an esophoria which increases to the right must be due to insufficiency of some muscle of the right eye (or over-action of some muscle of the left). If it increases *fast* to the right, it must be due to insufficiency of the right externus. (See page 750 Note.)

B. Comitant Esophoria.—The varieties of comitant esophoria and convergent squint and the characters by which they are distinguished are as follows:

	Periodic Deviations.		Continuous Deviations.
	Convergence-excess.	Divergence-insufficiency.	Convergence-excess with divergence insufficiency.
For distance all tests (screen, parallax, Maddox rod, phorometer) show	Orthophoria or slight esophoria.	Esophoria marked.	Very marked esophoria or a marked convergent squint at all ranges (somewhat greater for near than for distance if the convergence-excess was the primary condition, and greater for distance than for near. if the divergence-insufficiency was the primary condition).
For near all tests show	Esophoria marked (much more than for distance).	Esophoria slight or none (decidedly less than for distance).	
Convergence near-point.	Normal (or abnormally close).	Normal.	Normal or excessively close.
Prism-convergence (adduction).	Normal.	Normal.	If it can be elicited (i.e., if there is no suppression), normal or supernormal.
Prism-divergence (abduction).	Normal.	Low (less than 2Δ-3Δ) or negative (i.e., insuperable homonymous diplopia for distance).	Low. Usually (in case of squint, always) negative (i.e., there is homonymous diplopia at all ranges, so that no prism, base in, is overcome, or there is monocular vision with suppression).
Primary and secondary screen deviation	In all types deviation of one eye behind the screen, measured with prisms, equals precisely that of the other eye (distinction from non-comitant esophoria).		
Field of monocular and binocular fixation.	In all types excursions of each eye normal in all directions. Neither eye lags behind the other in making lateral, vertical, or oblique movements (distinction from non-comitant esophoria). Screen deviation comitant.		
Field of binocular single vision.	Homonymous diplopia if present, not increasing (if anything, diminishing) as eyes are carried to right and left (distinction from non-comitant esophoria). Increases somewhat in looking down, diminishes in looking up.		

Convergence-excess is usually accommodative, i. e., the patient in trying to see well forces his accommodation and hence also forces his convergence. This happens (*a*) when he is trying to overcome a hyperopia or astigmatism; (*b*) when he is trying to see with the aid of an accommodation which has been reduced below normal by debilitating disease or by the instillation of homatropine or atropine; (*c*) when vision is rendered difficult by opacities in the media, reduced illumination, etc. Convergence-excess, sometimes permanent, may also be set up by the use of prisms, base out. As a secondary affection, convergence-excess occurs in consequence of a divergence-insufficiency which has lasted a long time.

Donders was the first to point out the relation between hyperopia and the convergence-excess that leads to the ordinary type of esotropia. He explained this relation in the following way: Hypermetropes have to make an unusually strong effort of accommodation to see distinctly; but as this effort, on account of the connection between accommodation and convergence, is possible only when combined with a strong impulse toward convergence, the latter function acquires a preponderance and a latent convergence (esophoria) develops. From this in turn a squint develops, if there are additional factors present that act to reduce the visual power of one eye (see page 788).

In what way accommodative effort leads to convergence-excess and squint is very beautifully demonstrated by the following experiment likewise propounded by Donders: We cause a man who has perfect muscular equilibrium of the eyes to fixate a near object. If, now, we cover one eye, it remains fixed in the correct position behind the screen. But if we place a concave glass before the uncovered eye, so that the person under examination has to accommodate quite strongly in order to see the object distinctly, the covered eye at once turns inward—i.e., an artificial convergent strabismus is thus produced owing to the increased effort of accommodation.

The rare *non-accommodative convergence-excess* may be due to hysteria or to some of the irritative conditions producing spasm in general (see page 759). But often the cause remains unknown. Both accommodative and non-accommodative convergence-excess may appear under the guise of a tonic, perhaps suddenly developing and suddenly varying *convergence-spasm*. Extreme cases of this sort are produced mainly by hysteria. In these at every attempt to perform fixation of an object, either far or near, both eyes at once assume a position of extreme convergence, the pupils at the same time being greatly contracted, and the accommodation thrown into a state of spasm. In other cases, the spasm occurs only at times and principally in fixing on a near object, especially one on which the patient is directed to converge as it is brought closer and closer to his eyes.

Divergence-insufficiency is usually secondary to a progressive convergence-excess (see page 782) although it may be primary. The most marked instance of the latter is the rather rare *divergence-paralysis*. This is characterized by homonymous diplopia and a convergent squint which, great in amount for distance, diminish steadily as the object of fixation is brought toward the eyes, until at some comparatively near point there is single vision with orthophoria. Contrary to what takes place in a paralysis of the abducens, which this condition superficially resembles, the squint and the diplopia diminish or remain the same as the eyes are carried to the right and left, and the excursions of both eyes are of normal extent. The condition usually develops suddenly and is often due to organic brain disease.

683. An esophoria may disappear if the conditions causing it are abolished or off-set. Thus an accommodative convergence-excess usually disappears if the causal hyperopia and astigmatism are corrected early enough; and it disappears in presbyopes who no longer accommodate excessively because they can not. Again, in some cases a convergent squint disappears in youth, because in the growth of the face the orbits get more divergent so that the interni work with more difficulty than before (Weiss).

In other cases an esophoria increases. This is seen in the *development of a convergent squint*, which usually begins as an accommodative convergence-excess. The steps in this development are as follows:

(1) A child with a good deal of hyperopia or astigmatism, as soon as he begins to use the eyes much for near work, develops a varying, evidently more or less *spasmodic esophoria*, (convergence-spasm see supra) which is marked for near when his convergence is called into play, but which is little or not at all marked for distance.

(2) Little by little the deviation increases until binocular fixation for near becomes impossible, and the periodic esophoria is transformed into a

periodic convergent squint. This at first is intermittent and occasional, afterwards more and more constant. There is now more or less esophoria for distance also, although always less than for near, and there is still binocular fixation for distance.

Careful tests at this stage will show in nearly every case that *the squint is associated with diplopia.*

(3) After this condition has lasted for a long time, perhaps for some years, the patient gives up binocular fixation for distance, so that he now has a convergent squint for both distance and near (*continuous squint*). The deviation as measured by the screen, however, is still much more marked for near than for distance. Diplopia can still usually be elicited by the tests. The condition is still mainly one of convergence-excess.

(4) Later still, the amount of deviation for distance begins to increase until ultimately the squint for distance equals that for near points, and both are absolutely greater than they were. This change is due to the superadding of a divergence-insufficiency to the primitive convergence-excess; the conditions now, in fact, are those shown in the table as obtaining for *a convergence-excess and divergence-insufficiency combined.* The rotations of the eyes are still normal in amount, indicating that the muscles themselves are as yet unaffected.

At this stage, diplopia, if it can be elicited at all, no longer obtrudes itself upon the patient's notice nor causes confusion. In many cases it cannot be elicited by any tests, suppression of one of the images having occurred (see page 763).

(5) Later, *secondary muscular changes* (contracture of the internus, stretching and weakening of the externus) take place in the squinting eye, producing an absolute reduction in the rotation outward and excess of rotation inward.

In some cases it seems that a convergent *squint may develop from a divergence-insufficiency.* The steps in this case are, first, an increase of the esophoria for distance, then the superadding of a convergence-excess producing an esophoria for near. The result is, first a periodic convergent squint (esotropia for distance, esophoria with binocular fixation for near); later a continuous squint for all ranges. Until late in their course such cases are distinguished from those starting with a primitive convergence-excess by the fact that the deviation for distance always exceeds that for near

684. Exophoria and Divergent Squint.—The varieties of exophoria and divergent squint are:

A. Non-comitant (Paretic) Exophoria.—The characteristic mark is that the exophoria increases progressively as the eyes are carried either to the right or to the left. It is due to insufficiency of an adductor (internus or superior or inferior rectus) or over-action of an abductor (externus or an oblique). May also be due to insufficiency of an abductor if combined with a comitant

exophoria (see below) great enough to overcome the esophoria due to the insufficiency. Thus an exophoria increasing to the right may be caused by insufficiency of the left externus (which would produce an esophoria diminishing to the right) combined with an exophoria of constant amount due to a convergence-insufficiency.

In any case, an exophoria which increases to the right must be due to insufficiency of some muscle of the left eye (or over-action of some muscle of the right). If it increases fast to the right it must be due to insufficiency of the left internus. (See page 750 Note.)

B. Comitant Exophoria.—The varieties of comitant exophoria and divergent squint and the characters by which they are distinguished are as follows:

	Periodic Deviations.		Continuous Deviations.
	Convergence-insufficiency.	Divergence-excess.	Convergence-insufficiency with divergence-excess.
For distance all tests (screen, parallax, Maddox rod, phorometer) show	Orthophoria or slight exophoria.	Exophoria marked.	Marked exophoria or marked divergent squint at all ranges (somewhat greater for near than for distance if the convergence-insufficiency was the primary condition, and greater for distance than for near if the divergence-excess was the primary condition).
For near all tests show	Exophoria very marked (much more than for distance).	Exophoria decidedly less than for distance.	
Convergence near-point.	Remote (more than 9 cm. from base line connecting centers of eyes.	Normal.	Remote. In case of actual squint, none (patient may try to converge but cannot secure binocular fixation at any distance).
Prism-convergence (adduction).	Low and accomplished only with effort and difficulty.	Normal.	Low. In case of actual squint negative (i.e., there is either crossed diplopia at all ranges so that no prism is overcome, or there is monocular vision with suppression).
Prism-divergence (abduction).	Normal.	Abnormally great (over 9Δ).	If it can be elicited (i.e., if there is no suppression) it is much greater than normal.
Primary and secondary screen deviation.	In all types deviation of one eye behind the screen, equals precisely that of the other eye (distinction from non-comitant exophoria).		
Field of binocular fixation.	In all types excursions of each eye normal in all directions. Neither eye lags behind the other in making lateral, vertical, or oblique movements (distinction from non-comitant exophoria). Screen deviation comitant.		
Field of binocular single vision.	Crossed diplopia not increasing as eyes are carried to right and left (distinction from non-comitant exophoria). Increases somewhat in looking up, diminishes in looking down.		

Convergence-insufficiency is often *accommodative*, that is, it occurs in those who do not require to use their accommodation in the normal way. The accommodation and convergence being intimately associated, the disuse of one means the disuse of the other. A myope, for example, to see objects near by, needs to use either little or no accommodation; consequently with him the impulse to convergence is too weak. This disuse of the convergence often shows itself at first simply by the presence of a large amount of exophoria for near without any recession of the convergence near-point, but later it develops into a true convergence-insufficiency.

Accommodative convergence-insufficiency occurs in myopes who are not using concave glasses, and may also occur (from sudden relaxation of the accommodation) in hyperopes and presbyopes when putting on convex glasses for the first time. A somewhat similar relaxation of the convergence is found in those who for any reason (presbyopia, marked hyperopia, anisometropia, etc.) can not see well for near; and in this case the convergence-insufficiency may be relieved by convex glasses.

The relation between myopia and the form of accommodative convergence-insufficiency that leads to many of the ordinary cases of divergent squint was first pointed out by Donders.

A *non-accommodative* convergence-insufficiency,—i.e., one which develops without any reference to a refractive error,—occurs quite often in hysteria, traumatic neuroses, neurasthenia, anæmia, and debilitated conditions, however caused; also in connection with intra-nasal disease, and not infrequently seems to occur in those who are apparently healthy. It is also often secondary to a divergence-excess (see page 785).

An extreme variety of convergence-insufficiency is *convergence-paralysis.* This is characterized by a crossed diplopia and a divergent squint which increase progressively as the object of fixation is carried toward the eyes. The deviation and the diplopia do not increase either to the right or to the left. Movements of each eye outward and inward are normal. The convergence reaction of the pupil is absent. Convergence-paralysis occurs in tabes, multiple sclerosis, and other central nervous diseases, either organic or functional. It may be associated with paralysis of accommodation (§ 797).

Divergence-excess is common as a primary condition. It also occurs as a regular secondary effect of a progressive convergence-insufficiency (see below).

685. A convergence-insufficiency, especially if due to temporary causes, may disappear; or it may increase by the superadding of a divergence-excess. A divergence-excess often remains stationary for a number of years, but it may also increase by the superadding of a convergence-insufficiency.

These combined forms (convergence-insufficiency with divergence-excess) constitute the ordinary continuous *divergent squint.* This develops most often from a convergence-insufficiency, the steps of development being as follows:

(1) Marked exophoria for near with at first a normal near point of convergence; orthophoria for distance and normal diverging power (*periodic exophoria*).

(2) Beginning divergent squint for near with recession of the convergence near-point; beginning exophoria for distance with increase in the prism-divergence (*periodic divergent squint*). This squint, at first intermittent, is afterwards constant.

(3) Marked increase of the exophoria for distance with gradual transformation into squint (consecutive divergence-excess) converting a periodic into a *continuous squint.*

(4) *Muscular changes* (contracture of the externus, stretching and weakening of the internus) producing absolute impairment of rotation inward and excess of rotation outward.

A divergent squint also quite often develops from a divergence-excess; the steps in the development being as follows:

(1) Marked exophoria for distance with excessive prism-divergence; exophoria for near, little or none, and near point of convergence normal (*periodic exophoria*).

(2) Beginning divergent squint for distance with crossed diplopia and still further increase in the prism-divergence; beginning exophoria for near, but with convergence near-point still normal (*periodic divergent squint*). This squint is at first intermittent, afterwards constant.

(3) Marked increase of the exophoria for near and gradual recession of the convergence near-point (*consecutive convergence-insufficiency*), producing a squint for near, which, however, is less than for distance).

(4) Marked divergent squint which is of about the same amount for both distance and near (*conversion of the periodic into a continuous squint* by the superadding of a convergence-insufficiency to the primitive divergence-excess). Diplopia still often present, but now usually but little noticed by the patient. The rotations of the eye inward and outward are still about normal.

(5) *Muscular changes* (weakening of the interni; contracture of the externi) causing impairment of rotation inwards and excess of rotation outwards.

686. How Squint Develops.—As seen from § 683 and § 685 a convergent squint usually develops out of a simple convergence-excess to which is added a divergence-insufficiency; and a divergent squint develops out of a simple convergence-insufficiency to which is added a divergence-excess or out of a divergence-excess to which is added a convergence-insufficiency.

The processes by which these changes take place are not located in the muscles themselves, for until very late in the development of most cases of squint the rotations of the eyes are normal, indicating that the power of the muscles per se is neither impaired nor excessive. The changes that take place are doubtless *central*, following, as Hoffmann pointed out, the law promulgated by Sherrington, that excessive stimulation of the center for one movement produces inhibition of the center for the opposing movement, and vice versa. In other words, in cases of convergence-excess the center for convergent movement, being subjected to continuous over-stimulation, becomes more and more active, producing a continually greater excess of convergence action and at the same time causing greater and greater inhibition of the center for the divergent movements. The consequence is that, superadded to the convergence excess, we have ultimately a progressive divergence-insufficiency. It is only late in the course of the condition that the persistent stimulation of the internus, coupled with the maintenance

of one eye in the forced inward position, produces hypertrophy or contracture of this muscle and stretching or atrophy of its opponents.

A moderate proportion of cases of squint do, however, *develop out of a true muscular defect.* Thus, some cases of convergent squint, both in childhood and in adult life, develop out of a primitive paralysis of the abducens which has been converted into a comitant deviation by consecutive overaction and contracture of the internus (see page 742). Still more frequently it happens that a vertical deviation due to congenital insufficiency of the superior rectus (less often of other muscles) is the starting point of a convergent or divergent strabismus (see pages 757 and 788). Furthermore, a convergent squint due to a convergence-excess may be aggravated by the presence of a primitive weakness of the externi or over-development of the interni. Again, a divergent squint may be produced by developmental changes in the orbit causing a relative preponderance of the externi (see page 781), or by weakness of the internus due to operation. Such factors, however, play a subordinate part in the production of squint.

Age at which Squint Develops.—Some cases of squint are *congenital* or at least develop in the first months of life. These however, are in nearly all instances due to congenital weakness or absence of one or more of the muscles and therefore come under the head of paralytic squint, to which therefore reference should be made (see page 736). Ordinary convergent strabismus develops, as a rule, at the age—i. e., between one and four—when the attempt at accurate and long-maintained fixation begins and thus puts a greater strain on the accommodation. This is true not only of the ordinary type of accommodative squint, but also of the lateral squint that develops as the result of a congenital vertical deviation (pages 757 and 788). At first usually the strabismus is noticed only when near objects are looked at (periodic squint). This may remain so during life, but generally a constant strabismus develops from the periodic one, the patient soon getting to squint also when he looks at distant objects. Even then the squint at first is usually greater in the fixation of near objects, in accordance with the greater accommodative effort required for this purpose; but later on the strabismic deviation becomes continuous. (See page 782).

Exceptionally it happens that as they grow up children with convergent strabismus gradually cease squinting about puberty. They "outgrow" their squint. But the eye that previously deviated is left with its sight permanently weakened, and accurate binocular vision is not usually restored.

Unlike convergent squint, divergent strabismus does not usually develop in very early life but later on in childhood, or in youth. This is naturally the case with the accommodative divergent squint due to myopia, since myopia itself does not begin in early, but in late childhood; but it is likewise true of the many cases of divergent squint due to non-accommodative conditions (non-accommodative convergence-insufficiency,

divergence-excess). In myopes as the myopia increases, the work imposed on the accommodation and consequently the impulse for convergence steadily decrease, while the demands made upon the convergence increase in proportion, owing to the approximation of the near point. This state of things must ultimately lead to a point where the convergence is no longer able to answer the demands made upon it. The convergence first becomes incompetent in the fixation of near objects, for which a stronger effort is required, and thus one of the eyes deviates out. Many myopes remain all their life in this condition of periodic strabismus; in others a constant strabismus develops from this condition, one eye getting to deviate out in looking at distant points as well. Quite a similar statement obtains for the progress of non-accommodative divergent squint (see page 785).

A spontaneous cure, such as is sometimes observed in inward squint, never occurs in divergent strabismus; on the contrary, it tends to increase with age.

Contrary to the rule that short-sighted persons squint outward, strabismus convergens is sometimes found conjoined with high myopia. This is distinguished from ordinary strabismus convergens by the fact that it does not develop in childhood, but in the later years of life, and is often associated with troublesome diplopia. At the same time it is quite possible that the but slightly mobile eye should be unable to converge for a far point so near as such eyes possess, and in this case there is also a relative divergent squint present when reading is performed without glasses.

687. Why Squint Develops.—It seems quite certain that *in their inception the different periodic deviations* (*convergence-excess, convergence-insufficiency, divergence-excess, divergence-insufficiency*) *are accompanied by diplopia.*

Now the more nearly continuous (non-periodic) a deviation is the easier it is for the patient to ignore the diplopia to which it gives rise, and the less pronounced, consequently, are the subjective disturbances which are dependent on this diplopia.

Again, a large deviation generally causes much less subjective trouble than does a small one: First, because the image projected by the deviating eye is so faint and so far from the other that the patient can readily ignore it; second, because the deviation being so large that the patient can not possibly overcome it, he does not even make the attempt to do so, and thus escapes the reflex disturbance that such an attempt often produces. Accordingly, it is for the patient's interest that *if he has a periodic deviation it shall be transformed into one that is as nearly continuous as possible*; and, second, *that if it is small it shall increase in amount until the diplopia is readily negligible.*

As we have seen above (pages 782 and 784) both kinds of change occur regularly in the conversion of a heterophoria into a convergent or a divergent squint, and the squint itself, therefore, is to be regarded as

the final outcome of a series of instinctive endeavors that the patient makes *to avoid diplopia and confusing images.*

688. Conditions Favoring the Development of Squint.—The development of a squint out of a heterophoria is much more apt to occur if there is any condition such as anisometropia, opacities of the media, fundus disease, congenital anomalies, etc., that *renders the sight of one eye much worse than that of the other.* In such a case the image of the worse eye often confuses that of its fellow instead of reinforcing it, and hence the patient really sees better when he allows the worse eye to deviate. In many cases it is apparent that he actually forces it to deviate so as to get the disturbing image out of the way. This is particularly the case in divergent squint.

There is still another way in which the presence of unilateral visual defects may effect the development of squint. In the ordinary convergent squint of childhood developing as the result of hyperopia, the hyperope is placed in the following dilemma: If he wishes to see distinctly, he has to make too strong an effort of accommodation; but he can do this only by putting forth an excessive convergence, so that he sees double. But if he converges only so much as is necessary, he cannot bring the proper amount of accommodation into play, and hence sees indistinctly. He is, therefore, given the choice of seeing distinctly and double, or of seeing single and indistinctly. He prefers the former alternative whenever from the fact that the image in one of the eyes has grown indistinct, diplopia is made less disagreeable to him. A similar explanation may hold for the development of divergent strabismus.

Both explanations are quite certainly true, the second holding particularly when the amblyopia in one eye is extreme or when as in the case of blind eyes it is absolute. Blind eyes, in fact very frequently squint (usually outward).

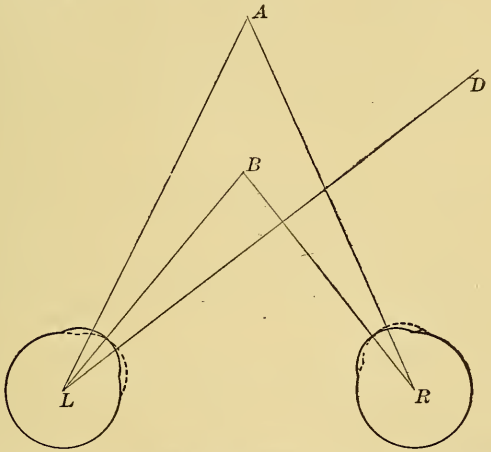
Another condition that favors the development of a lateral squint is the presence of a congenital vertical deviation (especially a congenital paralysis of the superior rectus). The patient in this case, being unable to fuse the double images on account of their difference in level, tries to obviate the confusion they produce either by tilting the head (page 741) or by separating the images laterally as far as possible. This he does by diverging or converging the eyes.

That this is the cause of the lateral squint in these cases is proved by the fact that in some of them, particularly before the lateral deviation has become inveterate, the latter disappears when the vertical deviation is relieved by an operation on one of the elevators or depressors.

A factor which by many is regarded as the predominating element in the production of squint is the *lack of development of the fusion-faculty* (Worth). The effect of this would be that the patient would have no tendency to blend the two retinal images, and hence would have no special

motive for maintaining binocular fixation. Consequently, if there was a deviation of the eye, he would not try to overcome it, but would allow one of them to squint.[25]

689. Squint a Bilateral Affection.—In squint the patient appears simply to fix with one eye and to turn the other inwards or outwards. In reality, however, both eyes are turned. Thus, as shown in Fig. 347 when a patient with convergent squint is told to look at the object A, both eyes tend to converge equally, so that they really look at some nearer point, like B; then, in order to get the image of A on the fovea of at least one eye, the patient turns both eyes to one side—say to the right—through the equal angles BRA and BLD. The right eye, R, is now directed towards A or fixes it, the left eye squints in by the angle ALD = 2× BLA. Thus, here really the squint is bilateral, each eye actually turning in by an equal amount, yet apparently the left eye alone squints, because the patient happens to use the right for fixation.



[Fig. 347.—Bilateral Nature of Squint. (After Posey and Spiller.)—D.]

Which eye the patient actually uses to fix with depends usually upon the vision. If he sees much better with one eye than with the other he will fix with the better and squint with the worse eye; the squint will then be unilateral. If the two eyes are about alike in vision and refracting power, we often find an alternating squint, the patient using either eye indifferently for fixation and squinting with the other. In some cases, particularly in anisometropia, the patient may fix with one eye for distance and with the other for near (see page 762).

690. Hyperphoria and Vertical Squint.—Hyperphoria in a great many instances is caused by paresis, or at least insufficiency of one of the elevator or depressor muscles of the eye. In this case the deviation and its evidences

[25] It has seemed to the translator that the lack of ability to perform fusion in these cases is not so much the cause of the squint as the result of it.

(vertical diplopia, etc.) increase and decrease characteristically in looking in some particular direction of the gaze (see page 743). Hyperphoria of high degree and real vertical squint (hypertropia and hypotropia) are almost always of this origin, being, therefore, *non-comitant* deviations.

In some cases, even when thus originating, a hyperphoria or vertical squint may become nearly comitant by virtue of consecutive contracture of the opponents of the paretic muscles (see page 742).

In a few instances with very high degree of hyperphoria a true vertical squint may be truly *comitant* from the start—i. e., we can find no evidence of insufficiency of any elevator or depressor muscle. Hyperphorias of low degree are usually comitant.

Some cases of hyperphoria seem to be *spasmodic* in origin, changing rapidly and lasting only a short time. Sometimes a spasmodic hyperphoria of this sort seems to be due in some way to accommodative action, since it disappears apparently as the result of the correction of a refractive error. In most cases hyperphoria once developed lasts indefinitely. It does not, as a rule, tend to increase, except in the form due to marked paralysis of an elevator or depressor where the deviation is often exaggerated as time goes on, either by secondary contracture of the opposing muscles or, because the paretic eye fixes and the sound eye hence deviates excessively (see page 745).

691. Symptoms of Heterophoria and Squint.—These are—

1. *Manifest deviation* of the eyes.

2. *Diplopia.* This, especially when slight so that the double images are close enough together to confuse each other, produces blurring of sight and sometimes vertigo.

3. The attempt to avoid diplopia may produce a peculiar *attitude of the head* (page 741). This may even result in false torticollis.

4. *Amblyopia* from disuse of the deviating eye (see page 763).

5. *Reflex Symptoms Induced by the Effort made to Correct the Deviation.*—These are asthenopia (asthenopia muscularis), pain in the eyes, conjunctival irritation, headache, which may be supra-orbital, orbital, temporal, or occipital, and other pains situated in various parts of the body. Occasionally, also, we find spasm of the facial muscles, and sometimes other forms of spasm; not infrequently also digestive disturbances, nausea, and interference with the general nutrition.

These symptoms, and especially the asthenopia, are the result of the strain imposed on the exterior muscles in the attempt to correct the deviation. If the patient does not correct the deviation, he sees double, and objects look confused; if he does correct it, the muscles after a time get tired from the continuous effort. Not infrequently there is an alternation between distinct vision with a sense of strain, and diplopia or confused vision when the strain is relaxed.

Reflex symptoms are especially pronounced when the deviation is so *slight* that the patient can overcome it, and consequently does so, although

with effort. They are particularly marked when the deviation is inconstant and varies under different conditions, because in this case the patient can not as readily allow for the changing position of the eyes as he can when the deviation is the same all the time. Consequently, reflex disturbances are *greater in heterophoria* than they are in squint and *greater in a periodic squint* than they are in one which is continuous. Indeed, as we have seen, this very fact seems to constitute the reason why a periodic is regularly converted into a continuous squint.

The most marked and disagreeable symptoms are found in hyperphoria and in divergence-insufficiency. In these conditions the symptoms are usually most troublesome in *distant vision.*

In convergence-insufficiency the commonest symptoms are asthenopia, headache, and conjunctival irritation, and these are developed generally by *near work.* They are much more likely to occur in the non-accommodative form of convergence-insufficiency.

A characteristic feature of the symptoms due to a muscular trouble is that they disappear if one eye is closed.

692. Treatment of Heterophoria and Squint.—1. *Correction of the refraction* is a very important element in the treatment of all of these anomalies and may alone suffice to effect a cure, particularly when the deviation is still of the periodic type. It is of special importance and efficacy in convergence-excess, particularly convergence-excess which is developing into squint. Here it is necessary to correct the whole of the hyperopia[26] and astigmatism present as found under atropine, and make the patient wear the glass all the time. Similarly, in convergence-insufficiency accompanying myopia, the patient must wear a glass fully correcting his error and wear it for near as well as for distance.

In cases in which the deviation is no longer periodic—i. e., in which, in addition to a convergence anomaly, there is superadded an anomaly of divergence,—correction of the refraction is less apt to afford a cure, and in the case of a continuous squint rarely does more than diminish the amount of the deviation. In any case, the effect of correction of refraction is rarely an immediate one; it usually takes some months for the full effect of the glasses to be secured. Moreover, if the correction with glasses is to do any good, it must be kept up continuously.

The reason why glasses often effect only a partial cure in continuous squint is that they eliminate simply the accommodative element. Thus, in a hyperope with a combined convergence-excess and divergence-insufficiency they eliminate the former only, so as to convert the deviation from a continuous to a periodic one. Later, to be sure, the residual divergence-insufficiency may diminish and even disappear, since the very relief of the strain on the convergence may diminish also the tendency to divergence inhibition.

[26] A deduction of 0.25 to 0.50 D. may be made from the full correction in some cases, particularly in older subjects. But, as a rule, the nearer to the full correction we give, the better results we get.

The earlier in the course of a muscular trouble, the treatment with glasses is initiated, the more likely it is to succeed, especially in cases of squint. Hence it is important to start it, if possible, at the very beginning of squint. As Worth shows, glasses may be worn even at the age of 1 or 2 years, and there should be no hesitation in prescribing then the approximate correction determined by objective tests (skiascopy). But, though we should endeavor to give glasses as early as possible in these cases, it must not be forgotten that their application sometimes succeeds when it is deferred till youth or even till adult life.

A fully developed divergent squint is rarely relieved by glasses, because here the converging muscles are generally weakened. But in cases of beginning divergent squint, especially, in youth and in cases of accommodative exophoria the deviation can often be lessened and its progress arrested by this means.

Cases in which the accommodative element is lacking do not yield to correction of the refraction. Such are especially cases in which the deviation is due either directly to a congenital weakness of the muscles or arises indirectly from such weakness, as is the case, for example, when a large vertical deflection of congenital origin causes an acquired lateral deviation to develop later (see page 788). Even in the latter case, there may be an accommodative element that acts to increase the deviation; and so far as the latter is due to this accommodative element it can be relieved by glasses.

The treatment of *heterophoria with glasses* is subject to the same rules as squint. The more fully the element of a convergence-excess enters into the case, the fuller should be the correction of the hyperopia. On the other hand, to presbyopes who show a marked convergence-insufficiency we should be careful to give as weak a reading addition as they can conveniently take, since the stronger the glass and the greater, therefore, the approximation of the near work, the more strain there is put on the weak converging power. Such patients often complain that their glasses "draw" and are too strong, and are much relieved when these are reduced in strength.

Cases of convergence-excess, especially with low accommodative power, in which the amount of esophoria for near is greater than for distance, may be helped by bifocal glasses in which the lower segments represent an addition of +2D or +3D to the correction for distance. The results from this treatment are said to be good, although the translator has not had much success with it.

693. (2) *Exercise of Fixation and Vision in the Squinting Eye.*—In squint, we must try to prevent the squinting eye from habituating itself to exclusion and thus declining more and more into a state of amblyopia. With this object we force the squinting eye to fixate by bandaging the non-squinting eye several hours every day. Since this measure is often baffled by the children's resistance, we may instead of it instill a drop of atropine into the sound eye every second or fourth day, so that for near

it does not see clearly and the child is compelled to use the squinting eye. This measure does not succeed if the squinting eye is very amblyopic already, in which case bandaging of the sound eye must be resorted to. Atropinization of the sound eye, if it straightens the squinting eye makes the sound eye squint, and if kept up too long can make the sound eye amblyopic. We use either atropinization or bandaging of the sound eye both in cases which for any reason do not wear glasses and also in cases in which glasses have been prescribed.

When excluding the good eye in this way, the patient should systematically exercise the squinting eye in seeing and fixing—by kindergarten games with minute objects, if he is very young, and by reading, if he is older. The exercises, which should be combined with the orthoptic exercises, should be done several times a day and should last from 10 to 30 minutes—their duration being increased as the child gets more expert. If they are kept up steadily for months, sometimes very substantial gains in vision are secured, and thereby the likelihood of a cure by orthoptic exercises or by operation is increased.

694. (3) *Orthoptic Exercises.*—With a squint that develops in early life much can be done by educating the fusion faculty. This can be done with the *stereoscope*, or, better still, with Worth's *amblyoscope*. Both of these instruments, by compelling the eyes to act together, train the fusion faculty. Both are at first adjusted for the patient's angle of squint, then by successive adjustments the patient is gradually taught to look into the instruments with the eyes more and more nearly straight and yet maintaining fusion. In this way the angle of squint is steadily diminished, and there is finally orthophoria.

This is not simply an exercise of fusion, but also and probably mainly an exercise of the divergence or convergence, as the case may be.

In applying the amblyoscope and, indeed, in all orthoptic exercises, the vision of the two eyes should as far as possible be placed on an equality. This is done by reducing the vision of the good eye with a dark glass or in the case of the amblyoscope by dimming the picture seen by this eye with an extra thickness of celluloid.

Another orthoptic exercise is *bar-reading*. This, as already stated, (see page 772) consists in reading with a bar or pencil held between the eyes and the print. Unless the patient is using both eyes at once for seeing, the bar will hide some of the letters. The systematic use of the bar is an exercise in binocular vision.

If the patient can recognize *double images* at all, he can be practiced in recognizing and, if possible, in overcoming diplopia. This is best done by placing a red glass before one eye and a green glass before the other—darkening the sound eye with an extra thickness of glass—and making the patient look at a light. As soon as he can be got to recognize the diplopia readily, it is well to replace this exercise with the amblyoscope and bar-reading.

695. (4) *Exercises in Moving the Eyes.*—Exercises of the eyes in converging on a pencil carried towards the nose or in overcoming prisms, base out, are useful in convergence-insufficiency and occasionally in divergence-excess. Sometimes they relieve the symptoms when they do not essentially diminish the deviation.

In practicing with prisms, base out, the test-object should be placed at the reading distance if we are dealing with a pure convergence-insufficiency, and at six metres if we are dealing with a divergence-excess. In mixed conditions, exercise at both distances should be used. Convergence-insufficiency may also be treated by exercising the convergence with the amblyoscope (see supra).

Convergence-excess may be treated by exercising the divergence with the amblyoscope or by practice in overcoming prisms, base in, the object in this case being held at the reading distance.

Other exercises have been tried, e. g., rotating the eyes from side to side, or up and down, or turning the head from side, while keeping the eyes fixed. Such exercises may be indicated when the muscles per se are weak, but would hardly have any influence in divergence or convergence anomalies.

696. (5) *Prisms for Wear.*—Deviations of low degree may be corrected by prisms worn constantly, either alone or combined with the glass correcting the refraction. This is most serviceable in hyperphoria. In esophoria and exophoria, prisms constantly worn often tend to increase the deviation, and their use is not generally advisable. Moreover, it is not usually possible for a patient to wear prisms of more than 3Δ, or at most 4Δ, before one eye, so that the total amount of deviation that we can correct by this means would not be more than 7Δ at most.

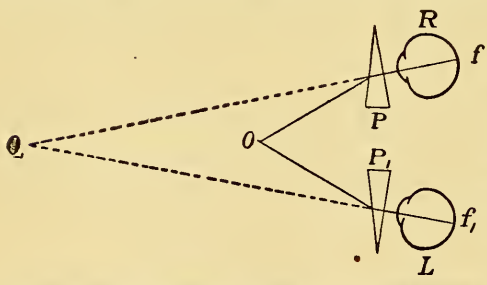


Fig. 348.—Employment of Prisms in Convergence-Insufficiency.

For the reasons given on page 757, prisms are rarely serviceable in paretic deviations.

The way in which prisms help in cases of deviation is shown in Fig. 348, which is supposed to represent a case of convergence-insufficiency. In the case shown the prisms are placed before both eyes and in such a way that their bases are directed inward (*P* and P_1, Fig. 348). The rays coming from the point of fixation, *O*, are deflected by each prism toward its base. The eyes, therefore, need only converge, as though, they were gazing at the more remotely situated point, O_1.

697. (6) *General treatment* must be employed in neurasthenia and hysteria and in conditions of debility from whatever cause. This will often relieve the symptoms without producing any material change in the muscular condition of the eyes.

698. (7) *Treatment of Associated Conditions.*—The treatment of other affections, particularly intra-nasal disease, which may possibly be sources of reflex trouble, should never be neglected.

699. (8) *Operation.*—When everything else has been tried and has failed, an operation is indicated. For methods, see § § 882-888.

With an *esophoria* or *convergent squint*, tenotomy of one or, better, of both internal recti is done, provided the condition is mainly one of convergence-excess. When the condition is one of divergence-insufficiency, the better plan is advancement of one or both externi.

Similarly, in *exophoria* or *divergent squint* caused by divergence-excess, tenotomy of one or, better, of both external recti, is indicated. Cases of convergence-insufficiency, on the other hand, should be treated by advancement of the interni. In marked divergent strabismus tenotomy of one or both externi, combined with advancement of the interni, usually has to be done.

In *hyperphoria*, if non-comitant, we proceed according to the rules laid down for operative interference in paretic deviations (see § 890). In a comitant hyperphoria the most successful operation is a tenotomy of the superior rectus, which should never be pushed so far as to cause the muscle to become paretic nor so far as to produce even a moderate over-correction.

In heterophoria an operation, if performed with judgment and with careful regard to the underlying conditions of the heterophoria, often relieves, sometimes to a surprising degree, the asthenopia, confusion, headache, and other symptoms. In squint the result obtained by the operation is, as a rule, simply cosmetic. The sight of the squinting eye is not influenced by the operation, and in only a few instances is even binocular vision restored. Nevertheless, the results that we secure by operation are not to be undervalued. A man with squint scarcely ever complains of the bad sight in his squinting eye, or of the absence of binocular vision; he is ordinarily unaware of the existence of either. He only desires relief from his disfigurement, and is very grateful if this is accorded him.—D.]

[IV. Derangements of the Associated Parallel Movements of the Eye

700.—The derangements of the associated parallel movements of the eyes comprise—A. Paralysis and spasm of the parallel movements (conjugate paralysis, conjugate spasm). B. Tremor and ataxia of parallel movements (searching movements, nystagmus, pseudo-nystagmus). C. Dissociated movements.

Conjugate Paralysis and Spasm

701. Natures of these Anomalies.—*Conjugate deviation*, i. e., the condition in which both eyes are deviated in the same direction, is usually due to *conjugate paralysis* (see page 753). Thus if there is paralysis of right rotation, so that neither eye can rotate to the right beyond the middle line, the two eyes and sometimes the head also are usually deviated to the left by over-action of the left rotators.

In some cases conjugate deviation is due to *conjugate spasm.* For example, we may find both eyes deviated to the left, not because the right-rotators are paralyzed, but because the left-rotators are in a state of primary spastic contraction. Such a condition is distinguished from paralysis by the fact that, although strongly deviated in one direction, the eyes can usually be moved more or less freely in the direction opposite.

Symptoms and Course.—Since the deviation is alike in the two eyes, conjugate paralysis and spasm do not cause diplopia. They may, however, cause *vertigo* and *false projection* (A. Graefe; M. Sachs). In a conjugate deviation to the left, due to paralysis, the patient would overshoot the mark when trying to touch an object situated on his right, while if the deviation were due to spasm he would undershoot the mark when trying to touch an object situated on his left.

Conjugate paralysis occurs in all degrees of *intensity,* being sometimes very slight, sometimes complete. It usually develops suddenly, and is often transient. The conjugate deviation which the paralysis produces often disappears long before the paralysis itself. This is especially true in cerebral disease.

Varieties of Conjugate Paralysis and Spasm.—1. *Paralysis of lateral movement* occurs under the following forms:

(A) Neither eye, when attempting to follow an object that is moving to one side, say the right, can move beyond the middle line. But the left eye can move to the right when converging on an object which is brought in towards the eyes in the median plane (retention of convergence).

(B) Neither eye can follow an object that is carried to the right, provided both eyes are open, but the left eye can follow the object to the right if the right eye is covered. This is really the same case as A, the movement inwards of the left eye when the right eye is covered being simply a movement of convergence (Jeffries, Moebius).

(C) The right eye can follow an object that is moving to the right, but the left can not. The left eye, however, can move to the right in performing convergence. This condition, which may be called conjugate hemiparalysis, is thus distinguished from a paralysis of the left internus in which the left eye can move inwards neither when converging nor when attempting to make a conjugate movement to the left.

(D) Neither eye can follow an object that is carried to the right, and the left eye, moreover, cannot move to the right when attempting to converge (combined paralysis of lateral movement and convergence).

Paralysis of lateral movement is sometimes bilateral—i. e., both eyes fail to follow an object that is carried either to the right or to the left, although it may be that both can still converge.

2. *Spasm of lateral movement* occurs under the following forms:

(A) Both eyes are deviated spastically to one side, but can be moved freely in all directions (pure spasm of lateral movement).

(B) Both eyes are deviated spastically to one side, and can not be moved to the side opposite (excessive spasm of one side-to-side movement, or spasm of one side-to-side movement combined with paralysis of the movement in the opposite direction).

(3) *Paralysis* and *spasm of up and down movements* are rare.

Site of Lesion in Conjugate Paralysis and Spasm.—*Paralysis* of lateral movement is caused by destructive lesion in—

1. The *cerebrum* and either in the cortex, in the subcortical association paths, or in the tracts connecting the cortex with the oculo-motor and abducens nuclei. Such lesions are usually of sudden onset and associated with apoplectoid symptoms (coma, hemiplegia). A lesion of the right hemisphere produces paralysis of movements to the left, and consequently a conjugate deviation to the right (the patient turns his eyes towards the lesion). Convergence is always retained.

2. The *pons*. Lesions on the right side of the pons will produce paralysis of right-hand movements, and vice versa—i. e., the eyes look away from the lesion.

Small lesions in the pons involving the abducens nucleus or its immediate connections cause paralysis of lateral movement without paralysis of convergence (paralysis of types A and B, page 796). Lesions high in the pons involving only the fibers connecting the abducens and the oculo-motor nucleus produce a conjugate hemiparalysis of the opposite internus for parallel movements but not for convergence (type C, page 796). Larger lesions in the pons above and below the abducens nucleus cause combined paralysis of lateral movement and convergence (paralysis of type D, page 796). (Jeffries, Schoeler.)

3. *The cerebellum.* The deviation here is sometimes toward the lesion, usually away from it.

Spasm of conjugate movement is caused by an irritative lesion in—

1. The *cerebrum* (in the cortex only). A lesion situated on the right side causes a spastic deviation to the left, i. e., the eyes are turned away from the lesion.

2. The *pons* (very rare). In this case the eyes look toward the lesion.

Paralysis of up and down movement is due usually to a lesion involving the anterior parts of both oculo-motor nuclei or the corpora quadrigemina or to one occupying the middle line close behind the corpora quadrigemina. It also occurs in pons lesions.

Some cases *simulating conjugate paralysis* are due to symmetrical defect in the muscles or to symmetrical lesions of the two oculo-motor nerves at the base.

Tremor of the Associated Parallel Movements. Nystagmus and Pseudo-Nystagmus

702. Searching Movements.—Both eyes make an occasional wide, comparatively slow, sweeping movement from the primary position in some direction, and then, either immediately or after a time, return to the primary position. The movements are apparently purposeful, as if the patient were trying to look at an object situated to one side of him. They may take place in any direction, vertical, oblique, or horizontal.

Searching movements are found in blind eyes and in eyes which from disease at the macula and other causes have lost the power of central fixation.

703. Pseudo-Nystagmus.—One or both eyes when carried to a point near the limit of their excursions in some direction, make a series of jerky movements on from this point and back to it again, but in returning do not re-pass it. These jerks evidently represent an extreme effort to keep up the original excursive movement of the eyes, the smooth steady pull of the muscle that is carrying the eye along being converted into a series of discontinuous, spasmodic tugs.

Pseudo-nystagmus is sometimes unilateral, more often bilateral.

Pseudo-nystagmus occurs almost constantly in hereditary ataxia and very often in multiple sclerosis, but also occurs in a great many other nervous affections and often in persons who are quite healthy. It seems to denote usually weakness or fatigue of the muscles which move the eye in the direction in which the pseudo-nystagmus occurs.

704. Nystagmus.—In nystagmus the eyes make a series of very regular, short, quick oscillations about a central point. Nystagmus differs from pseudo-nystagmus, first, in that it often occurs when the eyes are in the primary position or near it, whereas pseudo-nystagmus occurs only when the eyes are near the end of an excursion in some direction; second, the movements in nystagmus are oscillations to and fro about a central point, while in pseudo-nystagmus they are movements to a terminal point and back from it again; third, the movements in pseudo-nystagmus are jerky and bespeak effort, in true nystagmus are smooth and regular, and, except in miners' nystagmus, are not often associated with a restriction in the excursive power of the eyes, which is very frequently present in pseudo-nystagmus.

As regards the *character* of the movement, a distinction is made between pendulum-like nystagmus (nystagmus vibrans) and resilient nystagmus (nystagmus resiliens). In the former, the movement has the same velocity to and fro; in the latter, the eye moves slowly to one side and returns rapidly to the other. Pendulum-like nystagmus is the distinguishing mark of cases characterized by amblyopia (cases of optical nystagmus, but also occurs in other forms, e. g., miners' nystagmus). Resilient nystagmus characterizes cases due to disturbance of innervation in the labyrinth (aural nystagmus).

In *direction* the oscillations may be horizontal, vertical, rotary (wheel-like), or

mixed (combination of horizontal and vertical or horizontal and rotary). A peculiar variety of mixed nystagmus is *circumduction* nystagmus in which by making successive horizontal and vertical movements the center of the cornea describes a circle or ellipse. Horizontal and vertical nystagmus are called collectively nystagmus oscillatorius.

True nystagmus is nearly always bilateral and then the movements are almost invariably equal, simultaneous, and parallel in the two eyes (*conjugate* nystagmus). Bilateral nystagmus is usually horizontal, less often rotary or mixed. Vertical bilateral nystagmus is rare.

Very rare forms are (a) *disjunctive* nystagmus in which the two eyes swing rapidly toward and away from each other, i. e., make rapidly alternating converging and diverging movements, or revolve toward and away from each other; (b) *dissociated* nystagmus, in which the movements in the two eyes are quite dissimilar in direction or in extent; (c) *unilateral* nystagmus. Unilateral nystagmus is usually vertical.

In many cases nystagmus changes greatly in intensity or in character when the eyes are turned in some special direction, or when they are converged, or when either or both are covered. Many patients make the same vibratory movements with their heads as with their eyes, but in an opposite direction.

705. Etiology of Nystagmus.—Nystagmus is caused by—

(A) Conditions producing bilateral amblyopia in early infancy (*optical*, infantile, or so-called congenital nystagmus). Such conditions are opacities of the cornea, especially from ophthalmia neonatorum; congenital cataract; hæmorrhage, disease, or abnormalities of the retina and chorioid; total congenital color blindness, and albinism. Refractive errors, even when great, do not usually cause nystagmus, although they may do so. Optical nystagmus is probably not often really congenital, but develops during the first few months of life.

In what way does amblyopia produce nystagmus? Fixation is not a faculty inborn in man, but has to be learned by practice. Very small children do not perform fixation, but move their eyes about aimlessly. In what then does fixation consist? When an eccentric spot of the retina has impressed on it an image that excites the attention, there is set up by reflex action an eye movement which is just calculated to place the fovea opposite the object. As the eye moves and the image thus approaches the fovea, the image becomes more and more distinct, and most distinct of all precisely when the fovea is reached. The moment this occurs the movement ceases, for should the eye be carried by the position required for fixation, the object would at once appear more indistinct again. But such an inhibition of movement at the right moment presupposes that as regards vision the fovea greatly surpasses the adjoining portions of the retina. If—on account of the affections above mentioned—this is not the case, an arrest of movement at the right moment fails to occur, and the eye, as it were, vibrates in small excursions about the position of fixation, just like the tremulous hand of an old man that is trying to grasp some small object. Hence, nystagmus fails to develop if the amblyopia does not set in till later in life, at a time when the eyes have already learned how to perform fixation.

A certain degree of visual power must be present for nystagmus to exist; persons who are born blind, or who become totally blind very early, do not acquire nystagmus. In such persons the eyes move slowly and aimlessly about, making large excursions.

Unilateral amblyopia very rarely produces nystagmus but tends rather to produce squint.

Sometimes a nystagmus dating from infancy, occurs without there being any obvious lesion of the eyes to cause it. In such cases, according to A. Graefe, the nystagmus is probably attributable to a congenital retinal hæmorrhage which subsequently cleared up.

In small children a combination of head-nodding and nystagmus occurs as a transient affection which is known as *spasmus nutans*. According to some it is produced by the child's being kept in a dark room, according to others by rickets (craniotabes). The nystagmus in this case often assumes aberrant forms and may be unilateral. Whether it is optical or not is uncertain.

(B) Conditions developing *in later life* causing constant *strain of the eyesight.*

This variety of late acquired nystagmus is usually due to conditions incident to the patient's occupation (hence *occupational* nystagmus). The commonest form of this is miners' nystagmus, which is due mainly, if not altogether, to insufficient illumination—not, as often stated to working with the eyes in a constrained position. (The pick men in coal mines do not really work with the eyes turned up.) Nystagmus due to insufficient illumination occurs occasionally in other occupations (Frost), and a twitching (probably not a true nystagmus), which Snell ascribed to constrained position of the eyes, occurs in compositors, etc.

In the same category belong those cases in which optical nystagmus develops after the age of infancy as a result of high astigmatism or of traumatic cataract.

(C) Irritation arising from the ear (*aural* nystagmus). It may be produced by actual disease of the labyrinth, by changes in labyrinthine pressure due to rapid rotation of the body, and by operative manipulations carried on in the middle and external ear (probing, syringing, etc.).

(D) *Nervous Disease.*—True nystagmus occurs frequently in multiple sclerosis (in 12 per cent of the cases, according to Uhthoff), also in syringomyelia and in cerebellar disease. In other nervous diseases it is rare.

(E) *Poisons*, especially ether; rarely also alcohol, morphine, cocaine, sulphonal, arsenic, lead, quinine, ergot, and sewer gas.

Nystagmus, whether infantile or due to nervous disease, is sometimes *hereditary*. Some persons can produce nystagmus *voluntarily*.

Site of Lesion in Nystagmus.—In optical, occupational, and aural nystagmus, there is no actual lesion of the brain. In the nystagmus of multiple sclerosis and other organic nervous diseases, lesions have been found mainly in the corpora quadrigemina, corpus striatum, restiform bodies, cerebellum, and medulla.

Theory of Nystagmus.—The remarkable regularity and parallelism of the movements in nystagmus show that we have to deal with a perversion of the centers for parallel and parallel-rotary movements and not with peripheral lesions of the muscles themselves or their nerves.

This perverted state of the association centers is evidently produced in optical

and miners' nystagmus by long-continued abnormal stimuli passing up through the visual tract, and in miners' nystagmus perhaps also through the fifth nerve; in aural nystagmus by abnormal reflexes passing through the auditory nerve to the centers in the pons and perhaps also in the corpora quadrigemina; while in nystagmus due to nervous disease, the association centers and tracts are probably affected directly.

Nystagmus in all probability is produced by alternate, just as normal fixation is produced by simultaneous, discharges of motor energy from the two sides of the brain.

This explanation of the nature of nystagmus holds good undoubtedly even for the rare unilateral and dissociated forms.

706. Symptoms of Nystagmus.—They are—

1. *Apparent Movement of Objects.*—In miners' nystagmus and aural nystagmus this is a very prominent and distressing symptom. It is also often, but not always, present in the nystagmus due to nervous disease. On the other hand, in optical nystagmus due to infantile amblyopia, it is very rarely perceived. Sometimes in unilateral nystagmus and under exceptional conditions, in bilateral nystagmus it can be evoked (A. Graefe). Even then, however, it seems hardly ever to be troublesome.

2. *Reading in Vertical Lines.*—That the apparent movement of objects, even when it is not perceived by the patient, yet causes indistinctness of things seen, is shown by the fact that some patients with horizontal nystagmus in reading hold the book sideways, so that the lines of print run vertically. This, to a certain extent, obviates the confusion produced by the oscillations of the letters.

3. *Vertigo.*—This is marked in some of the late acquired forms especially in miners' and aural nystagmus.

4. *Nodding Movements of Head.*—These often occur in optical nystagmus. They were formerly thought to be compensatory in character, acting to prevent the tremor of objects produced by the oscillation of the eyes. This however is not the case.

5. *Poor Vision.*—In most cases of nystagmus of infantile origin, the vision is quite poor, and can not be brought up to normal by glasses. This is in part due to the blurring that the nystagmus produces, but much more to the pathological condition causing the nystagmus. In those cases in which the nystagmus increases greatly in one direction of the gaze and diminishes in another, the sight is very much better in that position in which the oscillation is slight—a fact which the patient gets to appreciate and utilize. In miners' nystagmus the disturbance of sight produced is considerable.

6. *Photophobia* may be present.

Course of Nystagmus.—Infantile nystagmus often lasts through life, although it may disappear. Nystagmus due to aural or nervous disease persists as long as its cause. Miners' nystagmus disappears if the patient gives up his work and lives under proper conditions as regards illumination and eye-strain. Nystagmus due to spasmus nutans and hysteria is prob-

ably always transient. A transient nystagmus may also result from traumatism, tenotomy, or other causes.

Treatment.—Infantile nystagmus is rarely affected by any treatment, although isolated cases have been cured by removal of a congenital cataract, by tenotomy or advancement causing relief of a squint, by exercises with a stereoscope and in reading with the aid of perforated diaphragm, by exercises in fixation in various positions of the gaze, and by exercises with rotating prisms. Some cases of nystagmus, especially acquired nystagmus, are relieved by the correction of astigmatism or other refractive errors. In miners' and other forms of occupational nystagmus change of occupation is imperative.

Dissociated Movements

707. In some cases the eyes no longer follow the law of associated movements, but each moves irrespective of the other. This may occur in eyes that have been totally blind from birth, in patients deeply under the influence of chloroform, and occasionally in other cases. Various forms of this anomaly have been described.

In one form of partial dissociation of the ocular movements each eye on alternate covering goes up or each eye goes down behind the screen. In these cases there may or may not be binocular fixation when both eyes are uncovered (see page 762, notes).

Some of the cases described as illustrating dissociation of the eye movements are not really of this character. Thus when several muscles are paralyzed in one eye and yet this eye is used for fixation, the secondary deviations of the sound eye, rapidly changing their form and varying their amount as the eyes are carried in different directions, make it appear as if the eyes were moving quite contrary to the ordinary laws of association.—D.]

CHAPTER XV

DISEASES OF THE ORBIT

Anatomy

708. The bony orbit forms a quadrilateral pyramid, whose base corresponds to the anterior aperture of the orbit, and whose apex to the optic foramen. The nasal walls of the two orbits are about parallel to each other; but the temporal walls diverge from each other a good deal from behind forward. The nasal wall is the thinnest, as it is formed by the lachrymal bone, which is as thin as paper, and by the delicate lamina papyracea of the ethmoid (Fig. 284, *T* and *L*, Fig. 349, *L* and *l*). At its anterior extremity it bears the fossa lacrimalis for the reception of the lachrymal sac (Fig. 302, *fl*). In the posterior portions of the orbit are found three apertures which connect the orbit with the parts adjacent. These are: 1. The optic foramen (Fig. 349, *o*) which passes between the two roots of the lesser wing of the sphenoid (*k*) into the middle fossa of the skull. Through it the optic nerve and beneath the latter the ophthalmic artery pass into the orbit. 2. The superior orbital fissure (*s*) which lies at the junction of the upper and outer wall, and is bounded by the lesser and greater wings of the sphenoid (*k* and *g*). It also opens into the middle fossa of the skull, and transmits the nerves for the ocular muscles and the first branch of the trigeminus. 3. The inferior orbital fissure (*i*) which is longer than the superior orbital fissure, and lies at the junction of the outer and the lower wall of the orbit, between the great wing of the sphenoid (*g*) and the superior maxilla (*M*). It connects the orbit with the temporal fossa (fossa spheno-maxillaris). Through it the twigs of the second branch of the trigeminus, the largest of which is the infra-orbital nerve, pass into the orbit.

The inferior orbital fissure is closed by a fascia with which numerous smooth muscular fibers are interwoven (musculus orbitalis of Müller). These are innervated by the sympathetic.

In the growth of the body the orbit expands in proportion as the eyeball enlarges. If the eyeball is backward in its growth, and more especially if it is entirely destroyed in childhood, the dimensions of the orbit also remain smaller. If, therefore, in such cases it is desired later on in life to wear an artificial eye, we must content ourselves with one which is too small when compared with the other eye.

The walls of the orbit at their anterior margin become thickened into a strong bony ring, the *margin* of the orbit. This constitutes the most important defense of the eye against external force, especially above and below where it juts farthest out. On the inner side there is no sharply defined margin to the orbit, but here the eye is protected by the bridge of

the nose. On the outer side the orbital margin recedes the farthest (Fig. 301, *A*), so that here the eye is most exposed to injuries. At the upper margin of the orbit is found the supra-orbital notch designed for the artery and nerve of the same name (Fig. 302, *i*, Fig. 349, *is*). At the lower margin of the orbit there is a canal (the infra-orbital canal; Fig. 349, *ci*) for the infra-orbital artery and nerve, and this opens upon the cheek—by means of the infra-orbital foramen (*fi*, Fig. 349)—about 4 mm. beneath the orbital margin. This point and the supra-orbital notch are of practical importance.

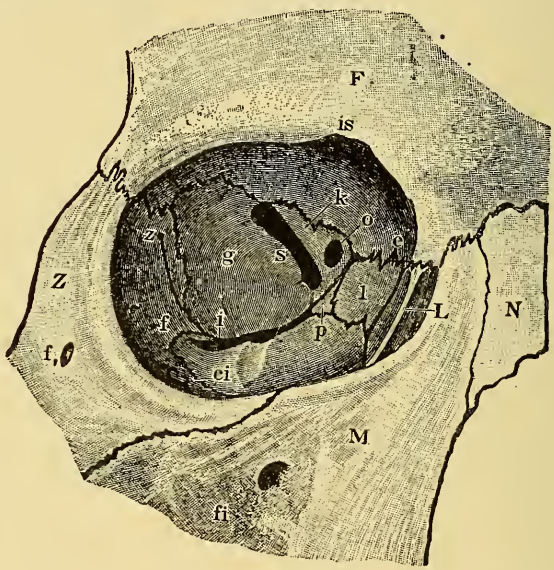


Fig. 349.—Wall of the Right Bony Orbit. (After Zuckerkandel.)

The wall of the orbit is formed on the outside by the malar bone, *Z*, and the greater wing of the sphenoid, *g;* above by the frontal bone, *F*, and the small wing of the sphenoid, *k*, which encloses the foramen opticum, *o*, and, together with the greater wing of the sphenoid, bounds the superior orbital fissure, *s;* on the inner side by the frontal process of the superior maxilla, *M*, the lachrymal bone, *L*, and the lamina papyracea of the ethmoid bone, *l;* below by the superior maxilla, *M*, and the orbital process of the palatal bone, *p*. Between the greater wing of the sphenoid and the superior maxilla lies the inferior orbital fissure, *i*, from which runs the sulcus infra-orbitalis, *ci*, which has its exit as the infra-orbital foramen, *fi*, on the anterior surface of the superior maxilla; *is*, supra-orbital notch; *f*, inner and, f_1, outer orifice of the zygomatico-facial canal; *z*, inner orifice of the zygomatico-temporal canal; *e*, anterior ethmoid foramen; *N*, nasal bone.

as constituting the points of exit of the aforesaid nerves. Sensitiveness to pressure at these spots is a frequent symptom in neuralgiæ of the trigeminus and also in essential blepharospasm.

The orbit is surrounded by several other cavities in disease of which it can itself be implicated. These cavities are the nasal fossæ and the cavities accessory to them—namely, the frontal sinus, the antrum of Highmore, the sphenoidal sinus, and the ethmoid cells.

709. Contents.—The contents of the orbit consist of the eyeball with the optic nerve and the muscles, the lachrymal gland, the vessels, and the nerves. The interstices between these structures are filled with orbital fat,

and the whole is maintained in a state of firm connection by a system of *fasciæ*. The latter display a greater strength and a more intimate union with one another in three places, viz.: 1. Along the walls of the orbit. They cover the latter under the form of a periosteum (here called periorbita), and likewise make a sort of anterior wall for the orbit. This anterior wall is formed by the fascia (fascia tarso-orbitalis) that starts from the margin of the orbit and is attached to both tarsi and also to the ligamentum canthi internum and externum. These structures combined represent the septum orbitale, which, when the lids are closed, shuts off the orbit anteriorly and keeps its contents in (Fig. 285). 2. The ocular muscles are surrounded by fasciæ which send out processes connecting the muscles with each other, with the lids, and with the margins of the orbit (see page 714). 3. Surrounding the eyeball the fasciæ are condensed into a fibrous capsule, the *fascia bulbi* (also called *Tenon's* or *Bonnet's capsule*). This extends forward as far as the conjunctiva of the eyeball and backward nearly to the optic nerve. It is thus open in front and behind, and may be said to represent a broad ring placed about the eyeball. It forms the articular socket for the eyeball, which can move in it freely in all directions. The contiguous surfaces of Tenon's capsule and of the eyeball are smooth, and are provided with an endothelial covering (Schwalbe). The intervening space, Tenon's space (*t*, Fig. 1), must be regarded as a lymph space[1] which is continuous posteriorly with the lymph space (supravaginal space, *s*, Fig. 1) surrounding the external sheath of the optic nerve. At the points where the tendons of the ocular muscles pierce Tenon's capsule, the latter is reflected upon the muscles and becomes continuous with the fasciæ covering them (lateral invaginations of the muscles, *e* and e_1, Fig. 1).

The *blood-vessels* of the orbit arise from the ophthalmic artery which springs from the internal carotid and enters the orbit through the optic foramen. The venous blood leaves the orbit through the superior and inferior ophthalmic veins, both of which make their way through the superior orbital fissure to the cavernous sinus, into which they empty. The above-mentioned veins form numerous anastomoses with the veins of the forehead.

Lymph vessels and lymphatic glands are wanting in the orbit.

The *nerves* of the orbit are motor—these being the nerves destined for the ocular muscles—sensory—these belonging to the first and second branch of the trigeminus—and sympathetic. To the outer side of the trunk of the optic nerve lies the ciliary ganglion. This contains motor fibers derived from the oculo-motor nerve (short root), and sensory fibers from the trigeminus (long root). The short ciliary nerves pass from the ciliary ganglion to the eye, through the posterior division of which they

[1] [This is denied by Langer.—D.]

enter the interior of the organ. The long ciliary nerves, which likewise enter the eyeball, do not arise from the ciliary ganglion, but come directly from the trigeminus (from the branch of it called the naso-ciliary nerve), and convey sympathetic fibers which originate from the plexus enveloping the carotid.

710. Position of the Eye in the Orbit.—This on an average is such that a straight-edge applied in a vertical direction to the upper and the lower margin of the orbit and pressed against them just comes into contact through the closed lids with the apex of the cornea, but does not sensibly compress the eye. Variations from this mean position very frequently occur: partly in consequence of individual differences in the formation of the face; partly, too, on account of changes in the quantity of orbital fat. When corpulence is on the increase, the eyes project farther from the orbit (goggle eyes); but when there is emaciation, they sink back into their sockets [hollow eyes].

The situation of the eyeball in the orbit not only varies in different men, but may also be unlike on the two sides in the same individual. This is combined with an asymmetrical formation of the face, which is frequently associated with a difference in the refraction of the eyes. In this case the difference in position may be only apparent, since the myopic eye is longer, and hence is more prominent, so that an exophthalmus is simulated.

711. Exophthalmus.—Pathological changes from the normal position occur chiefly in the way of a protrusion of the eyeball from the orbit—exophthalmus. On account of the great individual variations in the position of the eyeball small degrees of exophthalmus can be diagnosticated with certainty only when they are limited to one eye, so that, by making the comparison with the other eye, a guide to the diagnosis is secured. Higher degrees of exophthalmus attract our notice at once. The protrusion of the eyeball may advance so far that the lids are no longer able to keep the eye back in the orbit, and thus it prolapses in front of the lids—*luxatio bulbi*. The protrusion of the eyeball is either directed straight forward, or there is at the same time with this forward projection a lateral displacement of the globe of the eye.

Exophthalmus is caused either by an increase in the volume of the orbital tissue or by a diminution in the capacity of the orbit. The former is much the more frequent. But exophthalmus can also be produced by a diminution of the tone of the recti muscles, which draw the eye backward. This is the case in paralysis or in division (tenotomy) of them. [Such exophthalmus is sometimes apparent only (see § 886).]

The *consequences* of exophthalmus when it is of high degree are extremely disastrous to the eye: 1. The farther the eye comes forward, the more it pushes the lids apart. The palpebral fissure is therefore more widely open and more of the eyeball is visible in it than usual. In slight cases of

exophthalmus the dilatation of the palpebral fissure is often more conspicuous than the actual protrusion of the eyeball. The fact of the eyeball's being more exposed results in symptoms of irritation upon the part of the conjunctiva, such as redness of the ocular conjunctiva and epiphora. As the protrusion of the eyeball grows greater, the closure of the lids becomes imperfect (lagophthalmus) and then the cornea begins to suffer, because keratitis e lagophthalmo develops. This is the most dreaded sequel of exophthalmus, and one to which regard has primarily to be paid in the treatment (see page 687). 2. The pressure which the eyeball exerts upon the lids from behind leads to their eversion; ectropion of the lower lid develops. 3. The mobility of the eyeball diminishes in proportion as the protrusion increases, owing to the marked stretching of the recti muscles and of the optic nerve. 4. Vision is affected in various ways. In the cases in which, together with the protrusion of the eye, a lateral displacement of the organ is present, diplopia makes its appearance. Later on, the vision of the protruding eye may be abolished altogether by keratitis or by disease of the optic nerve. The latter, as long as the protrusion of the eye is but slight, is subject to no undue tension, its normal S-shaped curve (see page 604) being simply straightened out; it is not until the eye is caused to protrude pretty far that the optic nerve is put on the stretch. If this stretching takes place gradually, the nerve fibers often accommodate themselves to it in a wonderful fashion, so that they preserve their conductivity, and vision is maintained intact; but if the exophthalmus increases rapidly, the optic nerve owing to the traction soon loses its conducting power.

[A transient exophthalmus is produced by distention of the orbital blood-vessels e. g., under the stress of strong emotion (Arlt, Donders) or from the hyperæmia of menstruation (Cohn).—D.]

For measuring the degree of exophthalmus, instruments have been constructed, which are called *exophthalmometers* or statometers. [For these can be substituted a thin flat trial-frame so placed that the plane of the instrument is parallel with the frontal plane. Each eye in succession is made to sight an object straight ahead and while it is doing so, the distance is measured between the plane of the frame and the external angular process, also between the former and the apex of the cornea. A comparison of the measurements on the two sides will show how far each eye protrudes in front of its own orbit, and how far one is in advance of its fellow.—D.]

712. Enophthalmus.—The opposite condition to exophthalmus, namely, the recession of the eyeball into the orbit—enophthalmus—occurs:

1. In decrease of the orbital fat consequent upon extreme emaciation. In Asiatic cholera this condition develops within a few hours, owing to the enormous loss of water from the tissues (Von Graefe) 2. In diminution of the orbital contents due to operations in which a part of them is removed (e. g., in extirpation of an orbital tumor). 3. In paralysis of the sympathetic. 4. After injuries (*enophthalmus traumaticus*). In most cases of this kind the traumatism does not affect the eyeball itself, but the margin of the orbit. It is only exceptionally the case that the enophthalmus occurs right after

the injury. Usually it does not develop until after several weeks have elapsed. The anatomical changes causing it differ in the different cases. The most frequent condition is a cicatricial contraction of the orbital tissue or an indirect fracture of one of the orbital walls with depression of the fragment of bone outward so that the volume of the orbit is enlarged, and the eyeball is pushed back by the pressure of the external air. In one case the eyeball itself was luxated into the antrum of Highmore by a blow from a cow's horn. 5. In cases of intermittent exophthalmus (see page 822). 6. After the spontaneous subsidence of a pulsating exophthalmus (Bronner). 7. In neurotic atrophy of the face. 8. As a congenital anomaly of position. 9. In many cases of congenital paralysis of the external rectus the eye when adducted sinks back into the orbit [generally because the internus and an inextensible band replacing the externus pull on the eye like a pair of reins. See page 757].

I. Inflammations

(a) *Inflammations of the Bony Wall and of the Periosteum of the Orbit.*

713. Symptoms and Course.—Periostitis of the bones of the orbit is not rare, particularly at the orbital margin. Here, too, it is easiest to diagnosticate. A hard swelling is felt immovably attached to the bone and causing the margin of the orbit to appear thicker and misshapen—a fact which is particularly striking when comparison is made with the sharp margin of the orbit on the other side. In view of the great tendency toward œdematous swelling possessed by the lids and the conjunctiva, it is quite conceivable that such a swelling may be present to a greater extent upon one side than upon the other; still, it is usually easy to feel the tumefaction of the periosteum through the soft swelling of the lids. Moreover, the affected spot is distinguished by its greater sensitiveness to pressure.

If the periostitis is situated, not at the margin, but in the depth of the orbit, the diagnosis is much more difficult. We have at first simply the signs of a painful inflammation in the deeper parts of the orbit. That this inflammation starts from the periosteum is frequently not apparent until the periostitis leads to the formation of an abscess, and this breaks through to the outside, in which case we then come down upon the diseased bone with the sound.

The *course* of periostitis leads in favorable cases to complete resorption of the periosteal exudate or to the formation of a permanent deposit of bone (especially in syphilitic periostitis); it is more unfavorable when the periostitis goes on to suppuration which is followed by caries and necrosis of the bone.

When a periosteal abscess develops at the margin of the orbit, the skin over the affected spot first becomes reddened, then becomes thinned by the pus, and finally is perforated. A fistula is thus produced, through which the sound passes down to bare and roughened bone. Afterward there develops at the site of the fistula the funnel-shaped indrawn depression that is characteristic of bone disease. The discharge of pus from the fistula keeps up until all the diseased bone that has died has been eliminated, a

process for which not infrequently several years are required. Then the fistula heals and leaves an indrawn, funnel-shaped scar attached to the margin of the orbit. Through this can be felt the defect in the margin of the orbit left by the necrosis. Other consequences which frequently remain are ectropion of the affected lid and even lagophthalmus. These two conditions result partly because the lid becomes attached to the margin of the orbit and is drawn up strongly toward it, partly because a portion of the skin of the lid has been destroyed in consequence of the prolonged suppuration (Fig. 289).

When the periostitis which has gone on to the formation of an abscess is situated in the depth of the orbit, the disease runs its course with the symptoms of retrobulbar phlegmon, which will be described later on. The process is then much more severe and of longer duration since it takes a good while for the pus to make its way from the depth of the orbit to the surface. These deep suppurations, moreover, may be dangerous to life, if they are transmitted to the cranial cavity and give rise to meningitis or abscess of the brain. In this respect the periostitides of the roof of the orbit are particularly to be dreaded, because at this spot the cranial cavity is separated from the focus of pus in the orbit by only a very thin lamella of bone.

Chronic periostitis, particularly when syphilitic, may set up a gradually increasing thickening of the bones of the orbit, by which the orbital cavity is progressively contracted. The consequence is exophthalmus and compression of the nerves which enter the orbit, so that neuralgiæ and paralyses are produced. This complex of symptoms is like that seen in *leontiasis ossea*. This consists in a progressive thickening of the bones of the whole face, a process in which the bones of the orbit share, so that in this case, too, the symptoms of contraction of the orbit with simultaneous thickening of its walls are produced.

714. Etiology.—The causes of periostitis are : 1. Injuries. This traumatic periostitis is found most frequently at the margin of the orbit, because this is the part most exposed to injuries. 2. Dyscrasiæ, particularly scrofula (tuberculosis) and syphilis. These periostitides are likewise localized more frequently at the margin than in the depth of the orbit, because in this case, too, injuries play a part as exciting causes. Injuries of a comparatively trifling nature, such as bumps or falls upon the orbital margin, which in healthy persons would remain without further ill result, may in people affected with dyscrasiæ start up protracted specific inflammation. Scrofulous (tuberculous) periostitis occurs chiefly in children and principally affects the superior external and inferior external margins of the orbit, which are the parts most exposed to knocks; it leads, as a rule, to caries. Syphilitic periostitis, on the contrary, is ordinarily met with in adults and only exceptionally in children (those with hereditary syphilis). It belongs to the third (gummatous) stage of syphilis, and for the most part appears as a chronic affection under the form of periosteal thickening, more rarely

as an acute affection with suppuration following it. 3. Suppuration in the accessory cavities of the nose (page 825).

715. Treatment.—Treatment must, above all, have regard to the *etiological* factor. In this respect the best results are obtained in syphilitic periostitis, which usually subsides rapidly under a promptly initiated treatment with mercury and iodide of potassium. As *local* treatment moist hot compresses are applied, which in the beginning favor resolution, but in the later stages accelerate the softening of the abscess that is in process of development. As soon as there are signs that suppuration has taken place, there should be no delay about making the incision, so that the pus which has accumulated beneath the periosteum may not detach the latter still more extensively from the bone. Particularly in the case of deep-seated periosteal abscesses is early incision indicated, even though no fluctuation is yet perceptible, in order to prevent the transfer of the suppuration to the brain. After the abscess has been opened, a drainage tube or a strip of iodoform gauze is placed in the wound, so as to keep it open for the exit of the pus. If caries or necrosis succeeds periostitis, they are to be treated according to the general rules of surgery. Ectropion and lagophthalmus, which may develop subsequently, likewise call for relief by operation. This relief must be given without delay when the cornea is in danger because insufficiently covered. In other cases it is better to defer operative interference until the process in the bone has entirely healed, as otherwise the success of the lid operation might be jeopardized by the renewed formation of abscesses and fistulæ.

(b) Inflammations of the Cellular Tissue of the Orbit

716. Symptoms.—Inflammation of the orbital cellular tissue—*orbital cellulitis*—manifests itself by a marked œdema of the lids and conjunctiva (chemosis). An associated symptom and the most important of all is the protrusion of the eyeball, caused by the swelling of the tissues behind it. Because of this protrusion the eyeball's motility is impaired, and actual paralysis of the eye muscles may be superadded. The sight is often reduced or may even be entirely abolished owing to an inflammation of the optic nerve, which may be purely retrobulbar or may extend to the intra-ocular end of the nerve, in which case the ophthalmoscope shows an inflammation of the nerve-head. The gland behind the ear is swollen. At the same time there are violent pain, fever, and not infrequently also cerebral symptoms, such as headache, vomiting, stupor, retardation of the pulse, etc.

Cellulitis of the orbit may subside by absorption of the exudate that has been deposited, and in favorable cases everything may return to the normal. But often a slight degree of exophthalmus, or paralyses of the eye muscles, or atrophy of the optic nerve may be left. In other cases, however, the inflammation goes on to suppuration, the condition being then called *orbital phlegmon* (retrobulbar phlegmon, retrobulbar abscess). When the

symptoms have reached their acme, the skin of the lids at a certain spot grows red, then shows a yellow discoloration, and finally is perforated by a discharge of pus. After the discharge of the pus, which is present in large quantity, the inflammatory symptoms in most cases rapidly abate and the opening soon heals again. The sight may suffer permanent diminution or be altogether annihilated, if the optic nerve has been implicated either through inflammation or through thrombosis of its vessels. Detachment of the retina and even suppuration of the eyeball (panophthalmitis) also occasionally occur in retrobulbar phlegmon. If the suppuration is carried over from the orbit to the cranial cavity, it leads to a fatal issue through purulent meningitis or abscess of the brain.

717. Etiology.—Retrobulbar phlegmon may originate from the following *causes:* 1. Injuries, when the body causing the traumatism enters the orbit and carries infectious germs into the tissues. Those injuries in which a foreign body is left in the orbit are particularly dangerous. Operations, too, such as enucleation, may cause orbital suppuration if not performed aseptically. 2. Transfer of inflammation from the wall of the orbit or parts in the vicinity of the latter, such as the accessory cavities of the nose, to the orbital cellular tissue. 3. Erysipelas; the inflammation being transmitted from the skin to the deeper parts. 4. Metastases in pyæmia, typhoid fever, scarlet fever, smallpox, purulent meningitis, influenza, etc.

It is not always easy to determine the starting point of *retrobulbar cellulitis.* It often develops so suddenly and so without apparent cause that it is no wonder that the older physicians took refuge in the explanation which attributed it to the effect of cold. Recent discoveries have made us better acquainted with the sources of the orbital inflammation, which often are very obscure. The conditions most often causing inflammation of the orbit are empyemas of the cavities adjoining it, and in this regard the ethmoid cells are of more significance than the maxillary and frontal sinuses, since the former are separated from the orbit by a partition of bone no thicker than paper (cf. page 803 and Fig. 351). Hence in every orbital cellulitis not due to external injury we should always make a careful rhinoscopic examination of the nose.

In other cases there is a primary inflammation of the bony wall of the orbit and as the periosteal pus forces its way out from the deeper parts it sets up an orbital cellulitis. More difficult to recognize is the condition in which the diseased bone is situated far from the orbital cavity, as, for instance, in caries of the petrous bone or in suppurative periostitis of the alveolar process of the superior maxilla. The latter is the case in dental periostitis or after the extraction of diseased teeth. The path that the inflammation takes in these cases is either along the anterior surface of the upper jaw or through the maxillary antrum, in which the diseased tooth has already set up an empyema.

Orbital phlegmons may develop in pharyngitis and in suppurative parotitis by transmission of inflammation from behind forward; and by transmission from before backward in panophthalmitis, in abscess of the lids, and in dacryocystitis acuta, when, as exceptionally happens, perforation of the wall of the lachrymal sac takes place backward instead of forward.

Orbital phlegmons may lead to thrombosis of the cavernous sinus; although the converse process may also happen—i. e., the thrombosis starting from a thrombosed cavernous sinus may extend until abscess in the orbit is formed too.

718. Treatment.—When we have an orbital cellulitis to treat, we must first of all try as far as possible to remove its cause. Under this head belong the antiseptic treatment of wounds of the orbit, the provision for the free escape of secretion from wounds, the removal of foreign bodies from the orbit, the treatment of suppurating accessory cavities, such as the ethmoid cells, etc. The inflammation itself is combated by moist hot compresses, to which may be added the application of leeches to the temple, cathartics, diaphoresis, etc. If an abscess develops, the indication is to open it as early as possible in order to prevent the suppuration from spreading to the brain. We introduce a sharp-pointed scapel at the spot where we suppose the abscess to be, and do not hesitate to plunge it deep in. If the phlegmon starts from the bone or from the accessory cavities, the pus at the outset of the disease still lies beneath the periosteum. Then this must be split along the wall of the orbit and be stripped up until the pus is reached. The situation of the abscess is inferred from the way in which the eyeball is displaced. If, for instance, the latter is pushed forward and downward, the abscess is to be sought for in the upper part of the orbit. Even when we do not succeed in getting out any pus by our incision, because no abscess cavity of any size has yet formed, yet the relief of tension in the tissues produced by the incision and the profuse bleeding exert a favorable effect.

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719. Thrombosis of the Cavernous Sinus.—Symptoms similar to those which present themselves in the beginning of a retrobulbar phlegmon accompany *thrombosis of the cavernous sinus*. The lids and the conjunctiva swell up with œdema, and the eyeball protrudes and becomes difficult to move. The veins of the retina are seen, upon ophthalmoscopic examination, to be distended enormously. At the same time there is a doughy œdema in the mastoid region. These symptoms are referable to the fact that the veins of the orbit discharge the greater part of their blood through the ophthalmic veins into the cavernous sinus; hence thrombosis of the latter produces stasis in the veins of the orbit, and especially if the thrombotic process is continued on from the sinus into these veins. The stasis leads to protrusion of the eyeball and also to venous hyperæmia of the retina. The œdema of the mastoid region depends upon the fact that in this region an emissary vein of Santorini (the emissarium mastoideum) empties into the transverse sinus, and hence when the thrombosis is carried along from the cavernous to the transverse sinus, the mastoid region also shares in the venous stasis. When this œdema is present (which, to be sure, is not always the case), it forms an important diagnostic sign between sinus thrombosis and retrobulbar phlegmon in which latter it is absent. A further difference is that sinus thrombosis frequently passes over to the other side, so that the same complex of symptoms develops there also, while, on the contrary, a bilateral orbital cellulitis would be one of the greatest rarities. Finally, sinus thrombosis is associated with very severe cerebral symptoms, terminated at last by the onset of the fatal issue.

In occlusion of the sinus what we are dealing with is either a simple thrombosis produced by marasmus or a thrombosis due to infection. The latter usually originates from a focus of pus situated in the vicinity—e. g., from a phlegmon of the orbit, which gives rise to a thrombotic process in the superior or inferior ophthalmic vein, this process being then carried over into the cavernous sinus. Thrombosis of the sinus most

frequently originates in a caries of the petrous bone, the thrombosis extending from the sinus of the latter [the petrosal sinus] to the cavernous sinus. Suppurative processes starting from the posterior ethmoid cells, maxillary antrum, or buccal cavity (teeth or tonsils) also may lead to sinus thrombosis. Finally, thrombosis of the sinus may be set up by erysipelas and may also occur metastatically in pyæmia and infectious diseases.

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720. **Tenonitis.**—Tenon's capsule may be implicated in inflammation of the eyeball, so that an inflammatory œdema develops in the capsule itself and in the adjoining cellular tissue of the orbit, and the eyeball is thus pushed forward. Hence a slight degree of exophthalmus is sometimes found in severe cases of irido-cyclitis (particularly after injuries). This is quite regularly the case and to a much higher degree in panophthalmitis, in which exophthalmus is one of the most constant and striking symptoms. After the subsidence of the inflammation extensive adhesions are formed between Tenon's capsule and the eyeball—a fact of which we can convince ourselves if an enucleation is subsequently performed. Exudation into Tenon's space also occurs after it has been laid open by injuries, and particularly after squint operations, when infection of the wound has taken place from dirty instruments.

There is also a primary *serous tenonitis*. The symptoms of this rare disease can be best gathered from the description of the following case which I myself observed years ago: It concerned a woman of fifty-eight years of age, otherwise healthy, in whom the disease had begun, without known cause, six days before she came under my observation. I found the skin in the vicinity of the lids, and still more the lids themselves, very swollen and œdematous, so that the eyes could be opened to only a very small extent indeed by spontaneous effort. When I drew the lids apart I found the eye prominent and almost immovable. The conjunctiva of the lids was but moderately, that of the eyeball, on the other hand, quite markedly, injected, and the latter was so greatly swollen as to form a thick protuberance projecting from all sides over the cornea. The secretion was not increased. The cornea and also the deep portions of the eye were healthy, and vision was normal, except that there was diplopia due to the impaired mobility of the eyes. The disease was associated with moderate pain, and particularly with a sense of pressure and tension of the eyes. The treatment consisted in the administration of an infusion of jaborandi, in order to produce rapid absorption of the exudate by means of profuse diaphoresis. Under this treatment the œdema and the protrusion of the eye gradually went down, so that when the patient four weeks later was discharged from the clinic, the condition of the eyes had become once more perfectly normal.

The causes of tenonitis are obscure; gout, rheumatism, and refrigeration are the etiological factors that have been held accountable for it. Some cases (among them one case of primary purulent tenonitis) have been observed as a result of influenza. The disease tends to relapse, but leaves no permanent bad results behind.

Tenonitis like orbital cellulitis, is characterized both by œdema of the lid and the conjunctiva and by protrusion of the eyeball. The distinction between the two affections is to be made from the amount of protrusion of the eye compared with that of the chemosis. If the latter is very pronounced and the exophthalmus is slight, a tenonitis must be assumed to exist; while in retrobulbar cellulitis even when the protrusion of the eye is marked, the chemosis is often not very great and may even be confined to the area occupied by the interpalpebral fissure. Frequently, however, it is impossible to distinguish certainly between the two sets of cases, which in fact are not sharply differentiated even anatomically. I have no doubt that many cases which were formerly called tenonitis would now be regarded as orbital cellulitis.

II. Injuries

721. Injuries of the Soft Parts.—Injuries of the orbit affect either the soft parts alone or the bones as well. Injuries of the *soft parts* originate, as a rule, from the penetration of a foreign body into the orbit, the lids and the eyeball being, of course, very frequently implicated at the same time. The direct consequence of the injury is an extravasation of blood into the tissues of the orbit. If this is considerable it causes exophthalmus, and also, since the blood oozes slowly forward, it comes into view beneath the conjunctiva and the lids under the form of an ecchymosis.. Paralysis of the ocular muscles, too, may be produced by the injury, and so also may lesions of the optic nerve, the latter entailing as their direct result partial or complete blindness. It also happens sometimes that the eyeball is driven out of the orbit by the foreign body which has penetrated into the latter, and is thus found lying in front of the lids (*luxatio bulbi traumatica*). This is most apt to happen when the body that causes the injury enters from the outer side, since here the wall of the orbit recedes the farthest—about as far back, in fact, as the plane of the equator of the eyeball. There are parts of the world where such injuries are purposely inflicted in brawls, the eye being pried out of its socket by the thumb, which is thrust into the orbit from the outer side. Insane patients have sometimes enucleated one or both of their own eyes in this way. Ordinarily the luxated eyeball is lost, but cases are known in which after reposition the eye healed again in its place and retained its visual power.

722. Injuries of the Bones.—Injuries of the bone are most frequently produced by gun shot (especially a shot in the temple in attempted suicide) and the action of a contusing force (e. g., by a blow or a fall) upon the margin of the orbit. They are easy to diagnosticate when they affect the margin of the orbit itself. The site of a fracture in this locality is recognized by the unevenness, the sensitiveness to pressure, and in extreme cases by the crepitation.

Deep fractures of the orbit without fracture in the margin may be produced by deeply penetrating foreign bodies, but may also be caused apart from these and indirectly (by contre-coup). This is the case, for example, after a fall upon the head, the dropping of a heavy weight upon it, etc. Such fractures when deeply situated can only be suspected from the fact that they are accompained by orbital hæmorrhage, which manifests itself by a suddenly developing exophthalmus and the appearance later on of ecchymosis of the conjunctiva and the lids. A further diagnostic point would be supplied if directly after the injury partial or total blindness were determined to exist along with a normal appearance of the eyeball. This condition would necessarily be referred to an injury of the optic nerve in the canalis opticus, into the wall of which the fracture extends (Hölder and Berlin; see page 631). Similar symptoms also at times accompany fractures of the base of the skull, except that in this case the exophthalmus is wanting and the ecchymosis of the conjunctiva and lids sets in still later, since the blood takes a longer time to push its way so far forward.

Spontaneous hæmorrhages into the orbit are of extremely rare occurrence, tak-

ing place as a consequence of whooping-cough or in persons who in general are predisposed to hæmorrhages [e. g., in infantile scurvy, in which they produce exophthalmus (Stephenson).—D.]

723. Emphysema of the Orbit.—When in consequence of the fracture the orbit is made to communicate with the neighboring cavities, emphysema may develop in the orbit. Wherever the air has accumulated in the anterior portions of the orbit, it can be felt through the lids; but air that has come out behind the eyeball makes its presence manifest by exophthalmus. Whether such an exophthalmus is caused by exuded blood or by air can be determined by noting that in the latter case the protruded eye can be pushed back into the orbit again with the finger, since the air is displaced by the pressure. On the other hand, the exophthalmus is increased by straining during the acts of coughing, blowing the nose, etc., owing to the fact that fresh supplies of air are forced into the orbit.

Emphysema of the orbit and lids develops after a contusion affecting the eye. By the force that acts on it, the eyeball is driven back into the orbit, and the orbital fat is made to recede to one side. But it meets with a firm resistance in the wall of the orbit everywhere except on the nasal wall which is formed of the thin lamina papyracea. This is pushed in, and thus one of the ethmoid cells is put in communication with the cellular tissue of the orbit.[2] The mere presence of such a communication between the orbital tissue and a pneumatic cavity does not suffice to produce emphysema; to effect this, air must be driven through the fracture into the orbital tissue. Ordinarily this occurs from blowing the nose, in doing which strong expiration is made while the nasal orifice is kept shut and consequently the air in the nasal fossa is put under high pressure. Thus is explained the circumstances that emphysema often does not develop until several hours after the injury and then quite suddenly when the patient blows his nose. Violent blowing of the nose in some rare cases has produced emphysema even when there was no antecedent injury. This happens in men in whom the lamina papyracea has particularly thin spots which are forced open by the heightened pressure of the air.

The air which passes through the fracture in the lamina papyracea gets first into the orbital tissue (*orbital* emphysema). The eyeball is driven forward and so is the tarso-orbital fascia which is squeezed against the skin of the lids so that the lids in their whole extent are stretched tight like a drum and protrude, for which reason these cases are usually regarded as an emphysema of the lids. Owing to the entrance of air behind the septum orbitale (see page 653) the lids are squeezed together and the palpebral fissure is narrowed, while in exophthalmus of other kinds the lids are pressed apart by the eye. If the pressure under which the air enters the orbit is particularly high and in addition the tarso-orbital fascia is weak (as is the case in old age) the air may break through the fascia and then gets under the skin of the lids—*orbito-palpebral* emphysema. We can then feel the air beneath the thin skin of the lids much better than we can when it is beneath the tightly-stretched fascia. Moreover, the swelling extends to the free border of the lid, while in the other case it stops at the convex border of the tarsus where the fascia is attached (Heerfordt). A purely *palpebral* emphysema without implication of the orbit is extremely rare. It occurs when the point

[2] If the contusion has resulted in a rupture of the sclera, emphysema does not develop, because the eyeball being collapsed does not push the orbital fat aside with enough force to produce this result. Emphysema and rupture of the sclera, therefore, although both are consequences of a contusion affecting the eye, are mutually incompatible conditions.

at which the air enters the tissue lies in front of the septum orbitale, e. g., in case of injury of the lachrymal sac.

724. Results and Treatment of Injuries.—The injury, if no important organs have been destroyed, may heal after the resorption of the extravasated blood with a restitutio ad integrum. In other cases disturbances of motility of the eyeball remain, owing to its adhesion to the neighboring structures, or as a result of paralysis of the ocular muscles. Moreover, if the optic nerve has been injured, permanent blindness of the eye may ensue. Still worse are the cases in which the injury results in a phlegmon of the orbit, in which case it may even end fatally.

The *treatment* of a recent injury varies according to circumstances. If the wound is very lacerated and dirty, the indication is to remove the shreds of tissue that are likely to die and to cleanse the wound; furthermore, if a foreign body of any considerable size is lodged in the orbit, this must be removed at once, and the wound kept open with a drainage tube or strips of gauze. If, on the other hand, the lips of the wound are in good condition or have actually perhaps already united, we do not even wash them off, but simply place a dry sterile dressing over them. If in this case there is a foreign body in the orbit, we put off making any attempts at extracting it until we find whether or not it is going to remain without causing trouble. Projectiles, in particular, often do so remain. If there is a considerable hæmorrhage into the orbit or an emphysema, a pressure bandage contributes to the rapid absorption of the blood or the air. As soon as the symptoms of deep suppuration have set in, we must proceed as in the case of a retrobulbar phlegmon.

725.—**Contusions of the Eye.**—It may be of service to the general practitioner to give a brief, comprehensive presentation of all the consequences which contusion of the eye or of the adjoining parts may entail. The physician before whom a case of this kind comes will deduce from this summary the changes that may possibly be present. He will then look for these changes, and thus perhaps will arrive at the discovery of lesions of this sort which otherwise might have escaped him because they are not very conspicuous. The changes produced by contusion are:

In the *lids*, ecchymoses, emphysema, solutions of continuity.

In the *margin of the orbit*, fractures with or without displacement of the fragments.

Changes in the *position of the eyeball* including exophthalmus which may be caused by the effusion of blood or air (emphysema) into the retrobulbar tissue or by the formation of an arterio-venous aneurysm due to rupture of the carotid into the cavernous sinus page 822). [Enophthalmus may also occur (page 807).—D.]

The *tension* of the eyeball is greatly reduced when a perforation of the tunics of the eye has taken place. But the tension may be found to be temporarily diminished even apart from any gross material lesion, this alteration being then due to a decrease in the fluids of the eye and particularly of the vitreous (Leplat), which again must be regarded as a consequence of a disturbance of nutrition dependent upon nervous influence [cf. page 520].

In the *conjunctiva*, ecchymoses, lacerations (with or without coincident scleral rupture).

In the *cornea*, epithelial desquamation, and, later on, deep non-suppurative inflammations; rarely purulent processes or rupture of the cornea.

In the *aqueous chamber* and the *vitreous*, effusion of blood (hyphæma hæmophthalmus).

In the *iris*, iridodialysis partial or complete (aniridia traumatica), radial lacerations, recession of the iris, likewise paralysis of the iris (mydriasis) with or without paralysis of accommodation.

In the *lens*, astigmatism, subluxation, and luxation due to partial or complete laceration of the zonula, likewise the formation of cataract.

In the *sclera*, rupture in the anterior division, attended, it may be with prolapse of the uvea, the lens, or the vitreous.

In the *chorioid* and *retina* extravasations of blood, detachment, rupture; in the retina alone, cloudiness (commotio retinæ), formation of a hole in the fovea.

In the *optic nerve*, compression by effusion of blood, and contusion or rupture by fracture taking place in the optic canal; avulsion of the papilla from the scleral canal.

III. Basedow's Disease

726. Symptoms.—Basedow's disease (*exophthalmic goitre*) belongs to the domain of internal medicine, and hence can be considered here only in so far as *exophthalmus* belongs among its most important symptoms. This exophthalmus is bilateral; the eyes are pushed straight forward, sometimes but little, sometimes to such a great extent that they can not any longer be perfectly covered by the lids. Even when the exophthalmus is great, there is little or no limitation of mobility of the eye. It is obvious that when the exophthalmus is considerable, it becomes noticeable at the first glance; but even when the protrusion is slight the peculiar appearance of the eyes strikes one at once. This appearance is caused by the fact that the upper lids are raised unusually high. The eyes look as if forced wide open, and give the patient an expression of astonishment or fear (Dalrymple's sign). When the eyes are depressed the upper lids do not descend properly with the eyeball, but remain elevated, so that a broad portion of the sclera is visible above the cornea (Von Graefe's symptom). Winking takes place less frequently (Stellwag's symptom), and hence desiccation of the deficiently covered cornea is favored. Convergence of the eyes is rendered difficult (Moebius' symptom). [Eversion of the upper lid is sometimes difficult (Gifford).—D.] The eyeball itself, as long as the cornea has not yet suffered harm, is normal and the visual power is good. Sometimes there is pulsation of the retinal arteries. [When a stethoscope is placed over the closed lids, there is occasionally heard, besides the constant hum due to contraction of the orbicularis, a murmur, said to be systolic in rhythm and hence regarded as vascular in origin (Snellen and others). But according to Sattler it, too, is due to muscular contraction.—D.]

The two other main symptoms of Basedow's disease are the *swelling of the thyroid gland* and the acceleration of the heart's action (*tachycardia*). The former is distinguished from ordinary goitre by the fact that when the hand is placed upon the thyroid gland it feels the strong movement of

pulsation in the arterial vessels, which is communicated to the entire gland. So also the carotids are found to be dilated and strongly pulsating. The intensity of the heart beat is increased, the frequency of the pulse is regularly over a hundred. The slightest bodily exertion or mental excitement at once increases the frequency of the pulse very considerably. The physical examination of the heart, apart from the enlargement of the left heart, gives a normal result. In patients with Basedow's disease there are almost always vibratory tremor and increased secretion of sweat. The general state of the patients is disordered to this extent that they are for the most part very excitable, and suffer from the symptoms of anæmia or chlorosis. Not infrequently rapid emaciation is present, even when the appetite remains good.

Basedow's disease was first described as a special form of disease by English physicians, and particularly by Parry and afterward by Graves, and hence it is even now called *Graves's disease* by the English. These authors, however, had not recognized the exophthalmus as being one of the essential symptoms of the disease; this was first done by Basedow, who in 1840 did the pioneer work in demonstrating the whole triad of symptoms of the disease.

In marked cases no disease is easier to diagnosticate than this; even from a distance we can tell what the patient's trouble is. But, on the other hand, there are numerous cases in which some of the symptoms are less pronounced or are even wanting altogether, so that the diagnosis becomes difficult. Taking only the exophthalmus into account, we find that it may be slight, absent altogether, or confined to one eye. When exophthalmus is absent, Von Graefe's symptom is still sometimes present, so that it is not to be regarded as due simply to the exophthalmus; but it too is not constant, and in the same case may be sometimes present, sometimes wanting.

727. Etiology.—The disease principally attacks women, beginning at the time of puberty, and extending up to the appearance of the menopause. Men rarely suffer from it.

The *cause* of Basedow's disease is assumed to be a poisoning of the body by the secretion of the thyroid gland, the result of which is that a disturbance of innervation is set up. So far as the eye is concerned, it is the sympathetic whose function appears to be disturbed. In consequence we find a dilatation of the vessels in the district supplied by the carotids—a dilatation which is manifest even to external observation in the pulsation of the carotids. It is owing to this distention of the arterial vessels in the thyroid gland and the orbit that the goitre and the exophthalmus develop, and both conditions therefore disappear at death. Landström gives another explanation of the exophthalmus. According to him, there are smooth muscle fibers in the orbit running from the region of the equator of the eyeball forward to the septum orbitale. Since these muscle fibers like the musculus tarsalis are thrown into tonic contraction, they draw the eyeball forward. Von Graefe's symptom, too, must be referred to a disturbed innervation of the sympathetic, which supplies the organic levator of the lid (musculus tarsalis superior). This is in a state of tonic contraction, as we convince ourselves if we grasp the upper lid by the cilia and try to draw it down. We then encounter greater resistance than we do in healthy persons. [Gifford's sign (page 817) is probably referable to the same cause.—D.]

728. Course and Treatment.—Basedow's disease generally develops quite gradually. Palpitation usually sets in first, and with this is afterward

associated the thyroid dilatation, and, last of all, the exophthalmus. As a general thing it takes months or even years before all the symptoms of the disease are distinctly marked. Then it usually remains at the same point for years, after which it subsides again very gradually and not without leaving a tendency to subsequent recurrences. In many cases, however, the disease is not recovered from, but remains until the patient's death, nay, more, it may even—by the exhaustion or the complications which it produces—be itself the cause of death. As a general thing the disease runs a severer course in men and in elderly people than in women and in the young. Basedow's disease is a source of danger to the eyes from the fact that when the exophthalmus is of a pretty high degree closure of the lids is imperfectly performed and keratitis e lagophthalmo develops. In this way blindness of one or even of both eyes may be produced.

Precisely as the separate symptoms of Basedow's disease may show great variations in the extent of their development, so also may the course of the disease vary exceedingly. Although as a rule this is very chronic, yet cases are known in which the disease had a most acute onset. Trousseau tells of a woman in whom the symptoms of the disease developed in the course of a single night, which she had spent in tears, lamenting her father's death. The subsequent progress of the disease, too, may be so speedy that within a few weeks it leads to recovery or to death.

Treatment need be considered here only in so far as it relates to the eyes. The exophthalmus requires treatment only when it leads to imperfect closure of the lids and thus endangers the cornea. In this case we must, by bandaging the eyes at night, provide for their being covered during sleep. If the bandage turns out to be insufficient, we must perform tarsorrhaphy, by which the palpebral fissure is permanently closed in its outer portion.

IV. Tumors of the Orbit

729. Tumors of the orbit—in the widest sense of the word—have in common one very important symptom, namely, exophthalmus. If this is present the main thing to determine is whether an inflammation or tumor growth causes it. Such a determination is easy, as a rule, since inflammatory exophthalmus starts up acutely and with inflammatory symptoms (swelling of the lids and conjunctiva, pain), while exophthalmus due to tumor growth develops gradually and without signs of irritation. Yet not infrequently diagnostic errors do occur especially in chronic inflammations attended with tissue growth (syphilitic or tuberculous inflammations—cf. page 824).

To determine the site of the tumor, we must consider the nature of the protrusion: whether it is directed straight forward, or whether there is also a lateral displacement of the eyeball. With the same object in view we test the mobility of the eye in all directions. Then we try to palpate the tumor itself, so as to be able to tell its size, form, consistency, mobility, etc. If the tumor lies deep in the orbit, we endeavor to push the little finger as deeply as possible between the margin of the orbit and the eyeball (under narcosis,

if necessary), so as to get at the tumor. Finally, the examination should be completed by determining the visual acuity and the ophthalmoscopic finding, by which we ascertain whether and in what way the optic nerve has been injured by the tumor. [Tumors of the orbit include the rarely occurring tumors of the lachyrymal gland (page 710) These do not in general cause exophthalmus, but a swelling perceptible with the finger under the upper outer margin of the orbit. Sometimes the gland is dislocated downward so as to appear as a swelling under the upper retrotarsal fold.—D.]

730. Cysts.—The most frequent of these are the *dermoid* cysts, which are congenital, but often develop to a greater size after birth. They generally lie in the anterior part of the orbit, and usually in its upper and outer or upper and inner angle. On account of their superficial situation, they do not displace the eyeball, but push forward the skin of the lids, through which they can readily be felt as round, movable tumors as big as a bean or walnut. Upon extirpating them one can convince himself that they are, as a general thing, unilocular cysts with pultaceous or sebiform contents; sometimes processes from them extend pretty deep into the orbit, and thus render complete removal difficult. The only harm that dermoid cysts produce is the disfigurement caused by them, and this, too, is the reason why, quite often, we proceed to extirpate them. In doing this, we must go to work very carefully so as to dissect out the cysts, as far as may be, unopened. If the cyst wall, which often is thin, breaks prematurely, part may easily be left behind and cause recurrences.

Fig. 350.—Cephalocele Orbitæ Anterior.

Histological examination of dermoid cysts shows that their wall possesses essentially the structure of the external skin—that is, it consists of a substratum of connective tissue, the cutis, which supports an epithelial lining like that of the external skin, and which not infrequently contains hair follicles and glands (sebaceous and sweat glands). The contents of the cyst are mostly like porridge or sebum, and are formed of the exfoliated epithelial cells and of the secretion of the glands contained in its walls; in many cases hair, and in very rare cases teeth, have been found in them. Sometimes the contents of the cyst are converted into an oily or honey-like liquid (oil cysts and honey cysts or meliceris); or they may even become like serum. The anatomical structure of these cysts ranges them in the category of dermoid cysts—i. e., of those which we regard as originating from an invagination of the external germinal layer, which then develops afterward into a cyst (Remak). Some of the cysts with serous contents may perhaps have originated from an analogous invagination of the nasal mucous membrane (Panas). In structure and mode of development dermoid cysts

are allied to the dermoids of the corneal margin, which likewise are to be regarded as aberrant islands of skin (see page 223). The two varieties of tumor are distinguished from each other clinically by the fact that the first are deeply placed cavities; the second, superficially situated, flat, expanded, wart-like structures.

Another form of tumor with which dermoid cysts might be occasionally confounded is the *hernia cerebri* (cephalocele). That form of it which requires consideration here, the cephalocele orbitæ anterior, projects into the orbit between the ethmoid and frontal bones (Fig. 350). At this spot the dura is wanting in the space occupied by the hernia, being adherent to the periosteum at the margin of the gap in the bone. The sac protruding from the gap is formed of arachnoid and pia mater, and contains remains of brain substance or, when the ventricle extends as far as the sac, is lined with ciliated epithelium (Stadfelt). Clinically, therefore, the cephalocele of the orbit usually represents a tumor which is situated in the upper and inner angle of the orbit, is covered by normal skin, is distinctly fluctuant, and has existed since birth. Since dermoid cysts also are congenital and frequently occupy the same spot, an error in diagnosis might be readily made. Such an error might possibly lead to the worst consequences, if extirpation of the meningocele, which may be followed by meningitis, should be thereupon performed. It is hence important to know how such a mistake can be avoided. The signs which principally distinguish a cephalocele from the dermoid cysts are as follows: 1. A meningocele is immovably attached to the bone. Not infrequently we are able to feel with our finger the opening in the bone through which the cephalocele communicates with the cranial cavity (hernial orifice). 2. A cephalocele sometimes shows the pulsatory and respiratory oscillations which are communicated to it from the brain. 3. A cephalocele can be diminished in size by pressure with the fingers, since its fluid contents are in part pushed back into the cranial cavity. At the same time symptoms of increased cerebral pressure, like vertigo, nausea, deviation of the eyes, convulsions, etc., may make their appearance. 4. In order to be perfectly certain, we make an exploratory puncture of the cyst. In doing this we must proceed under rigid aseptic precautions, so as not to excite inflammation of the cyst and consequent meningitis. The diagnosis becomes more difficult, or is even impossible, when the communication between the meningocele and the cranial cavity (the subdural space) is obliterated; but in this case removal of the tumor is not associated with danger of any sort.

Besides those already given, the only *cysts* of the orbit requiring mention are those formed by *entozoa* (cysticercus and echinococcus), and also the *congenital cysts* of the lower lid in microphthalmus (page 482) and *blood-cysts* developing from extravasations of blood.

731. Vascular Tumors.—To vascular tumors, in the widest sense, belong vascular dilatations (aneurysms) and new growths consisting of vessels (angiomata). Both the one and the other occur, though rarely, in the orbit. Of hæmangiomata we here meet with two forms that also occur in the lids (*telangiectasis* and cavernous tumor). The former is congenital, and is originally seated in the lids, from which it may gradually extend into the orbit. The diagnosis, hence, is easy, since on the lids the tumor is obvious. Its treatment when in the orbit is the same as when on the lids. *Cavernous tumors*, in contradistinction to those just named, usually develop first within the orbit and grow slowly, pushing the eye farther and farther before them. As long as they lie wholly in the depth of the orbit, the correct diagnosis can be made only from the variable volume which these tumors possess. We can diminish their size by pressing the eye back into the orbit, and, on the

other hand, they swell in the acts of crying, straining, etc. [or when the patient bends far over. They may also enlarge during the menstrual period (Birch-Hirschfeld).—D]. When the vascular tumors grow big and extend farther forward they gleam with bluish luster through the skin of the lids, and dilated blood-vessels appear in the latter; in this case the diagnosis is, of course, easy. When we see that these tumors are endangering the eye by causing its progressive protrusion, we must remove them. Extirpation with the knife is principally adapted to those cases in which the tumor is sharply limited and is inclosed in a fibrous capsule; for the case of a more diffuse vascular tumor electrolytic treatment is indicated (see § 826).

732. **Pulsating Exophthalmus.**—Under this term is denoted the following complex of symptoms: The eye is protruded; the blood-vessels of the conjunctiva and the lids, and often of the surrounding parts, too, are dilated. If the hand is placed upon the tumor, distinct pulsation of the eyeball itself and of the surrounding parts is felt; and, if the ear is applied to it, blowing murmurs and a continuous whirring and rumbling sound are heard. The patient also hears the same sounds; he has a constant rumbling in his head, as if he were standing near a waterfall, and he is often more disturbed by this than by anything else. The eye can be pushed back into the orbit with the hand. A special feature distinguishing the disease is the fact that compression of the carotid of the same side as the exophthalmus diminishes both the pulsation and the sounds or causes them to disappear altogether. The visual power of the eye is in many cases abolished, and that, as the ophthalmoscope shows, by optic neuritis; a conspicuous feature that is brought to light at the same time being the enormous dilatation of the retinal vessels. At times there are violent pains in the orbit and impairment of the hearing.

It has been proved by a number of autopsies that the most frequent cause of this complex of symptoms is an arterio-venous aneurysm resulting from rupture of the carotid into the cavernous sinus (cf. Fig. 310). Through the spot where this rupture has taken place the blood of the carotid is discharged under a high pressure into the cavernous sinus and the veins of the orbit, which empty into it, so that these veins are very greatly dilated and are set pulsating. The rupture of the carotid is most frequently caused by traumatism, and particularly by severe injuries of the skull with fracture of the base; rarely a spontaneous rupture occurs from degeneration of the vessel wall.

In rare cases the aneurysm subsides spontaneously; otherwise it persists, and may produce death with cerebral symptoms or by hæmorrhage from the dilated vessels. The treatment is self-evident, from the fact that the symptoms disappear as soon as the carotid is compressed upon the affected side. Hence we try first compression by digital or instrumental pressure upon the carotid every day for as long a time as can be borne. If this procedure, after being continued for some time, is unsuccessful, ligation of the carotid is indicated—an operation by which most cases are cured. For some cases a suitable procedure is direct ligation of the dilated veins in the orbit, after access to them has been secured by temporary resection of the malar bone (see § 896).

There are cases of *intermittent exophthalmus*, which appear only at intervals, and in fact mainly when the head is depressed, while in the erect position some enophthalmus may be present. As in such cases the exophthalmus increases when pressure is made upon the jugular vein, and as sometimes dilated veins are visible even upon external examination, it is assumed that the exophthalmus is caused by varicose veins in the orbit, which, however, in contradistinction to what occurs in pulsating exophthalmus, are not in communication with an artery.

733. **Other Tumors.**—Besides the tumors enumerated above the following have

been observed in the orbit, as rarities: Teratoma, angioma lipomatodes, lymphangioma, neuroma simplex, neuroma plexiforme, cylindroma, endothelioma, psammoma, enchondroma, and osteoma. The *osteomata* start from the bony wall of the orbit, and most often from the frontal bone. They may, however, also [and, according to some most frequently] develop in one of the cavities adjoining the orbit, particularly the frontal sinus, and enter the orbit itself afterward through erosion of the orbital wall. They are usually sessile, being attached to the bone by a broad base, but rarely are pediculated, in which case it occasionally happens that they necrose spontaneously and are then eliminated. Most osteomata are as hard as ivory, so that chisel and saw can make scarcely any impression on them (exostosis eburnea); but there are also osteomata of spongy or partly cartilaginous structure. They grow very slowly, and in time displace the eye from the orbit and destroy the sight by compressing the optic nerve. [They frequently also cause death.] For this reason they call for removal by operation, which because of the great hardness of the growth is often very difficult, and, moreover, is not devoid of danger, since in its performance the cranial cavity must be opened. Hence we often abstain from taking the growth out radically, and satisfy ourselves with removing only so much of it as projects into the orbit. When the eye has been pushed out of the orbit and rendered blind by a large osteoma, it is sometimes better to relieve the patient of his troubles by enucleating the useless eye, rather than expose him to the dangers of an extirpation of the growth.

Usually bilateral are the lymphomatous tumors of the orbit, the lymphomata and lymphosarcomata, the leuchæmic and pseudoleuchæmic tumors, and the chloromata. An enlargement of the lachrymal and parotid glands on both sides is called *Mikulicz's disease*. Here, too, it is often lymphomatous tumors that are present but, in other cases, chronic inflammatory processes possibly of tuberculous nature.

734. Malignant Tumors.—The most common primary tumors of the orbit are sarcomata. These may start from bone, periosteum, orbital muscles or connective tissue, the lachrymal gland, and even the optic nerve and its sheaths. Orbital sarcomata are usually rounded, pretty soft, and sharply defined, because they are inclosed in an envelope of connective tissue. Primary carcinomata of the orbit are very rare; they spring from the lachrymal gland.

The secondary formation of tumors in the orbit occurs when tumors of the eye (sarcomata, gliomata) perforate posteriorly into the orbit. So too, carcinomata of the lids or conjunctiva, if not removed early, grow into the orbit. Neoplasms may also grow into the orbit from the neighboring accessory nasal sinuses or even from the cranial cavity. Finally metastases of malignant tumors occur—rarely, however—in the orbit.

If malignant tumors are not removed early, they push the eye more and more out of the orbit, afterward destroy it, and finally fill the entire orbit, from whose anterior opening they project as large, ulcerated, readily bleeding masses. Still later they pass over to structures adjacent to the orbit, and especially to the brain; the neighboring lymphatic glands swell up, and metastases form in the internal organs. The patient succumbs from exhaustion or from a transmission of the growth to vital organs. To this course a stop can be put only by as early and as radical a removal of the growth as possible. Small encapsulated sarcomata can be cleanly enucleated with preservation of the rest of the contents of the orbit. Large

tumors, particularly if they are not sharply circumscribed, demand the removal of the entire contents of the orbit in doing which the eye, even when it still retains its ability to see, must be sacrificed. (For methods of operating see § § 896, 897.)

735. **Inflammatory Pseudoplasms.**—Classed under the name of pseudo-tumors [pseudoplasms] are the cases in which from the symptoms a tumor is assumed to be present in the orbit, and yet on the operating table we find that we are dealing with a chronic inflammatory growth. This may be of tuberculous, syphilitic, or unknown etiology. It is not always possible to differentiate certainly such cases from neoplasms before operation.

V. Disease of the Cavities Adjoining the Orbit

736. Character.—The accessory cavities of the nose, namely the maxillary antrum, the frontal sinus, the ethmoid cells, and the sphenoid sinus, may implicate in their diseases the orbit and the optic nerve. This is most apt to occur in disease emanating from the ethmoid cells, for the latter are separated from the orbit only by the thin lamina papyracea which, moreover, sometimes has gaps in it. The most posterior ethmoid cell not infrequently extends into the small wing of the sphenoid and then comes into closer relation with the optic nerve because it now borders on the optic canal; and it may even extend so far beyond the median line that it actually adjoins the optic canal of the opposite side. Otherwise the wall of the optic canal is formed in part by the sphenoid sinus (Fig. 351, *vk*) and involvement of this not infrequently affects the eye.

Inflammatory infections of the above named cavities, called by the name of *sinusitis* [or more properly sinuitis] are generally the result of an inflammation of the nasal mucous membrane. This may be either a catarrhal inflammation (coryza) or produced by infectious diseases, whether acute (the most frequent example of this being influenza) or chronic (tuberculosis, syphilis). Furthermore, injuries, new growths, and, in the case of the maxillary antrum, diseased teeth may cause the inflammation. The inflammation is associated with purulent secretion or empyema (called open empyema if the secretion can escape through the natural orifice of the cavity, closed empyema if the ostium of the cavity is shut off). In the latter case the secretion accumulates in the cavity and distends it; at the same time the contents of the cavity which at first are purulent gradually become mucous and at length watery (dropsy of the cavity).

Inflammation of the accessory cavities often produces *symptoms* which are very striking. If for instance, after a coryza there is marked frontal headache and the forehead above the eyebrows is sensitive to percussion, or the skin in this region actually becomes somewhat swollen, we should at once think of an inflammation of the frontal sinus. But often the inflammation of the accessory cavities runs a course which is pretty much void of symptoms, and particularly so when it is one of the deep seated cavities that is concerned. Hence a diagnosis to be certain always requires a rhinoscopic examination. This is not always by any means a simple matter, and often can not be perfectly performed until after some preliminary operative procedure (removal of some portions of the turbinal bodies, opening of the wall of the sinuses).

737. Transfer to Orbit.—The inflammation of the cavities may at any stage pass over to the orbit. In the *acute* stage an orbital cellulitis (see page 810) most frequently develops.

With marked headache and some fever the lids and the conjunctiva swell up, the eye protrudes and becomes less movable, and frequently there is added a paralysis of the eye muscles or an inflammation of the optic nerve. These threatening symptoms soon abate, but the paralysis of the eye muscles or the inflammatory atrophy of the optic nerve may remain permanently. A rarer sequel of such accumulations of pus in the accessory sinuses is suppuration in the orbit occurring under the guise either of a subperiosteal abscess or of a retrobulbar phlegmon with its destructive consequences.

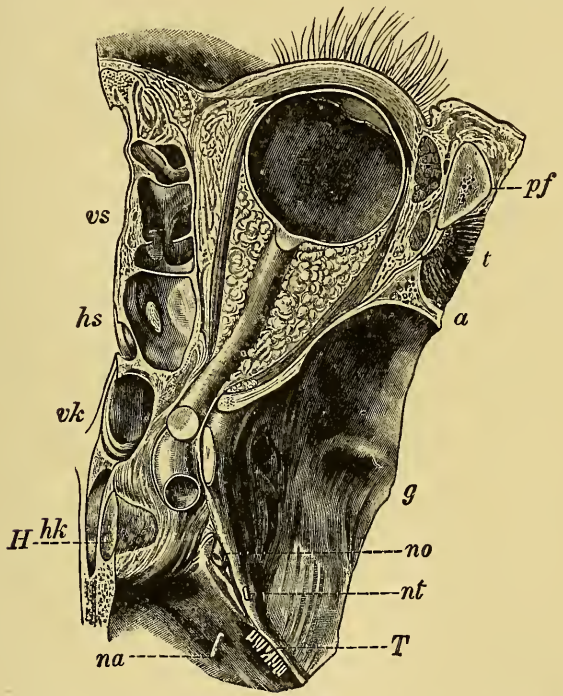


FIG. 351.—HORIZONTAL SECTION THROUGH THE RIGHT ORBIT.

Adjoining the internal wall of the orbit and separated from it by the lamina papyracea are the cells of the anterior and posterior labyrinth of the ethmoid bone, *vs* and *hs*. Right behind this is the cavity of the sphenoid bone, which in this case is divided into two segments by a projecting ridge of bone. The anterior division, *vk*, adjoins the optic canal at the inner side of the latter, and also extends beneath it. Over the posterior division, *hk*, lies the hypophysis, *H*, which fills up the sella turcica, and upon which rests the chiasm, which in this preparation has been removed along with the brain. At the outer side of the orbit, between the eyeball and the frontal process of the zygoma, *pf*, are two lobes of the lachrymal gland, the lower border of which was just met by the section. Behind the frontal process of the zygoma in the temporal fossa is the cross section of the temporal muscle, *t*, and behind this the cross section of the great wing of the sphenoid, *a*, succeeding which is the middle fossa of the skull, *g*. At the internal and posterior border of the latter the tentorium was detached along its insertion when the brain was taken out. Along the site of this insertion the nerves of the eye muscles pass through the dura, the furthest forward being the oculomotorius, *no*, while the trochlearis, *nt*, is somewhat further back. To the inner side of the latter and hence within the posterior fossa of the skull, the abducens, *na*, goes down into the dura. Furthest back is the trunk of the trigeminus, *T*, passing from the cranial cavity through a slit, which lies between the upper edge of the pyramid of the temporal bone and the attachment of the tentorium. The optic nerve where it passes from the optic canal into the cranial cavity has been cut off so that the internal carotid which lies beneath it is visible.

Chronic empyemata of the accessory cavities may produce sluggish inflammations of the orbital structures, and, since such empyemata often run their course without causing any symptoms, the diagnosis sometimes encounters great difficulties, as an example adduced below shows.

Dropsy of the accessory cavities is not so apt to produce inflammation

as it is to cause by distention of the walls of the cavities an encroachment on the orbit and hence a displacement of the eyeball.

Thus in dilatation of the frontal sinus we see the frontal bone protruding above the margin of the orbit and find the eyeball displaced forward and downward. In distention of the ethmoid cells we get a protrusion of the inner wall of the orbit upon which we can feel through the soft parts the thin lamina papyracea crepitate under the pressure of the fingers. Inflammation of the sphenoid sinus may manifest itself early in its course by disease (inflammation or atrophy) of the optic nerve. [Involvement of the optic nerve from sinus disease may show itself first by an enlargement of the *blind spot*.—D.]

[In not a few cases disease of the optic nerve and uveitis may be produced not by pressure but by absorption of *toxic* matter from a chronically inflamed sinus. In such cases we may find the causal organism in the blood, or complement-fixation tests may indicate its activity.—D.]

Dilatation of the accessory cavities may also be produced by new growths like polypi, osteomata, or malignant neoplasms.

738.—**Diagnosis.**—The following history shows on what points the diagnosis can be constructed—a diagnosis upon which in this case depended the proper treatment and as a result of this the prevention of blindness. A man forty years of age had become blind in the right eye. The ophthalmologist who treated him had made the diagnosis of neuritis of the optic nerve, which in spite of all therapeutic measures went on to atrophy with blindness. Five years later the patient consulted me because he had noticed for a year back a disturbance of sight in the left eye which had been hitherto sound. During the day he had a slight glimmering light before the eye, while in the evening he saw perfectly well. At the same time there was slight pain in the depth of the orbit and now and then attacks of left-sided supra-orbital neuralgia. As similar pains on the right side had preceded the blindness of the right eye and in fact sometimes manifested itself on that side still, the patient was in great apprehension that now he was going to lose the left eye too. The patient was otherwise healthy, and admitted having only a slight sensitiveness of the left supra-orbital region to percussion. The left eye appeared normal externally and with the ophthalmoscope, and also had normal vision; but by careful examination of the visual field with small red objects a very minute central scotoma could be discovered, in the confines of which the red color appeared to be less saturated. As chronic intoxication with tobacco or alcohol, which so frequently produces a central color scotoma, could be excluded in this case, a retrobulbar inflammation of the optic nerve (see page 623) had to be thought of as the probable cause. Indicative of the same thing was the pain in the depth of the orbit, which gave rise to the conjecture that in this situation there was a sluggish inflammatory process which was being transmitted to the optic nerve. A process of this sort perhaps had also been present on the right side six years before and had produced the blindness. What could be the cause of such a bilateral, deep seated, sluggish inflammation? In any case it could be of but slight intensity. Arguments for this were the slight degree of the pain, the slightness, considering the long duration of the trouble, of the injury to the left optic nerve, and above all the absence of an exophthalmus. Any marked inflammation of the tissue at the apex of the orbit would be associated with considerable swelling of the tissue and would hence produce exophthalmus. Since this was absent, severe inflammations such, for example, as would result from syphilitic or tuberculous disease of the bones or from new growths in the depth of the orbit, could be excluded. So by exclusion I arrived at the hypothesis that there was a chronic inflammation in the most posterior ethmoid cells or in the sphenoid sinus which might give rise to a permanent hyperæmia or slight inflammatory infiltration in its vicinity, that is, in the tissue at the apex of the orbit or in the optic canal. The rhinologist, who was called in, opened the most posterior ethmoid cells and the sphenoid sinus on

the left side. They were filled with a mucous membrane which had undergone very great degeneration with the formation of polypi and which upon examination showed a chronic inflammatory infiltration. After the operation the pain in the orbit and the neuralgic attacks gradually ceased and the central scotoma disappeared all except a slight remnant.

Of this history which is interesting from a rhinological aspect also, I will emphasize only those details which are important with regard to the eye lesion. 1. In the optic canal the dural sheath of the optic nerve is transformed into the periosteum of the bony canal. The optic nerve therefore in this situation is enveloped only by the delicate pial sheath which in most places is applied to the periosteum and on the upper side is actually adherent to it. This anatomical relation facilitates the direct transfer of inflammation from the cavities in the vicinity of the canal to the optic nerve itself. 2. This inflammation first affects the pial sheath of the nerve and we should therefore expect that the peripheral bundles of nerves which are next to this sheath would be first diseased. These bundles supply the periphery of the retina; but in this case it was on the contrary the center of the retina that became insensitive, for there was a central scotoma. This proved with certainty that there was a lesion of the papillo-macular bundle (see page 606), which in the canalicular segment of the nerve occupies just the middle of the nerve trunk, i. e., is the part farthest distant from the inflamed sheath. This paradoxical behavior is repeated in many lesions affecting the optic nerve on its surface, as for instance when a neoplasm begins to press on the nerve. Quite regularly, too, in acute retrobulbar neuritis the papillo-macular bundle is the part that is first and most affected, although in the majority of cases this disease certainly has its main seat in the sheaths of the nerves. We explain this fact by assuming that the fibers of this bundle are particularly vulnerable, and connect this vulnerability with its especially exquisite and delicate function, with which perhaps there is associated a correspondingly delicate anatomical structure. "The more delicate a machine, the more readily it is destroyed." 3. The lesion of this bundle can have been but slight, for the vision was still normal; if we had simply stopped at determining this, we should not have discovered the disease of the optic nerve at all. But the testing of the visual field with a small red object showed in the center a diminution of the ability to appreciate red. The examination of the eye with colors accordingly, affords us a particularly delicate test for demonstrating even the slightest impairment of sight. 4. The central scotoma made itself apparent to the patient by nyctalopia, since it was only by day that he had a "glimmering light" before his eyes, while in the evening he thought he had normal sight. When the patient makes statements of this kind, therefore, one should always look for a central scotoma. 5. The first branch of the trigeminus runs through the cavernous sinus to the superior orbital fissure, and in this part of its course lies close to the lateral surface of the body of the sphenoid. This is the reason why it may react under the form of occasional neuralgias to an inflammation of the mucous membrane of the sphenoid sinus.

739. Treatment.—As long as the communication between the cavities and the nose is open, treatment is carried on by way of the nose (the maxillary antrum is often opened up and treated through an alveolus of one of the teeth). If the ostium of the cavity is closed either provision must be made for a permanent escape of the secretion or the cavity must be obliterated.

[In cases in which there is a toxæmia or a remote infection from absorption of bacterial matter from the sinus, it may be necessary to supplement treatment of the sinuses by the use of vaccines.—D.]

PART IV

ANOMALIES OF REFRACTION AND ACCOMMODATION

740. The eye is constructed upon the principle of a camera obscura. This consists of a box blackened on the inside, the anterior wall of which is formed by a strong convex lens, which throws upon the posterior wall an inverted image of the objects that may chance to be in front of the camera. In the human eye we find instead of the convex lens quite a number of refracting surfaces constituted by the surfaces bounding the refracting media of the eye, namely, the cornea, aqueous humor, lens, and vitreous; and in place of the posterior wall is found the retina, which not only receives the image, but also at the same time perceives it. Hence a diminution of visual power may be produced by two different causes: either the dioptric apparatus of the eye is defective, so that a sharp image is not thrown upon the retina, or it is the retina that is at fault in that it is not sensitive enough.

In order to throw a sharp image upon the retina the dioptric apparatus of the eye must fulfill two conditions. In the first place, the refracting media must be perfectly transparent. Hence opacities of the cornea, lens, etc., make distinct vision impossible. The second condition is, that the refractive power of the media should be such that they project an image of external objects which is both perfectly distinct and also lies precisely upon the retina. The variations from this rule we designate as errors of optical adjustment or as errors of refraction and accommodation. The theory of these errors, as we see it presented to us to-day, forming a harmonious, well-compacted whole, is chiefly Donders's work. It is the exactest portion of ophthalmology, and, in fact, of all medicine, for it is based directly upon the application of physical and mathematical laws to the eye. These laws, therefore, as far as they will require consideration here, must be supposed to be known in advance.

ANOMALIES OF REFRACTION AND ACCOMMODATION

CHAPTER I

THE THEORY OF GLASSES

741. Convex and Concave Glasses.—The refractive power of a lens is determined by the position of its principal focus. Under the latter name is denoted that point at which rays are united which come from an infinite distance, and hence are parallel when they strike the lens.

In *convex lenses* which render parallel rays convergent, the principal focus (*F*, Fig. 352) lies on the opposite side to that upon which the rays fall. Here all the parallel rays are collected or condensed (hence "condensing" lens). If the rays emanate from a point, they will also be united into a single point, but if the object that emits the rays has an extension in space, they are

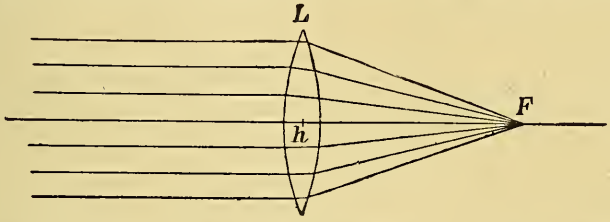


Fig. 352.—Union of Parallel Rays, refracted by a Convex Lens.

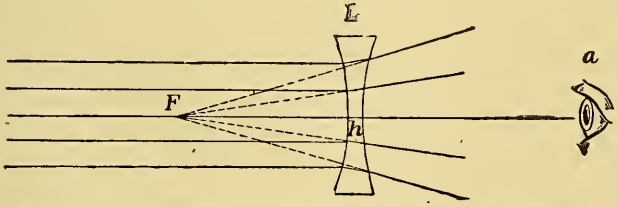


Fig. 353.—Dispersion of Parallel Rays by a Concave Lens.

united into a diminished, inverted image of the object. This image is real—i. e., formed by an actual union of the rays at this spot. Just as rays falling upon the lens in a parallel direction ultimately reach the principal focus, *F*, so also rays which go in the opposite direction, from *F*, and impinge upon the lens, will emerge from the latter in a parallel direction.

Concave lenses so refract the rays which impinge upon them in a parallel direction that the latter become divergent when they emerge; hence the name "dispersing" lenses (Fig. 353). These rays never come together, but, on the contrary, diverge farther and farther from each other. Hence an actual (real) focus—i. e., point of union of the rays—does not exist in this case. But if an observer is stationed behind the lens—e. g., at *a*—and receives the diverging rays upon his eye, he gets the same impression as if

these rays came from a point situated upon the other side of the lens, a point located at *F*, where the rays would meet if prolonged backward. The observer accordingly believes that he does see at this point the image of the object, which emits parallel rays, although there is no image in reality present at this spot, and, in fact, there is no image formed at any spot whatever. This apparent image is called virtual (formed at the virtual focus), and lies upon the same side as that from which the rays come to the lens. Here, precisely as in the case of convex lenses, the law holds good that the path of the rays is the same when the direction is reversed. If rays impinge upon the lens from its posterior aspect (*a*) with a convergence such that they are directed toward *F*, they will be parallel upon their emergence from the lens at its anterior aspect.

The distance between the principal focus, *F*, and the optical center *h*, [or really the first principal point of the lens] is called the *principal focal distance.* In convex lenses this lies upon the opposite side to that upon which the rays impinge; it is known as positive focal distance, and convex lenses are hence given the sign +. The converse is true of concave lenses, whose negative focal distance is denoted by the sign —. The refraction of rays by a lens is greater, cæteris paribus, the more curved its surface is—that is, the shorter its radius of curvature.

The *effect* of the glasses depends not only upon their refractive power, but also upon their distance from the eye. Generally speaking, the effect of concave glasses is weakened and of convex glasses is strengthened the farther they are held from the apex of the cornea. [This is true for distance glasses, both convex and concave. It is also true of concave, but not of convex reading glasses. Whether the effect of the latter is increased or is diminished by carrying them off from the eye depends partly on the strength of the glass, partly on the distance of the object looked at. The fact that the effect of glasses is altered by carrying them away from the eye is of importance in estimating the refraction by the direct method (see page 103). Unless the ophthalmoscope is held at the proper distance (about half an inch) from the patient's cornea the estimate will be faulty.—D.]

The stronger the glass the greater the influence which its distance from the eye exerts. For this reason the intervals between the separate numbers are not required to be so numerous in the case of the stronger glasses as in the weaker ones, since the effect of the former can be readily increased or weakened by slight alterations in their distance. This change of strength from change of distance is mainly of advantage to those that have been operated upon for cataract, who possess no power of accommodation, and hence with their strong convex glasses can never have their sight adjusted except for one definite distance. By slightly displacing the glass they so far help matters that with the same glass they can see sometimes a little closer to them, sometimes a little farther off.

742. Numbering of Lenses.—The numeration of lenses, the object of which is to show their refractive power, is based upon their principal focal distance, for, the more strongly the rays are refracted, the closer their point of union comes to the lens, and the shorter, therefore, is their principal focal distance. The latter accordingly is in inverse proportion to the

refracting power of the lens, and can hence be utilized as a measure for it. A principal focal distance of 1 metre is assumed as unity, and the lens which has this principal focal distance is called a metre lens, and its refractive power a dioptry (D). If 2 metre lenses are placed in apposition, twice the refractive power is obtained, namely, 2 dioptries (2 D). The principal focal distance is now one-half that of the former lens—i. e., $\frac{1}{2}$ metre = 50 cm. Instead of placing 2 metre lenses in apposition, we may grind a lens whose curvature is twice as great (or whose radius of curvature is half as large) as that of the single metre lens. We thus get a lens of a 2-dioptry refracting power and a principal focal distance of 50 cm. In analogous fashion a lens of 4 D would have one-fourth the focal distance of the metre lens—i. e., 100 cm. $\div$ 4 = 25 cm. A lens of $\frac{1}{2}$ D refractive power has, on the other hand, a focal distance of 100 cm. $\div \frac{1}{2}$ = 200 cm. The focal distance of a lens of n D is thus 100 cm. $\div$ n. The trial cases ordinarily employed for examining the eye contain lenses from 0.25 D [or 0.12 D] up to 20 D.

743. **Old System of Numbering Lenses.**—In times gone by lenses were not numbered according to the metric system, but according to the *inch* system, and even at present opticians sell lenses of this sort. The unit which formed the basis of the old system of numbering was a lens of 1-inch focal distance. A lens of 10 inches focal distance has only a tenth part of the refractive power of the 1-inch lens, and hence is denoted by the figure $\frac{1}{10}$; for the same reason a lens of 30 inches focal distance is called a $\frac{1}{30}$ lens, and so on. The refractive power of the glass is thus expressed by a fraction whose denominator is the principal focal distance, in accordance with the law that the refractive power is the reciprocal of the focal distance. Upon the lenses themselves are engraved not the fractions but their denominators—i. e., the focal distances. The numbers contained in the old cases of glasses run ordinarily from the weakest glass, No. 80, up to No. 2, or $1\frac{1}{2}$. To speak precisely, these numbers do not give the focal distances of the glasses, but their radii of curvature. The optician does not grind lenses of a certain refractive power, but lenses of a certain curvature—corresponding to the curvature of his grinding tool—and marks the radius of curvature upon the glass as its number. And, in fact, if the index of refraction of the glass is 1.5, the radius of curvature of a biconvex or biconcave lens is equal to its focal distance. But as the glass used for lenses almost always has a higher index of refraction than this, the focal distance of a lens is ordinarily somewhat less than the number engraved upon the glass. In practice this small difference can be neglected, and it therefore was an important advantage which the old system of numbering glasses had to offer, that from the number of the glass one knew at once its focal distance, and did not have to calculate it, as with the metric system. To compensate for this, the old system of numbering had so many disadvantages that it was seen to be necessary to give it up. The refractive power of the lens was expressed by fractions, which made the work of calculating the value of lenses rather complicated. The very unit of the system—namely, the inch—varied in magnitude in the different countries, so that a No. 10 glass bought in Paris had a different focal distance from that of one of the same number which had been made in London or in Vienna. And anyhow, as a matter of fact, the inch everywhere is bound to drop into oblivion. So when Nagel, in 1866, first proposed the metre lens as the unit of a new system of numeration, his idea soon found acceptance, and finally also passed into practical use, after an inter-

national commission appointed for this purpose, upon the motion of Monoyer, had declared itself in favor of the metric system of designating glasses.

The *conversion* of the numbers of one system into those of the other is very simple. The metre measures from 37 to 39 inches, according to the length of the inch in different countries. If we are not concerned in making a precise estimate, we may, in order to be able to carry the calculation in our heads, regard the metre as about 40 inches (the more so as the old No. 40, owing to the somewhat high refractivity of the glass—see supra—had generally a focal distance of somewhat less than 40 inches). A glass of 40 inches focal distance ($\frac{1}{40}$ glass) is therefore about a dioptry. A No. 10 glass ($\frac{1}{10}$)—i. e., of 10 inches focal distance—has one-fourth the focal distance of a No. 40 glass, and is therefore four times as strong as the latter; it is accordingly 4 dioptries. Hence, we get at the value of the old number in dioptries if we divide 40 by the old number. The conversion of the new numbers into the old is done in a similar way; e. g., a glass of 5 D has one-fifth the focal distance of 1 D. The latter is about 40 inches; a glass of 5 D, therefore, has a focal distance of $40 \div 5 = 8$ inches. Hence, the old number is converted into the new, or the new into the old, by dividing 40 by the given number, when the number in the other system is then at once obtained.

744. Menisci.—The lenses so far considered have been biconvex and biconcave. In addition to these plano-convex (Fig. 354 A) and plano-concave (Fig. 354 B) lenses are employed, the refracting power of which

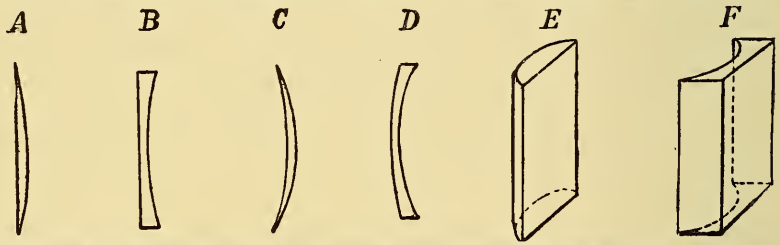


Fig. 354.—A, plano-convex lens; B, plano-concave lens; C, positive meniscus; D, negative meniscus; E, convex cylindrical lens; F, concave cylindrical lens.

is one-half of that possessed by doubly curved lenses having the same surface curvature. There are also lenses one surface of which is convex, the other concave. If the radius of curvature of the two surfaces is the same, so that they are parallel with each other, the glass acts like a plane glass. This is the case in the coquille or curved protective glasses. Such lenses have refracting power only when the curvature of one surface surpasses that of the other (meniscus). If the convex bulges more than the concave surface, the lens acts as a convex lens (positive meniscus, Fig. 354 C). If, on the contrary, the concave is more strongly curved than the convex side, the combination acts as a concave lens (negative meniscus, Fig. 354 D). Menisci have the advantage over ordinary lenses that we see as distinctly when looking through the marginal portion of the glasses as when looking through their center, while ordinary lenses give distorted images when we look through their marginal portion. Hence, menisci are also called periscopic [1] glasses. [Some menisci are called toric, because ground with a toric[2] instead of a spherical surface.—D.]

[1] From περί, about, and σκοπεῖν, to see.
[2] [A toric surface is one generated by rotating a circle about some point other than its center.—D.]

745. Cylinders.—Besides the lenses just given, which are called collectively *spherical* lenses, there are also *cylindrical* lenses. If we imagine a segment cut out of a cylinder (Fig. 355), this segment forms a convex cylindrical lens (Fig. 354 E). Rays which impinge on such a lens in a plane that passes through the axis, *aa*, of the lens, suffer no deviation. Rays, on the other hand, which lie in a plane perpendicular to the axis (corresponding to *bbb*) undergo the maximum refraction—i. e., that which would correspond to the curvature of the cylindrical surface. The same thing is true of concave cylindrical lenses (Fig. 354 F), which may be regarded as representing the mold of the positive cylinder.

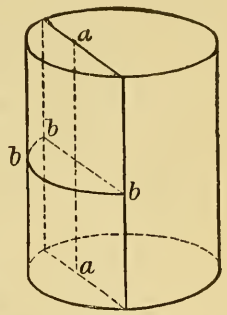


FIG. 355.—THE CONVEX CYLINDRICAL LENS CONSIDERED AS A SEGMENT OF A CYLINDER.

Since cylindrical lenses refract unequally the rays impinging upon them in different meridians, they are calculated to compensate for a difference in refractivity existing in the different meridians of the eye; they serve, accordingly, to correct regular astigmatism.

[A spherical lens if tilted acts to some extent like a cylinder.—D.]

[STRENGTH OF CYLINDERS:—The indicated strength of a cylinder is the refractive power of the strongest refracting meridian, i. e., that which is at right angles to the axis. Thus a cylinder of +2D is one in which this meridian has the same refractive power as a +2D spherical lens. The axis, on the contrary, has no refractive power; i. e., in the plane of its axis the cylinder acts like plane glass.—D.]

[AXIS OF CYLINDERS:—According to the notation prevailing in this country the direction of the axis of a cylinder is indicated by the angle which it makes with the horizontal, the angles being numbered continuously from 0°, which is situated at the left side of either eye (nasal side of the right eye, temporal side of the left eye), round to 180° at the right side of the eye. This system is shown in Fig. 356.

According to another system the vertical meridian is indicated by 0° or *V*, and from this point the angles are numbered on either side to 90° (or *H*), which is the horizontal, those angles on the temporal side being indicated by *t*, those on the nasal side by *n*. This system is shown in Fig. 357. A third system recommended by the International Ophthalmological Congress of 1909, is shown in Fig. 358. The angles are numbered in each eye from 0° at the nasal to 180° at the temporal side.

These systems are also employed for indicating the direction of the axis of a prism —e. g., prism 1°, apex at 75°, indicates a prism of 1° refracting angle with its apex turned to the left 15° from the vertical.—D.]

746. [Combination of Glasses.—1. Two spheres of the same sign make a sphere equal to their sum; e. g., —3.00 sph. ⌒ —2.00 sph. = —5.00 sph.

2. Two spheres of opposite signs make one sphere equal to the difference of the two; e. g.,—3.00 sph. ⌒ + 4.00 sph. = +1.00 sph.

3. The above rules apply to cylinders when both have the same axis; e. g., + 1.00 cyl. axis 90° ⌒ +2.00 cyl. axis 90° = +3.00 cyl. axis 90°; and +1.00 cyl. axis 90° ⌒ — 250 cyl. axis 90° = —1.50 cyl. axis 90°.

4. Two cylinders of the same sign and same maximum strength placed at right angles to each other make a sphere of the same sign and same strength; i. e., +2.00 cyl. axis 90° ⌒ +2.00 cyl. axis 180° = +2.00 sph.

5. A cylinder by the addition of a sphere of equal strength and opposite sign is converted into a cylinder of opposite sign and with its axis at right angles; i. e., +1.00 cyl. axis 90° ⌒ −1.00 sph. = +1.00 cyl. axis 180°.

6. Two cylinders, on which one (*a*) is plus, the other (*b*) is minus, when crossed at right angles, make either a plus sphere equal in strength to *a* and combined with a minus cylinder equal in strength to *a*+*b* and having the axis of *b*; or they make a minus sphere equal in strength to *b* and combined with a plus cylinder equal in strength to *a*+*b* and having the axis of *a*. Thus +2.00 cyl. axis 90° ⌒ −3.00 cyl. axis 180° = either +2.00 sph. ⌒ −5.00 cyl. axis 180° or = −3.00 sph. ⌒ +5.00 cyl. axis 90°.

These rules are not strictly applicable unless the glasses that are combined are in absolute contact.—D.]

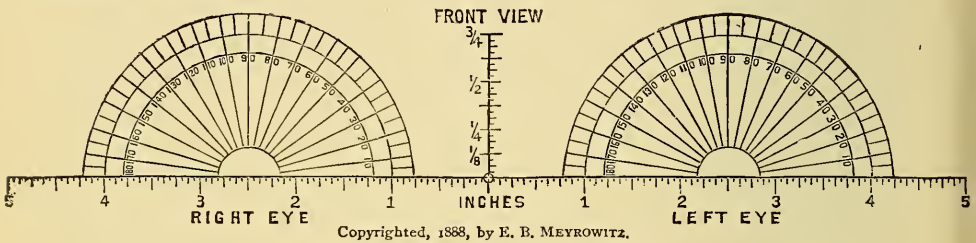


Fig. 356

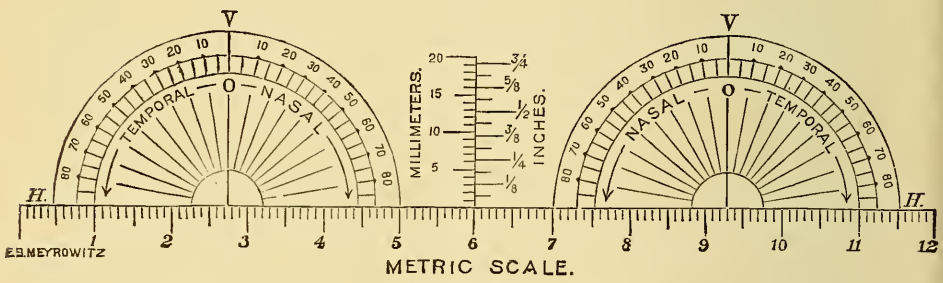


Fig. 357

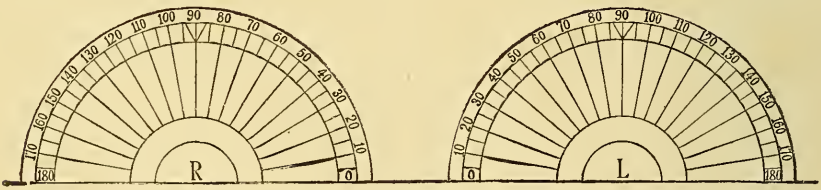


Fig. 358.

747. Prisms.—Prisms are employed for glasses either alone or combined with lenses. The number engraved upon prisms indicates their refracting angle [3]; the deflection which the rays undergo amounts in the case of weak prisms to one-half this angle. [For more precise statement see page 766 and for uses of prisms § 672 passim and pages 757 and 794.—D.]

[3] [Prisms are often and much better numbered by their deflecting power measured in centrads or prism-dioptries. A *centrad* (denoted by the sign ▽) is the one-hundredth part of the arc which is equal in length to the radius (i. e., is a deflection of 0.57°). A *prism-dioptry* (denoted by the sign △) is an angle whose tangent is 0.01, i. e., corresponds to a deflection of 1 cm. at 1 metre. The terms, however, are practically interchangeable, since a prism of *n* centrads and one of *n* prism-dioptries are both almost precisely equivalent to a prism of *n*° refracting angle.—D.]

[A prism always displaces towards its apex the projected image of an object seen through it (e. g., in Fig. 343 displaces o to o').

A *spherical* lens may be regarded as composed of a series of prisms, piled on each other, base on apex, the strength of each component prism increasing from the center to the periphery of the lens. In a convex lens the apices of the component prisms all point away from the center of the lens, in a concave lens they all point toward it. It is on this fact that the focusing properties of spherical lenses depend. In a convex lens, for example, the rays passing through the center traverse practically a plane glass and are not deflected; rays passing through the lens a little way from the center are deflected slightly because they traverse a weak prism while those that are further out are deflected still more strongly because they pass through a stronger prism. These several rays, being thus bent at different angles, cross each other, and if the component prisms are of suitable strength can be made all to intersect at a common point—the focus.

For the same reason, when we look through the center of a convex lens at an object the latter appears undisplaced, because we are looking at it through plane glass. If, however, we look through the upper part of the lens at the object, the latter will appear displaced upward by the particular component prism through which we see it. The nearer we are to the periphery of the lens, the greater the displacement will be. The lens, in other words, has a prismatic action, increasing steadily in amount from its center to its periphery. This property is made use of when, instead of prescribing prisms, we *decenter* lenses, i. e., direct them to be worn so that the wearer looks through some point other than the optical center. The decentration, required to produce a desired prismatic effect, is given well enough for all practical purposes by the simple formula

$$d = \frac{10p}{L}$$

where p is the prismatic effect in prism-dioptries, L is the strength of the lens in dioptries, and d is the decentration in mm.—D.]

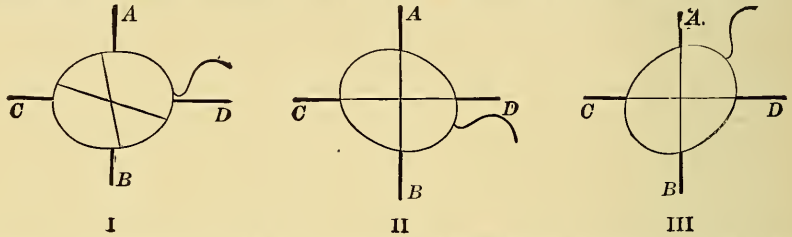
748. **Analysis of Glasses.**—We are very often called on to determine the precise strength and character of the glass a patient is wearing. To do this we first ascertain whether it is convex or concave. This we do by means of the parallactic displacement. This is the displacement produced in the apparent position of an object when we look at it through a lens and move the lens to and fro. This takes place as follows. An object that we look at through the center of a convex lens appears in its natural place, but if we move the lens down so as to see the object through the upper part of the lens, the object will appear displaced upward (see supra), i. e., will move in a contrary direction to the lens. Per contra, an object that we see through a concave lens will appear to move in the same direction that we move the latter. If in either case we place in contact with the lens another of equal strength and opposite sign, we shall neutralize the convexity of the one with the concavity of the other, so that the combination has the effect of plane glass and now an object seen through it no longer moves when it moves.

A *cylinder* when moved in a direction at right angles to its axis causes a parallactic movement like a convex or concave spherical glass. When moved in the direction of its axis it produces no movement, because along its axis it acts like plane glass. A cylinder also distorts objects seen through it. Hence a cross seen through a cylinder will look skewed (Fig. 359 I). If now the cylinder is rotated, the cross will become straight whenever either arm of it coincides with the axis of the cylinder (Fig. 359 II and III).

The presence of a *prism* is shown by a displacement of the arms of the cross, which, however, always remain parallel to their original position (Fig. 360 I). By rotating the glass we can ascertain the direction of the prism and the amount of prismatic displacement (Figs. 360 II and III).

Accordingly, to *analyze a glass*, we look through it at a cross, rotate the glass until

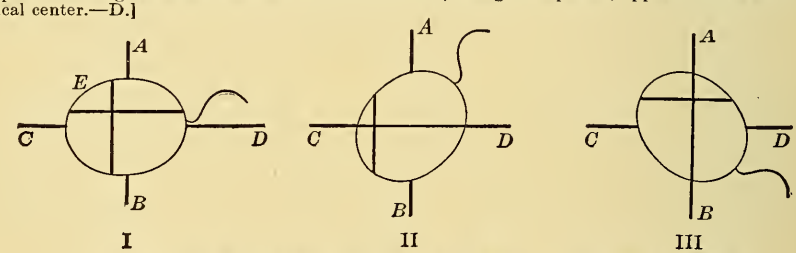
the cross seems no longer skewed (Fig. 359 II), and add a sphere of opposite sign till all movement in the direction AB is abolished. Leaving this neutralizing glass in place, we move the lens in the direction CD and, according to the direction of the parallactic movement, neutralize this also with a convex or concave cylinder having its axis placed in line with AB. The combined addition with reversed signs will equal the sphero-cylinder in the glass. If in addition there is a prism as shown by a displacement of the cross arms like that in Fig. 360, we determine its strength by neutralizing it with a prism placed in the opposite direction and of such strength that it will abolish the displacement.



I II III

[Fig. 359.—Distortion Produced by a Cylinder. Determination of Axis and Optical Center. (From Posey and Wright.)

A right-angled cross, *AB–CD*, is seen through a glass containing a cylinder. If (I) the axis of the cylinder does not coincide with either *AB* or *CD*, the cross appears skewed, so that the arms no longer make a right angle. The cross, however, is not displaced as a whole either to one side or the other (cf. Fig. 360). If now the glass is rotated until the axis of the cylinder coincides with one arm *AB* of the cross, the cross will appear right-angled and unbroken (II). The same thing will happen if the glass is rotated 90° further (III), so that the axis of the cylinder coincides with *CD*. In both II and III the point on the glass where the two arms *AB* and *CD*, being undisplaced, appear to intersect is the optical center.—D.]



I II III

[Fig. 360.—Displacement Produced by Prism. (From Posey and Wright.)

A right-angled cross, *AB–CD*, is seen through a glass containing a prism. (I) The apex of the prism does not lie in line with either arm of the cross, being, in fact, directed toward *E*. Both arms of the cross appear displaced bodily, but neither is skewed. The amount of displacement of *AB* indicates the lateral effect, and the displacement of *CD* the vertical effect of the prism when in this position. If now the glass is rotated, both arms will appear to shift, but each will always remain parallel to its original position. (II) The glass has been rotated until *CD* appears unbroken, while *AB* is displaced toward *C*. The amount of its displacement now represents the total strength of the prism, and the apex of the prism points toward *C*. (III) The glass is rotated 90°. Now the line *AB* appears unbroken, and *CD* is deflected toward *A*. The apex of the prism points toward *A*, and the amount of deflection of *CD* corresponds to the total strength of the prism. A prism placed with its base at *A* and neutralizing the displacement *CD* measures the strength and gives the direction of the prism in the glass.—D.]

It is often important to determine the *optical center* of a glass. If on looking through the glass we see the cross arms skewed (Fig. 359 I) we rotate the glass till they are so no longer, and then if the arms are displaced, as in Fig. 360 I, we slide the glass up and down or in and out until the cross is unbroken (Fig. 359 II), and mark on the glass the point where the two arms seem to intersect. This will be the optical center.

749. Bifocal Glasses.—It is frequently the case that a person needs glasses both for distant and near vision, but of a different strength. This is especially true of presbyopes. The glass for near work in those who wear concave glasses must be less concave or actually be convex, in those who wear convex glasses must be more convex than the distance glass. Hence, in order to avoid changing the glasses constantly, spectacles have been constructed which have a stronger refractive power in the lower part than in the upper. The first person to make such a glass was Franklin who constructed it for his own use by joining together two half lenses in such a way that they

were in contact along a horizontal line (Fig. 361 A). Since the horizontal dividing line causes a good deal of confusion when the gaze is directed straight forward, such glasses are now constructed so that the frame contains the glass designed for the distance, and upon the lower part of this is cemented a [paster or] small semilunar convex lens (bifocal glass (see Fig. 361 B and C)). In near vision, in which the visual plane is depressed, the eye looks through this part of the glass, while for distant vision the upper part of the glass is used. [Instead of cementing the segment upon the distance glass, it may be fused or ground into the latter (fused bifocal, kryptok).—D.]

750. Adjustment of Glasses.—In prescribing glasses attention should be paid to having the optical centers of the glasses separated from each other as far as are the pupils of the person wearing the glasses, as otherwise he would be looking through the edges of the latter. In this case the images are less distinct, and, moreover, the glasses then act like weak prisms [(see page 837). (For the method of determining the optical center see page 838 and of determining the pupillary distance see page 76.) In reading, the visual lines are converged and directed downward. Hence in a reading glass the centers of the lenses should be approximated 4 to 5 mm. and dropped 3 to 4 mm., and the lenses themselves should be tilted forward about 10°, so that the visual

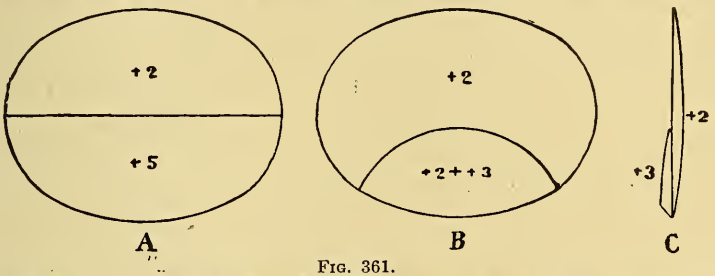


FIG. 361.

A, Franklin glass, B and C, bifocal glass for an old hypermetrope, who no longer has any accommodation and hence needs for near work a glass 3 D stronger than for distance.

lines shall strike them perpendicularly and in their centers. Even for distance glasses this is advisable, since most of our seeing is done somewhat below the horizontal plane. For this reason particular care has to be taken in adjusting bifocal segments, so that the upper line shall not be too high and interfere with distinct vision when the eyes are directed somewhat down as in walking on the street. Adjustment of glasses along these lines is particularly important in anisometropia (see § 794).

Many prefer meniscus and especially toric glasses, because they give a somewhat larger field of distinct vision. But in the case of strong convex lenses of this type, the centers, owing to the concavo-convex form, are thrown so far from the eye as often to make the glasses appreciably too strong (see page 832).—D.]

751. Stenopæic Spectacles.—Stenopæic[4] spectacles consist of a blackened disk of metal, in which is placed a small round hole or a narrow slit. They are sometimes employed with advantage in cases of corneal opacities. They are adapted for those cases in which a part of the pupillary area of the cornea is clear, while another part is occupied by a semitransparent spot of cloudiness, which by diffusing the light causes confused vision. If the disk is held before the eye in such a way that the aperture lies in front of the transparent portion of the cornea, the latter alone is used for seeing, and the opacity which causes the confusion is excluded. Since a man has only a very small field of vision in looking through such an aperture, and since, moreover, he can not move his eye about behind it, stenopæic spectacles are not suited for going about, but they often enable the patient (who can best manage them by holding them in his hand) to read when it would be impossible to do so in any other way.

For *protective glasses* and *eye-guards* see page 53.

[4] From στενός, narrow, and ὀπή, peep-hole.

CHAPTER II

OPTICAL PROPERTIES OF THE NORMAL EYE

(*a*) *Refraction*

752. *By the refraction of the eye we mean its optical adjustment when in the state of rest—i. e., in the absence of any accommodative effort.* The optical adjustment of the normal eye is correct, i. e., parallel rays impinging upon the cornea are united so as to form a sharp image upon the retina. The retina, therefore, is situated at the principal focal distance of the dioptric apparatus of the eye, and thus constitutes its focal plane. Such a refractive condition is called *emmetropia*,[1] *E* (Donders).

[True emmetropia is rare. Nearly all very young children are hyperopic, and the proportion of emmetropia increases during the growing period. Thus in 4800 school children the proportion of emmetropes was found to be 4 per cent, the percentage being 0 at the age of 5 or 6, 0.5–1.0 before 9, and 7 at the age of 12 (Tenner).—D.]

In order to follow the passage of the rays through the refracting media of the eye we must know precisely the curvature of the refracting surfaces, their distance from each other, and the index of refraction of the individual refracting media. On the basis of these data we can by a complicated calculation find the path of the rays from one refracting surface to the other and ultimately to the retina. In order to facilitate this calculation for practical purposes, Donders has devised a simplified schematic model, the *reduced* or *schematic eye* (Fig. 362). This has an axial length of 20 mm. (*h b*) and consists of a single refracting substance, which has an index of refraction of $\frac{4}{3}$, and the anterior surface of which has a radius of curvature of 5 mm. Hence, the center of curvature (*k*) lies 5 mm. behind the refracting surface and 15 mm. in front of the retina, which is at a distance from the former equal to the principal focal distance of the eye (20 mm.). Since there is only one refracting surface, its center of curvature coincides with its nodal point (nodal point of the eye)—i. e., with the point having this property that all rays passing through it ("principal rays") undergo no refraction.

This schematic eye varies from the normal human eye, the principal focal distance of which [measured from its posterior principal point] is a little over 21 mm. while that of the schematic eye is assumed to be 20 mm. Moreover, since the lens is left out of the latter, we must, in order to get a sufficient refractive power, make the radius of curvature of the anterior refracting surface correspondingly smaller (5 mm. as opposed to 7.5 mm., which is the mean value of the corneal radius). However, calculations in

[1] From ἔμμετρος, in due measure, and ὤψ, sight.

regard to the size of retinal images, of diffusion circles, etc., which are made upon the basis of the schematic eye, give results which approximate very closely to those found for the real eye. Hence, for practical purposes the schematic eye can without hesitation be made the basis of calculation.

The calculation which the ophthalmic practitioner has most frequently to make concerns the size of the retinal image of a given object. To do this the size of the object and its distance from the eye must be known. We find the retinal image of an object by drawing from the terminal points, o, o_1 (Fig. 362), of the latter the principal rays through the nodal point, k, to the retina, which they meet in b and b_1. Accordingly, $b\ b_1$ is the retinal image of the object $o\ o_1$. The triangles $o\ o_1\ k$ and $b\ b_1\ k$ are similar; hence, $b\ b_1 : o\ o_1 = b\ k : o\ k$, and so $b\ b_1 = \frac{o\ o_1 \times b\ k}{o\ k}$. If we call the size of the images ($b\ b_1$) B, that of the object ($o\ o_1$) O, and the distance of the latter ($o\ k$) from the eye E, then $B = \frac{O \times 15\text{ mm.}}{E}$. The size of the retinal image is therefore directly proportional to the size of the object and inversely proportional to the distance of the object from the

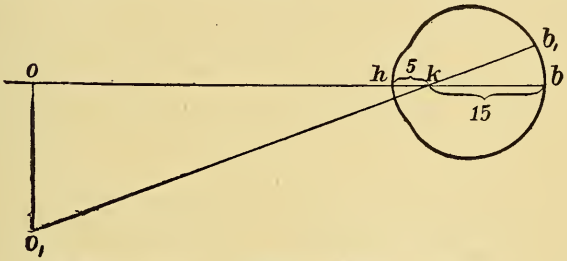


Fig. 362.—Reduced (Schematic) Eye of Donders.

eye. E. g., a rod 1 metre high placed at a distance of 15 metres from the eye would give a retinal image whose altitude $B = \frac{1{,}000\text{ mm.} \times 15\text{ mm.}}{15{,}000\text{ mm.}} = 1$ mm. This rod, if approximated to one-third of the distance—i. e., 5 metres—would give a retinal image of $\frac{1{,}000\text{ mm.} \times 15\text{ mm.}}{5{,}000\text{ mm.}} = 3$ mm. or three times as great as before. This sort of calculation is often applied in order to discover the size of a diseased area of the retina, corresponding to which is a scotoma in the visual field, whose size can be determined, by examination. The scotoma is then regarded as the object for which the size of the retinal image is calculated.

(*b*) *Visual Acuity*

753. The smaller the objects that an eye can distinguish or the greater the distance at which it can distinguish an object of given size, the greater is the acuity of vision that it possesses. Suppose, for instance, that the eye is able just to distinguish the object ab (Fig. 363) at the distance ak. Another better eye still distinguishes the object when it is carried twice as far off, to the distance $a_1\ k$. The size of the retinal image $a\ \beta_1$ is in this case reduced to half of that in the former ($a\ \beta$)—that is, the visual acuity of the second eye is twice as great as that of the first. Instead of

carrying the object *a b* twice as far off, we may leave it at the same spot but make it half as small ($a\ b_{11}$). In this case, too, the size of the retinal image would be reduced one-half. In either case, therefore, a retinal image of the same size is obtained, and that, moreover, because the angle *v* remains the same. This angle is subtended by the rays which pass from the terminal points of the object through the nodal point of the eye to the retina. It is called the *visual angle* (angulus visorius), and is the true measure of the visual acuity. For estimating the visual acuity—i. e., the minimum visual angle—there are from the above example two ways open to us, both of which are made use of. We may take an object of given size and carry it off with us from the eye until the farthest point at which it can be recognized is reached. This, for instance, is done when we try to find at what distance an eye is able to count the fingers when extended. The second way consists in conducting the test at a constant distance,

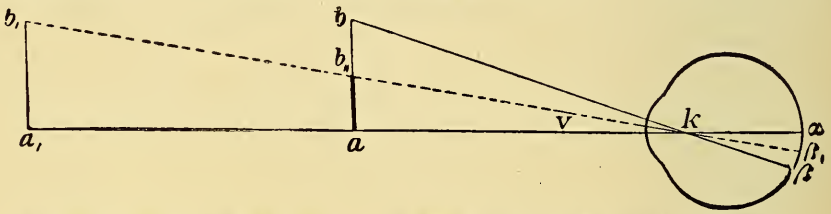


Fig. 363.—Behavior of the Visual Angle when Objects vary in Size and Distance.

objects of different size being presented to the eye and the attempt being made to find the minimum size which the object can have and still be recognized. This method is employed when we test the visual acuity with test types.

In estimating the vision, just as in estimating the refraction, the influence of the accommodation must be excluded, for which reason the object used must be placed at a distance at which no accommodation of any notable amount is required, i. e., at a distance of 5 to 6 metres.

754. Tests of Visual Acuity.—What objects are best adapted for testing the visual acuity? A single dot, the distance of which from the eye is altered, is unsuitable because the visibility of a dot depends less upon the visual angle which it subtends than upon its luminosity. The fixed stars, radiant as they are, are nothing but mathematical points even when seen with the most powerful telescope; they have, therefore, a visual angle equal to zero, and yet they are seen very clearly. Every one can recall that a cross upon the top of a church spire, when sparkling in the luster of the setting sun, was seen at distances at which the church spire itself was scarcely distinguishable. Hence we select for the test not one but two dots (or two parallel lines) and then determine the greatest distance from the eye at which they can still be perceived as separate objects. From this can be readily calculated the minimum visual angle, which for

a normal eye amounts to about 1′. On the basis of this determination Snellen has constructed his test types. Snellen's *test types* are placed at a distance of 6 metres. They consist of letters of varying size arranged in rows. Each row contains letters of the same size, and has a number inscribed over it. This number gives the distance in metres at which the separate letters of the row appear to a normal eye under a visual angle of 5′. For example, this is the case with the letter *F* of the row with the superscription 12, when placed at a distance of 12 metres. Like all the other letters of the test card, it is inscribed within a square, whose sides are divided by partition lines into 5 parts each (Fig. 364). If, therefore, at 12 metres the whole square appears under an angle of 5′, the angle for each partial square is 1′. This is the minimum visual angle for the normal eye, and since the partial squares correspond to the details of the letter, these details consequently will still be made out by a normal eye. Hence the numbers standing over the letters give the distance at which each entire letter appears under an angle of 5′, and its details appear under an angle of 1′, and is thus the distance at which each row of letters can be made out if the vision is normal. If the distance at which the letter can be seen is greater or less than that indicated, the vision is greater or less than normal. The vision, therefore, may be expressed by the ratio between the distance *d*, at which the letter is actually recognized, and the distance *D*, at which it ought to be recognized, and which is inscribed over the letter—i. e., $S=\frac{d}{D}$ (where *S* stands for sight—denoted also by *V* = visus, or vision). Hence, an eye that sees the letter *F* (Fig. 364) at 12 metres has $S=\frac{12}{12}=1$. As a general thing, however, we make the person under examination stand always at the same distance from the card—namely, 6 metres. If at this distance he distinguishes the lowermost row which has 6 written over it, he has $S=\frac{6}{6}=1$; but if, for instance, he distinguishes only the uppermost letters, No. 60, $S=\frac{6}{60}$.[2] This is equal to $\frac{1}{10}$ of the normal visual acuity, but we always write the fraction in full without reducing.

№ 12

Fig. 364.—A Letter from Snellen's Test Card.

Many test types have been constructed on the principle enunciated by Snellen, so that unity in the denotation of the visual acuity has been lost and with it any plan admitting of general comprehension. To restore these features, the International Ophthalmological Congress in Naples in 1909 adopted the test types constructed by Hess for the purpose and have pronounced them international. The *international test types* are

[2] [In this country and in England the distances are generally expressed in feet—i. e., $V=\frac{20}{20}$, or $\frac{20}{xx}$ means that a patient at 20 feet saw the line marked 20.—D.]

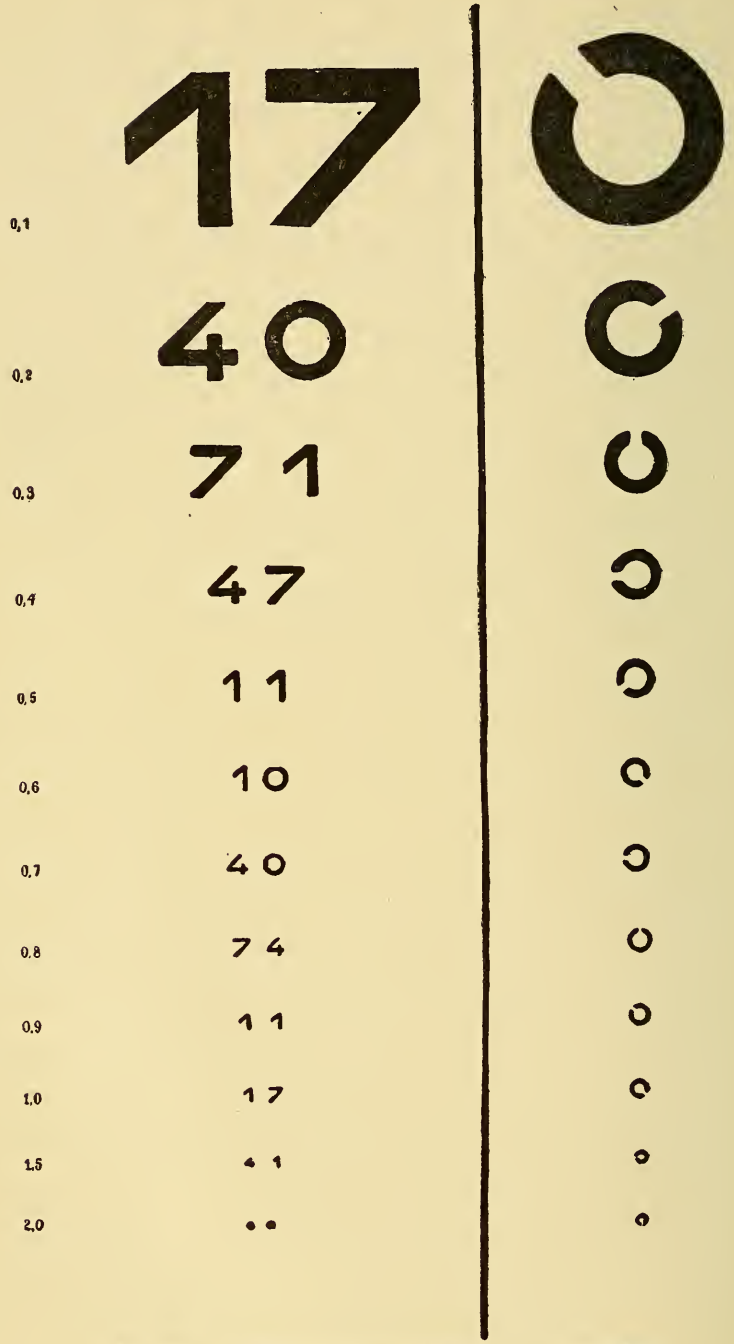
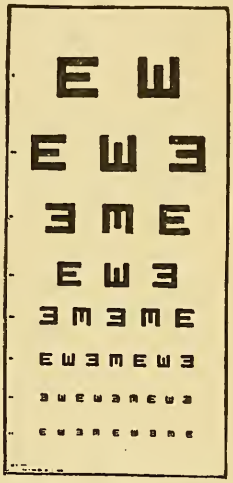


Fig. 365.—International Test Types, Reduced One-Third.

designed for a distance of 5 metres because a space of 6 metres as demanded by the Snellen types is often not at the physician's command. The visual acuity found is expressed in decimals. These are placed alongside the several lines and denote the visual acuity which must be present if the two figures on the line are read at a distance of 5 metres. This means that, for example, the figures on the line marked 1.0 are seen under an angle of 5′ at 5 metres and their details appear under an angle of 1′, which just represents the degree of visual acuity that is regarded as normal. For number 0.5 this would be the case at 10 metres. If, however, one must bring the card up to 5 metres in order to recognize the figures on the line, he would need for this purpose twice as large a visual angle, and would therefore have only 0.5 of the normal visual acuity. This way of denoting the visual acuity differs from that of Snellen only in the fact that instead of a vulgar fraction the equivalent decimal fraction is written, e. g., V = 0.5 instead of $V=\frac{6}{12}$. The plate annexed [Fig. 365] is a reproduction of the international test types reduced one-third. Beside figures it also contains the rings proposed by Landolt. [Analogous to the latter are the old E-shaped figures which are used for illiterates and children. (See Fig. 366.) These have the advantage that they are more readily understood, especially by children, than those of Landolt and that by turning the card upside down or horizontally entirely new combinations of symbols are produced.—D.]



[Fig. 366.—Test Types for Illiterates, Reduced One-Sixth.—D.]

When the vision has become so reduced that the largest letters of the test card can no longer be recognized at 6 or 5 metres, the patient must go up nearer to it, or we select, instead of letters or numbers, objects that are easier to distinguish—e. g., the outspread fingers—and try to find at what distance they can be counted. When the visual acuity is still worse, nothing can be distinguished but the movements of the hand before the eye. When even this is no longer the case, so that the eye simply distinguishes light from darkness, we say that qualitative vision is lost and that only quantitative vision—i. e., mere perception of light—is present.

[Of particular importance are the tests of the vision in case of obstruction in the refracting media (corneal opacity, cataract). The vision] must correspond approximately to the amount of dioptric obstruction that is visible. When the opacity is so dense that only quantitative vision is present, the latter is to be tested with a candle flame. We darken the room and station ourselves with a lighted candle opposite the patient. Now, by alternately holding the hand in front of the light and then withdrawing

it, we test whether the patient can tell aright the change from light to darkness. We first make this test near by, and then withdraw farther and farther from the patient, so as to find the greatest distance at which he is still able to distinguish between the alternation of light and darkness. The degree of direct perception of light is thus determined. With regard to it we may say that the quantitative perception of light in the center and at the periphery is not abrogated by even the densest opacity. If the retina and optic nerve are sound, the glimmer of the candle must be recognized in a darkened room at a distance of at least six metres, and it should also be seen by the patient on all sides of him and its place be correctly given (see page 115). If this is not the case, the percipient portions of the eye are not normal [cf. § 855].

Küchler, in 1843, and Arlt, in 1844, were the first to introduce letters of different size (measured in lines) as a standard for determining the visual acuity. Ten years later Jäger published his scale of types, which soon acquired general acceptance, and which even at the present time is frequently employed. Practically these are very useful, since they present a great number of successive grades in the size of the letters; the objection that they are not arranged upon any scientific principle has been met recently by the issue of a modified set of them.

The test types most extensively employed are those of Snellen. Snellen based them upon the assumption that the minimum visual angle for a sound eye amounts to 1′, so that No. 6 of the test types, the details of which appear under an angle of 1′ at 6 meters, can still just be read at this distance. He therefore assumed $S=\frac{6}{6}$ as the *normal visual acuity*. But we are not to suppose that this is absolutely the greatest visual acuity that there is. Most eyes in young persons see No. 6 at a greater distance, as far as 12 metres or even farther, so that they may possibly have $S=\frac{12}{6}=2$, or more. $S=\frac{6}{6}$ is accordingly to be looked upon simply as the minimum to be required of a normal eye; if the visual acuity sinks below this minimum, the eye is no longer to be considered as perfectly normal. The eyes of very aged persons are an exception, as such people even without any disease show a visual acuity less than $\frac{6}{6}$. [At between 60 and 70 the normal vision in non-cataractous eyes may be taken as $\frac{5.5}{6}-\frac{5}{6}$, after 70 as $\frac{4.5}{6}-\frac{5}{6}$.—D.] The cause of this lies mainly in the lessened transparency of the refracting media at an advanced age, and more particularly in the very dark-colored nucleus and unequal refractivity of the lens.

In order that persons who have a vision $S>\frac{6}{6}$ may not have to go beyond a distance of 6 metres, Snellen's cards are provided with still smaller letters—namely, with the numbers from 5 to 2. These, moreover, can be used for those cases in which the physician does not have a room 6 metres in length but has at his command one of, say, only 5 or 4 metres. For persons who cannot read, there are cards with figures and with hooks. [See Fig. 366.] The international test types to indicate a visual acuity >1 have numbers 1.5 and 2.0.

[If the examining room is only 9 or 10 feet long, the distance can be doubled by placing the test card back of the patient and making him look at its reflection in a mirror at the opposite end of the room. In this case the card must have no symbols but those whose legibility is unimpaired by mirror reflection (T, X, 8, etc., or those shown in Fig. 366).—D.]

The *illumination* must be considered in making the tests for vision. Artificial illumination is the best one for the letter card, because it can always be made of the same strength, while the illumination produced by daylight varies according to the

weather and the hour of the day. On a dark day we must correct the result obtained from the patient by our own visual power. If a physician, who with good illumination has $S = \frac{6}{9}$ (International 1.0), sees on a cloudy day only $\frac{6}{12}$ (International 0.5), the visual acuity of the patient found upon the same day must also be doubled. [This, however, would not answer if the patient had hemeralopia (see page 634).—D.]

755. Absolute and Relative Visual Acuity.—To know the *absolute visual acuity* of an eye we must test it in a condition of emmetropic refraction and with the accommodation completely relaxed. If the eye under examination is not emmetropic but has an error of refraction, it must first be corrected by glasses up to the point of emmetropia (*E*). The visual acuity, which an ametropic eye shows without glasses is its *relative visual acuity*, and furnishes no measure whatever of the general usefulness of the eye for vision.

[This distinction must be borne in mind when we are testing the *sight in a case of eye disease*. As De Schweinitz well says, we shall often be misled as to the progress of disease and its effect on the sight, if, as is too often done, we test the visual acuity without attempting to correct as far as we can any existing error of refraction. In iritis, for example, in which a temporary myopia of 1 or 2 D may develop, we shall get the erroneous impression that the vision has deteriorated greatly if we test the case without applying concave glasses.

Although the relative visual acuity (i. e., the acuity of the uncorrected eye) is not ordinarily an index of the usefulness of the eye, since in most occupations glasses can be used, yet there are certain conditions in which a low relative acuity is a handicap. Thus the exigencies of certain occupations require that there shall be good vision without glasses. This is particularly true of the *military and naval service*. Except for the Marine Corps, in which a vision of $\frac{1}{20}\frac{8}{}$ without correction, brought up to $\frac{20}{20}$ with glasses, is required, and except for certain non-militant departments, the United States Navy requires of candidates for admission a vision in each eye of $\frac{20}{20}$ without correction; and gun-pointers must have $\frac{20}{15}$ without correction in the sighting eye. The Army requires $\frac{20}{40}$ uncorrected vision in the right eye and $\frac{20}{100}$ in the left, except for the ordnance and hospital corps, in which $\frac{20}{70}$ in each eye, corrigible to $\frac{20}{40}$ with glasses, is allowed. (For obvious reasons in both services persons are excluded who are color-blind or have disease of the eyes.) In other occupations, too, e.g., the railway service and manufacturing of various kinds, in which glasses are constantly obscured by dust, grease, and moisture, they are a handicap, and those who do not see well without them are at a disadvantage. Moreover, a strong glass particularly a strong cylindrical glass, if decentered or tilted causes distortion or prismatic deflection (see pages 835 and 837), and hence produces asthenopia and confusion unless the patient is careful to keep it constantly adjusted.—D.]

756. Visual Acuity at Near Points.—In determining the visual acuity for near the accommodation comes into play and must receive consideration accordingly. The testing of vision for near is made with fine print, especially with the lower numbers of Jäger's or Snellen's test types. In doing this, we try to determine two distinct things—namely, what is the smallest-sized print that can be read, and what is the least and the greatest distance at which it is legible. From the minimum distance at which the print can be seen we estimate the accommodation of which the eye is capable [although this is better and more accurately tested in another

way—see page 857], while the maximum distance depends upon the refraction and the visual power of the eye. It is evident from Fig. 363 that, the farther an object is from the eye the smaller is its retinal image, and hence the greater will be the visual power requisite for its recognition. Conversely, the retinal images enlarge as the object is approximated to the eyes. Hence, the smaller the objects are, the nearer we hold them to the eye. Very myopic eyes often have defective visual acuity, and yet pass among the laity as being excellent because they can distinguish extremely minute objects. But the only reason for this is that myopes are able to hold objects extremely close. Again, persons with diminished visual acuity like to bring objects up extremely close, so as to get quite large retinal images from them, and in this way make up for what the images lack in distinctness or the retina lacks in sensitiveness. Such persons are often wrongly regarded as very myopic.

(*c*) *Accommodation*

757. Diffusion Circles.—Suppose that we hold an open book at a distance of about 40 cm. from one eye and the point of a pencil at about half this distance between the book and the eye, the other eye in the meantime being kept closed. We can soon convince ourselves that the print of the book and the point of the pencil are never seen clearly at the same time. All that we can do is to see either the print or the point distinctly, and it takes some time to "focus" from one object to the other, and in doing this a change is felt to take place in the eye. This change is the accommodation, which is alternately thrown into a state of tension and relaxation, so that the optical adjustment of the eye is altered.

Why is it that, when we have our gaze fixed upon the print, we do not see the point of the pencil before us distinctly? Because we see it in *diffusion circles.* What does this mean? If the eye (Fig. 367) is focused for the rays emanating from the book, *B*, they are united upon the retina at *b*. The rays coming from the point of the pencil (*S*), which is nearer, have a greater divergence, and hence if the condition of the refracting media remains the same, are simply rendered somewhat less convergent by the latter; they would therefore unite at *s*—that is, behind the retina. As a matter of fact, the cone which they form has its apex truncated by the retina. The section thus made, which represents the image upon the retina of the point *s*, is circular because the base of the cone—namely, the pupil—is circular; hence, we say that the point *S* appears upon the retina under the guise of a diffusion circle. Why vision should be rendered indistinct by the diffusion circles is easy to understand. Suppose that there are two points so far distant from each other and from the eye that when the latter is accurately focused they appear as two separate punctate images upon the retina (Fig. 368 A); the points are then readily recognized

as two. But if, in consequence of the eye's being incorrectly focused for the position of either point, a diffusion circle is formed upon the retina, the two circles, provided they are but a short distance from each others partly overlap (Fig. 368 B), and the eye imagines that it has before it only one elongated point. A line (Fig. 368 C), when seen in diffusion circles, does not look distinct, but appears broadened and hazy; for we may conceive a line to be composed of an infinite number of points placed

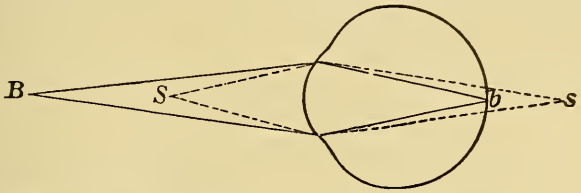


Fig. 367.—The eye being focused for a certain distance, *B*, rays emanating from a nearer point, *S*, form a diffusion circle upon the retina.

side by side, and if each one of these is seen as a diffusion circle, and the circles to a great extent overlap (D), the narrow line is converted into a broad band (E).

Vision, then, is always in diffusion circles when the eye is not properly focused for the object of fixaton. This may occur not only through improper accommodation, as in the example selected, but also on account of faulty refraction, such as myopia or hypermetropia. All indistinct vision which is caused by an anomaly of refraction or accommodation is produced by diffusion circles. The larger the diffusion circles are, the more indistinct is the vision. We must therefore inquire upon what the *size of the diffusion circles* depends:

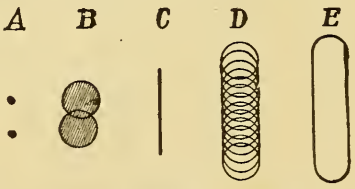


Fig. 368.—Diffusion Circles.

1. The diffusion circles get larger in proportion as the focus of the rays emanating from the object get further from the retina. Suppose (in the instance above adduced) that the eye is focused for the book, so that the point of the pencil (*S*, Fig. 369) appears in diffusion circles. If, now a second object, *P*, is brought between the book and the eye and nearer to the latter than *S* is, the adjustment of the eye for this object will vary more widely even than in the case of *S* from the condition requisite for distinct vision—that is, the rays will intersect still farther behind the retina at *p*, and the diffusion circle will be correspondingly larger. We may therefore say, the more faulty the adjustment the more remote from the retina is the point of union of the rays; hence, the further from the apex of the cone of rays is the intersection of this cone by the retina, and consequently the greater is the size of this intersection—namely, the diffusion circle.

The other factor which influences the size of the diffusion circle is—

2. The width of the pupil. The pupil forms the base of the cone of rays; the smaller it is the smaller will be the section of the cone, supposing the distance of this section from the apex to remain the same. If the pupil (Fig. 370) contracts from the size *a a* to the size *b b*, the diffusion circle of a point *P* will be at the same time reduced from $a_1\ a_1$ to b_1. When one of two equally near-sighted persons sees better at a distance with the naked eye than the other does, it is owing to the fact that the former has narrower pupils. Short-sighted persons often believe that they become

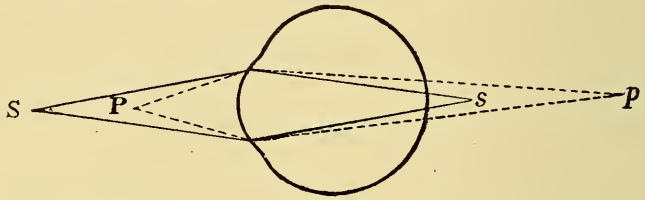


Fig. 369.—Varying Size of the Diffusion Circles According to the Distance from the Retina of the Focus of the Rays.

less near-sighted with increasing years because they see better at a distance; but this is often simply due to the circumstance that their pupils diminish in size with age. Far-sighted persons who are compelled to read close to them without convex glasses try to get as brilliant an illumination as possible, so that their pupils may become very greatly contracted and thus diminish the size of the diffusion circles. The same object is secured to a still greater extent by placing a fine stenopæic aperture before the eye. This allows only a very narrow beam of rays to pass, and reduces

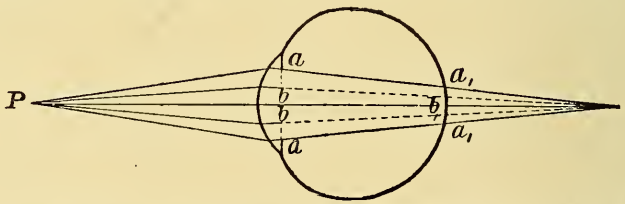


Fig 370.—Varying Size of the Diffusion Circles with Varying Width of the Pupil.

the diffusion circles so greatly that they no longer exert a disturbing effect. If we repeat the experiment made above of attempting to look simultaneously at the book and the pencil point, and while we are doing it hold a minute aperture before the eye, we see the print and the pencil point distinctly at the same time. By means of a stenopæic aperture myopic persons can see distinctly at a distance even without concave glasses.

The pupil, being the base of the cone of light, determines not only the size but also the *shape* of the diffusion circle, which accurately mirrors the shape of the pupil. In this way it happens that persons with irregular

pupils (owing, for example, to posterior synechiæ) are very well able themselves to perceive these irregularities entoptically.

758. Accommodation and its Mechanism.—In making the above experiment with the pencil and book, we feel that the eye is called upon to make an active effort when it is directed by a process of adjustment from the more distant book to the less distant pencil. In the same way, although not quite so distinctly, we feel a relaxation of this effort when adjustment is made for the book again. It may be concluded from this that the change of adjustment from a more distant to a less distant point is an active process —i. e., a muscular effort, which we call accommodation. On the other hand, the relaxation of the accommodation by which the eye is again adjusted for a greater distance consists in a relaxation of the contracted muscle. When in a state of perfect rest, the emmetropic eye is adjusted for infinite distance. This condition of adjustment we find existing when the ciliary muscle is completely relaxed, either naturally from paralysis of the oculo-motor nerve, or artificially from the use of atropine.

The *mechanism of accommodation* was determined mainly by the investigations of Helmholtz. It depends upon the elasticity of the lens, owing to which the latter always tends to approximate to the shape of a sphere. In the living eye the lens is inclosed in a capsule which is attached to the ciliary body by the fibers of the zonula of Zinn. These fibers are tightly stretched, and hence exert a uniform traction from all sides upon the capsule, so that the latter and the lens as well are flattened. The elasticity of the latter can make itself apparent only when the tension of the fibers of the zonula, and hence, too, of the capsule of the lens, is relaxed. This takes place most completely after division of the fibers of the zonula. When we remove the lens from the eye of a young person, we see it assume a spherical shape immediately upon the division of its connections. The same thing is observed in traumatic luxation of the lens into the anterior chamber. In the act of accommodation, the relaxation of the zonula is effected by the contraction of the ciliary muscle. It is the annular layer of fibers of the latter (Müller's portion; see *Mu*, Fig. 142) that is mainly of account in accomplishing this. When this fiber layer contracts, it lessens the size of the circle formed by the ciliary processes by approximating their apices to the border of the lens (shown by the black line in Fig. 371). In this way the space between the ciliary body and lens, that is bridged over by the fibers of the zonula, is contracted and the zonular fibers themselves are relaxed. It is the task of longitudinal fibers of the ciliary muscle (Brücke's portion; *M*, Fig. 142) to re-enforce the action of the circular fibers. The former have their anterior, fixed insertion in the corneo-scleral margin, while their posterior extremity loses itself in the movable chorioid. By the contraction of these fibers the flat portion of the ciliary body and the most anterior portion of the chorioid are drawn forward, and thus the

relaxation of the fibers of the zonula which lie upon the surface of these structures is facilitated; but the main part of the work of accommodation always falls upon the annular fibers of the ciliary muscle, for which reason we find these fibers particularly well developed in eyes which have to accommodate a good deal—e. g., those of hypermetropes (see Fig. 384).

By the relaxation of the zonula the tension of the lens capsule is diminished, so that the lens is enabled in conformity with its elasticity to assume a more curved shape. At the same time there is necessarily produced a corresponding decrease in the equatorial diameter of the lens. The equator of the lens, accordingly, recedes inward toward the axis of the eye, and is thus kept from coming into contact with the ciliary processes as they advance.

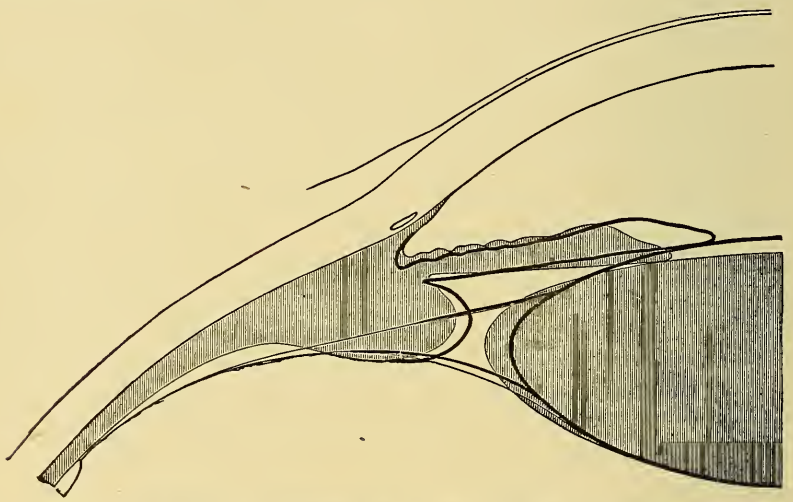


Fig. 371.—Schematic Representation of the Process of Accommodation.

The relation of the parts when the accommodation is at rest is designated by the shaded portions, and the relation when there is an effort of accommodation, by the thick black lines In the drawing it looks as if the zonula fibers were running through the ciliary process. In reality the fibers of the zonula for the most part lie in the depressions between the ciliary processes, and hence run to the lateral slopes of the ciliary process that is represented in the drawing. With these slopes they are connected by numerous fibers and hence, when the ciliary processes advance toward the sagittal axis of the eye, the zonula fibers are taken along with them and are likewise pushed inwards; and to a corresponding amount the equator of the lens recedes toward the sagittal axis of the eye. Both surfaces of the lens become more curved and the anterior surface is advanced. The iris is broader and at its pupillary border is displaced forward; at its ciliary border backward.

[Tscherning believes, in opposition to Helmholtz, that contraction of the ciliary muscle causes a tightening, not a relaxation, of the zonula and that, consequently, the bulging of the lens in accommodation is produced by an active compression instead of a passive dilatation. But experiments seem to show that this theory is erroneous, and that the explanation given by Helmholtz and described in the text is the true one (Hess).

The Helmholtz theory, however, does not explain all the phenomena of presbyopia (see page 861).—D.]

The increase in curvature affects both the anterior and the posterior surface of the lens, but the former to a [much] higher degree (Fig. 371). The posterior surface of the lens does not change its place in the fossa patelliformis of the vitreous, the increase in thickness of the lens being effected simply by the advance of its anterior surface. Hence, the anterior chamber becomes correspondingly shallower; at the periphery alone is there a deepening of the chamber, inasmuch as here the iris recedes a little. The sphincter pupillæ and, if vision is performed with both eyes, the two internal recti also, contract in conjunction with the ciliary muscle. The act of accommodation, accordingly, is regularly accompanied by a contraction of the pupils and a movement of convergence.

759. Measurement of the Accommodation.—In order to measure the magnitude of accommodation we must determine its limits, which are defined by the far point and the near point. The far point (*R*, punctum remotum [3]) is that point for which the eye is focused when the accommodation is completely relaxed. The near point (*P*, punctum proximum) is that point for which the eye is focused when the accommodation is strained to its utmost.

In the emmetropic eye, with which alone we shall for the present concern ourselves, *R* lies at an infinite distance, since the emmetropic eye when in the state of rest is focused for parallel rays. Such an eye, accordingly, can see the letters of the test card distinctly when the latter is hung up at such a distance (5 or 6 metres) that no accommodation worth considering is required, so that this distance in practice is regarded as infinitely great.

While the position of *R* is the same for all emmetropic eyes, that of *P* varies greatly. It is determined by bringing fine print closer and closer to the eye until the limit of legibility has been reached. Suppose, for example, this occurs at 10 cm. ($P = 10$ cm.). The space lying between *R* and *P*—i. e., in the example selected the space between ∞ and 10 cm.—is called the *region* or *domain of accommodation*. But the extent of this space affords no measure for the amount of work done by accommodation; this, in fact, being measured by the increase of refractive power which the eye undergoes in passing from the state in which the accommodation is at rest (*R*) to the state in which the utmost effort of accommodation is made (*P*). The amount of increase of refractive power is called *amplitude* [or *range*] *of accommodation*

[3] The far point receives its name because of the fact that for the emmetropic and myopic eye it is the farthest point at which the eye can see distinctly. This definition, however, is not applicable in hypermetropia, in which the far point lies behind the eye. If the accommodation is put into play then in proportion as the hypermetropia is corrected by it, the point for which the eye is adjusted recedes from the eye until it has got out to infinite distance. If the accommodation is not strong enough to adjust the eye for infinite distance (absolute hypermetropia), the near point, too, lies behind the eye and in fact at a greater distance from the latter than is the far point. As an example of this may serve an eye with *H* of 4 D, whose region of accommodation is represented in Fig. 388 A. If this eye should have an accommodation of only 2 D, its near point would be 50 cm. behind the eye, and with a range of accommodation of 4 D, it would be at infinite distance; i. e., in either case would be farther than the far point, which lies but 25 cm. behind the eye. For this case, therefore, the expression near point is not very sensibly chosen.

(A), and is accordingly the difference between the refractive power of the eye when the accommodation is exerted to its utmost and when it is at rest—i. e., $A = P - R$. For R and P in this equation should be substituted, not their linear values, but the corresponding number of dioptries; these being, in fact, our measure of the refracting power.

The method of determining the amplitude of accommodation requires some explanation which is best given by concrete examples. Let us assume the three following cases represented graphically in Fig. 372. 1. A young emmetrope whose far point lies at an infinite distance and near point at 10 cm. from the eye; 2. An older emmetrope whose $R = \infty$ and $P = 20$ cm.; and lastly, 3. A young myope, whose $R = 10$ cm. and $P = 5$ cm. The region of accommodation—i. e., the space lying between R and P—is of a very different extent in these three cases. In cases one and two it is infinitely large, since it extends to an infinite distance, while in case three it amounts to only 5 cm. If, therefore, we were to reckon the work done in accommodation by the extent of the region of accommodation, we would arrive at the erroneous view that, with regard to the former as well as the latter, there is an enormous difference between the first two cases on the one hand and the third case upon the other.

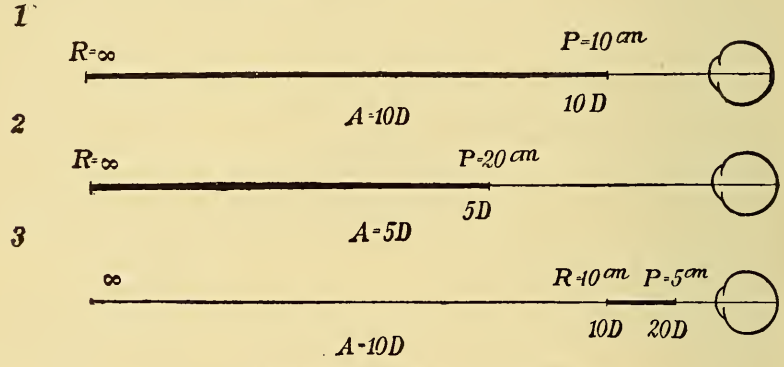


Fig. 372.—Region of Accommodation.
1, In a young emmetrope; 2, an older emmetrope; 3, a young myope.

But, as a matter of fact, the case is quite different, as can be gathered from the experimental test of the accommodation adduced above. Suppose that while we close one eye we hold before the other a book at a distance of 20 cm. and a pencil point midway between the book and the eye—i. e., at a distance of 10 cm. from the latter. We then first look out into space over the book, so that the accommodation is completely relaxed, and then fix our gaze upon the print of the book. In so doing, we have a sense of accommodative effort in the eye. We now turn our glance from the book to the pencil point, and endeavor to see the latter distinctly. If this is possible at all, it costs us a very considerable effort which will tax the energies of most persons more than does the effort required to adjust the eye from infinite distance to the book. Hence, the act of changing the accommodation from 20 cm. to 10 cm. costs us at least as much effort as the change from infinity to 20 cm. From this it is clear that we are not justified in taking the linear distance between the points of fixation as a measure of the accommodative effort, and that, therefore, the region of accommodation can not serve as an expression of the work done in accommodation.

We get a correct idea of the amount of accommodation that is called into play if we take into consideration the increase produced in the refractivity of the eye by

the accommodation. This increase of refractivity is effected by an increase in the curvature of the lens, a thing which we can also conceive of as accomplished by the addition to the unchanged lens of a second, weaker convex lens. This "supplementary" crystalline lens (*z*, Fig. 373 A) represents the increase in refractivity, and would form the best measure of the accommodation. Now, of course, we can not determine the refractive power of this supplementary lens directly, but we can determine what glass placed before the cornea of the eye would produce the same increase of refractive power as would such a supplementary lens, conceived to exist in the eye. What proceeding to adopt in doing this may be shown by case one of those assumed above (Fig.

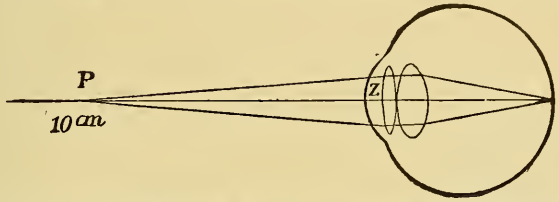


Fig. 373 A.—Accommodation represented schematically by a "Supplemental" Crystalline Lens, *z*.

372). In this, when the accommodation is making its utmost effort, the refractivity of the eye is increased by the supplementary lens to such an extent that rays emanating from *P*—that is, from a distance of 10 cm. from the eye—are united upon the retina (Fig. 373). We now paralyze the accommodation in this eye with atropine, so that the eye remains steadily focused for infinite distance, and try to find the convex glass with which the eye is enabled to see the point, *P*, distinctly. We find that for this purpose a glass, *L*, of a focal distance of 10 cm. = one of 10 D is necessary (Fig. 374). If this glass is placed in front of the eye—i. e., 10 cm. behind the point *P*—the latter will lie just in the principal focus of the glass.[4] The rays (*p p*, Fig. 374) emanating from *P* are

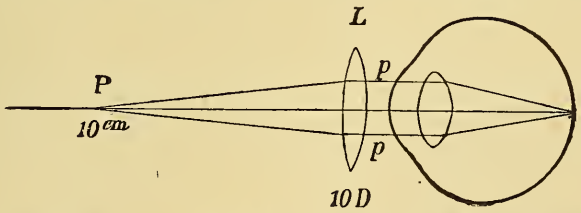


Fig. 374.—The Accommodation replaced by a Lens of Glass, *L*, set before the Eye.

hence made parallel by the glass, and being so, are united by the non-accommodating eye upon its retina. The lens *L*, therefore, does the same work as the natural accommodation (represented by the supplementary crystalline lens, *z*), and can accordingly be taken as the measure of the latter. Accommodation measured in this way we call *amplitude* [or *range*] of accommodation. This, therefore, would be in the first case $A =$ 10 D. In the second case (Fig. 372, 2) as may be shown in the same way, $A = 5$ D. Hence, in the emmetropic eye, *A* is expressed by the lens whose focal distance equals the distance of the near point from the eye; or $A = P$, when *P* is expressed in dioptries.

What relation does *A* bear in the third case (Fig. 372, 3)? Here *P* is situated at 5 cm., and hence $P = 20$ D. But this value can not be regarded as the expression of the work done in accommodation, since the eye in question being short-sighted,

[4] Properly, if we take into account the distance of the lens from the eye, we should have to choose a lens of shorter focal distance—e. g., if the distance between the lens and the nodal point of the eye amounts to 1 cm., one of 9 cm.

is, even when the accommodation is at rest, adjusted for a distance less than infinity —i. e., for 10 cm. This eye, when its accommodation is at rest, acts like a non-accommodating emmetropic eye before which has been placed a lens of +10 D (Fig. 374). For this eye, therefore, we can say $R = 10$ D. But when making its utmost accommodative effort, this eye acts like a non-accommodating emmetropic eye before which has been placed a glass of +20 D. Obviously, in order to determine the work done in accommodation—i. e., the increase in refractivity in passing from R to P—we must subtract from the value corresponding to P the value of R, or $A = 20$ D—10 D = 10 D. Hence, we may enunciate the following formula as representing the general law:

$$A = P - R,$$

in which P and R are expressed in dioptries. This formula holds good for all conditions of the refraction. For the emmetropic eye it is simplified by the fact that the latter, when the accommodation is at rest, is adjusted for infinite distance, so that $R = 0$ D, and consequently $A = P$, as we found above.

Let us take another survey of the three cases, and compare the region of accommodation with the range of accommodation. We find that the former is infinitely large in the first two cases, and only 5 cm. in the third case. Now, A in the first case is 10 D, in the second only half as great—i. e., 5 D—and in the third case again is as great as in the first. Accordingly, from the range of accommodation we get an entirely different and in fact a more correct conception of the work done in the accommodative act than we do from the region of accommodation. This is owing to the fact that different portions of the region of accommodation represent entirely different values. It takes as much accommodative effort to bring the accommodation from 10 cm. to 5 cm. (case three) as to accommodate from infinity to 10 cm. (case one); the value in both cases being 10 D. This fact is in harmony with the sensations that we experience in our eyes when, in the experiment previously adduced, we look successively at infinite distance, at the book, and finally at the pencil point. Displacement of the accommodation through 1 centimetre of the region of accommodation is significant of an effort which is the greater, the nearer this centimetre is situated to the eye.

The region of accommodation, however, gives us a good idea of the *availability of accommodation.* In case one the region of accommodation is so situated that the eye sees clearly at all distances which can be considered to exist in practical life. But in case three the region of accommodation lies so close to the eye that practically it has no value whatever; this eye would be no worse off without accommodation. (This, of course, holds good only upon the supposition that correcting glasses are not worn, as by these the location of the region of accommodation is shifted.)

760. Determination of the Far and Near Points.—The determination of the *far point* is synonymous with the determination of the refraction, for the latter is, in fact, the refractive state of the eye when focused for the far point. If we know the refraction R is determined too.

The determination of the refraction is accomplished by means of the test card, hung up at 6 (or 5) metres. Emmetropia ($R = \infty$) is present if the smallest number which the eye that is being examined can see at all is distinguished with the naked eye but is made dimmer at once by the weakest convex glass. If the letters on the test card are seen as well with convex glasses as with the naked eye or are seen better, there is hypermetropia (R is at a finite distance behind the eye); if they are seen better with a concave glass there is myopia (R is at a finite distance in front of the eye). In both cases it is then necessary to determine the correcting glass whose focal length is equal to the distance from the eye. [A hypermetropia can be persistently masked and a myopia

persistently heightened by the accommodation, so that the refraction and therefore the precise situation of *R* cannot in the majority of cases be determined without paralyzing the accommodation with atropine or homatropine. See pages 866, 881, 901.—D.]

The *near point* is determined by the low numbers of the reading tests, which are brought up to the eye until they cease to appear distinct. [A better test-object is the accommodation disk (Fig. 375). The disk is approximated to the eye until the black line on it which is 0.2 mm. in diameter begins to grow faint. The distance at which this occurs is measured with a rule graduated in cm. and D (Prince's rule). The measurement is preferably taken not from the eye itself but from its anterior focus (13 mm. in front of the cornea), as this is the point where glasses that are designed to replace and hence to measure the accommodation are placed. Except in the case noted in the next paragraph, the eye is provided with the full correction, i. e., is rendered emmetropic, so that R = 0 D and P (read off in D) gives at once A or the total range of accommodation.—D].

[Fig. 375.—Accommodation Disk. Natural size.—D.]

When *P* has receded to such a distance from the eye that small objects like fine print appear under too minute an angle, and hence can not be seen distinctly at all, the following expedient is adopted: We place before the eye a convex glass—e. g., one of 3 D, by which near vision is rendered distinct—and then determine the near point. This is found to be, say, 25 cm. The refraction corresponding to this distance is 4 D, from which, in order to learn the actual near point, we must subtract the 3 D supplied by the lens. Thus $P = 4\text{ D} - 3\text{ D} = 1\text{ D} = 100\text{cm}$. [If with such a convex addition the test object (accommodation disk) is carried away from the eyes until it blurs, we can likewise determine *R*. Suppose, for example, that with a +4 D before the eye the disk line just begins to blur at the point on the Prince's rule marked 4.50. Then $R = 4.50 - 4.00 = +0.50$; i. e., there is 0.50 D myopia. If the line had begun to blur at the point marked 3.25, *R* would have been $3.25 - 4.00 = -0.75$; i. e., there would be 0.75 D of hyperopia. If *R* and *P* are both taken in this way the range is given directly. Thus if with +3.00 added, R is at 3.50 and *P* is at 5.00, $A = 1.50$. This method is particularly valuable for determining the residual range when the eye is wholly or partly under the influence of a cycloplegic (see page 901).—D.]

761. Relative Accommodation.—In the course of our considerations in regard to the accommodation hitherto, we have started from the assumption that vision is performed with only one eye. When the two eyes are employed simultaneously the convergence as well as the accommodation must be taken into account. These two functions go hand in hand. When our gaze is fixed upon the distance, $A = 0$, and the visual axes are parallel—i. e., the convergence, too, is in a state of rest. When we look at a near point—e. g., one situated at a distance of 29 cm.—we are compelled both to accommodate and to converge for this distance. Hence, through constant practice an intimate connection is effected between accommodation and convergence, so that with any given accommodation the corresponding effort of convergence is always made, and vice versa.

This connection, however, is not one that is rigid and insusceptible of change. On the contrary, we have the ability of emancipating ourselves from it within certain limits—that is, while in the act of converging for a certain distance, of making a little more or a little less accommodation than corresponds to this distance. A man is made to fix his gaze upon fine print at distance of 33 cm. Now, let us suppose that the subject is emmetropic and thus has his far point (*R*) at infinity, while *P* is situated at 10

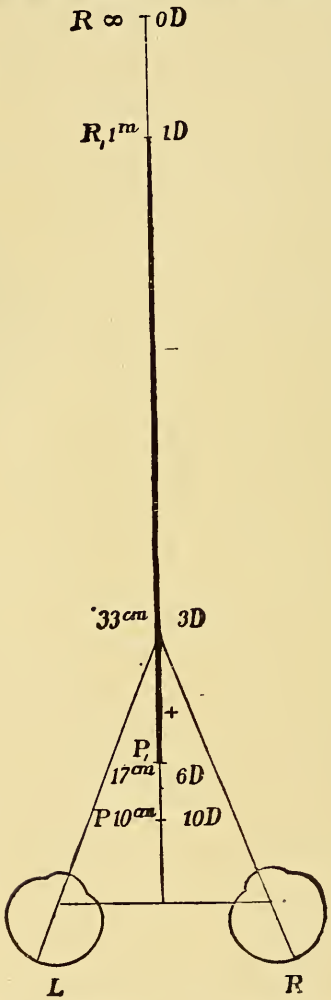


Fig. 376.—Schematic Representation of Relative Accommodation.

cm., which corresponds to a range of accommodation (*A*) of 10 *D* (Fig. 376). Of this range of accommodation 3 D (100 ÷ 33 = 3) will be employed if the subject is converging for a distance of 33 cm. (=3 metre angles, see page 722). Now, a concave glass of 1 D is placed before each eye. The subject will for the first moment have obscuration of vision, but will soon see distinctly. He has compensated for the diminution of the refractive power of this eye, caused by the −1 D glass, by exerting 1 D more of his accommodation. But the convergence meanwhile remains unchanged—i. e., it is still adjusted for 33 cm. The same phenomenon occurs when, instead of −1 D, a glass of +1 D

is placed before the eye. The refractive power of the eye is made too great by the convex glass, and this is neutralized by the eye's relaxing its accommodation through 1 D. In this way we can bring stronger and stronger convex and concave glasses successively before the eye until finally we come to those with which distinct vision is no longer possible. We thus find the limits within which the accommodation may be augmented or relaxed, the convergence remaining the same (*relative accommodation*).

In the example selected, suppose that the subject is able to see distinctly at a distance of 33 cm. with a convex glass of 2 D. This corresponds to a relaxation of his accommodation from 3 D to 1 D—that is, his relative far point, R_1, lies at a distance equivalent to 1 D, or 1 metre, from the eye. Suppose on the other hand, that the subject with the same degree of convergence overcomes concave glasses of 3 D, a thing which is effected by an augmentation of the accommodation from 3 to 6 D; his relative near point, P_1, then is at a distance equivalent to 6 D = 17 cm. The relative range of accommodation $A_1 = P_1 - R_1 = 6\text{D} - 1\text{D} = 5\text{D}$. This is the relative range of accommodation for a convergence of 33 cm.; for a different convergence the relative near point, far point, and range would be different still. On the other hand, there is only one absolute far point, near point and range of accommodation.

The range of relative accommodation is divided by the point upon which convergence is made into two segments. One lies upon the proximal side of the point of fixation, and thus in the example selected extends from 3 to 6 D. It represents the amount of accommodation which, if necessary, one can still press into service while keeping the convergence the same—i. e., it represents the amount of accommodation which one may be said to have in reserve. It is hence denoted as the positive portion (+, Fig. 376) of the relative range of accommodation. The other segment lies on the distal side of the point of fixation, and in our case extends from 3 to 1 D. It is the negative portion of the relative accommodation (−, Fig. 376). Hence, with a convergence of 33 cm. the positive portion of the relative accommodation amounts to 3 D, the negative portion only to 2 D.

[It is usually held that it is impossible to use the eyes continuously except for a distance at which the positive portion of the accommodation is at least as great as the negative, since otherwise the patient is using too much of his total accommodation, i.e., working too near the limits of his capacity. This statement holds good for young subjects at most, As a man grows older, more and more of his accommodation becomes latent (see page 862) and even when working at his near point he is not using anywhere near all of his ciliary power, i. e., is not near the limit of his working capacity. It is true that persons of any age often experience a good deal of discomfort in working at close ranges, but this is due in most cases not so much to accommodative fatigue as to fatigue of the converging muscles.—D.]

Changes of the Accommodation with Age

762. The accommodation diminishes with age, and this diminution is manifested by a continuous recession of the near point. The diminution in the accommodation can not be referred to the decrease in the power of the muscles in general and the ciliary muscle in particular, occurring in old age, for it begins in youth, and probably even in childhood—that is, at the time when the muscles are still gaining in strength. In fact, the cause of the diminution of the accommodation lies in the gradual decrease of the elasticity of the lens. This, again, stands in causal connection with the condensation of the lens due to loss of water and leading to a process of sclerosis that begins in the center of the lens (formation of a nucleus). The harder the lens becomes in virtue of this process the more its elasticity is impaired, so that even after the zonula is released the lens becomes less and less able to change its shape.

The state of the accommodation at different ages is shown in Fig. 377 and the accompanying table.

Normal limits of the accommodation in D, near point being measured from the anterior focus of the eye, i. e., from a point 13 mm. in front of the cornea.

Age	Lower Limit	Mean Value	Usual Upper Limit	Extreme Upper Limit
8	11.7	13.8	15.4	16.4
9	11.6	13.6	15.2	16.2
10	11.4	13.4	15.0	16.0
11	11.2	13.3	14.9	15.8
12	11.1	13.1	14.7	15.6
13	10.9	12.9	14.5	15.4
14	10.8	12.7	14.3	15.3
15	10.7	12.6	14.1	15.2
16	10.5	12.4	13.9	14.9
17	10.3	12.2	13.7	14.6
18	10.1	11.9	13.5	14.4
19	9.9	11.7	13.2	14.2
20	9.7	11.5	13.0	14.0
21	9.4	11.2	12.8	13.7
22	9.2	10.9	12.6	13.5
23	8.9	10.6	12.3	13.2
24	8.7	10.4	12.1	13.0
25	8.4	10.2	11.8	12.7
26	8.2	9.9	11.6	12.4
27	7.9	9.6	11.3	12.1
28	7.6	9.4	11.1	11.8
29	7.3	9.2	10.7	11.5
30	7.1	8.9	10.4	11.2
31	6.7	8.6	10.2	10.8
32	6.4	8.3	9.9	10.5
33	6.1	8.0	9.6	10.2
34	5.9	7.7	9.2	9.9
35	5.6	7.3	8.9	9.6
36	5.3	7.1	8.6	9.4
37	4.9	6.8	8.2	8.9
38	4.6	6.5	7.9	8.6
39	4.3	6.2	7.6	8.2
40	3.9	5.8	7.2	7.8
41	3.6	5.4	6.8	7.5
42	3.2	5.0	6.4	7.0
43	2.8	4.6	5.9	6.5
44	2.5	4.2	5.5	6.1
45	2.2	3.7	5.2	5.6
46	1.9	3.3	4.8	5.1
47	1.7	2.8	4.3	4.5
48	1.5	2.5	3.9	4.0
49	1.3	2.2	3.4	3.4
50	1.2	2.0	3.0	3.0
51	1.1	1.8	2.6	2.6
52	1.0	1.6	2.3	2.3
53	0.9	1.5	2.1	2.1
54	0.9	1.4	2.0	2.0
55	0.8	1.3	1.9	1.9
56	0.8	1.2	1.8	1.8
57	0.8	1.2	1.7	1.7
58	0.7	1.2	1.7	1.7
59	0.7	1.1	1.6	1.6
60 to 68	0.7	1.1	1.5	1.5

The diminution in the range of accommodation goes on from youth to age with perfect uniformity and not by sudden advances. It begins to be troublesome when the near point has receded so far from the eye that the finer kinds of work, and especially the reading of fine print, become difficult or impossible, so that there develops a desire for glasses. This is the

case when the near point recedes beyond one-third of a metre ($A = 3$ D), i. e., between the ages of forty-five and fifty. This is the period, therefore, to which we assign the beginning of *presbyopia*[5].

[In age, owing to lenticular changes (and particularly the fact that the lens becomes more homogeneous) the eye often becomes more hyperopic or less myopic—i. e., the far point as well as the near point recedes (Donders). Cf. page 883.—D.]

[The ordinary hypotheses with regard to presbyopia leave some things unexplained.

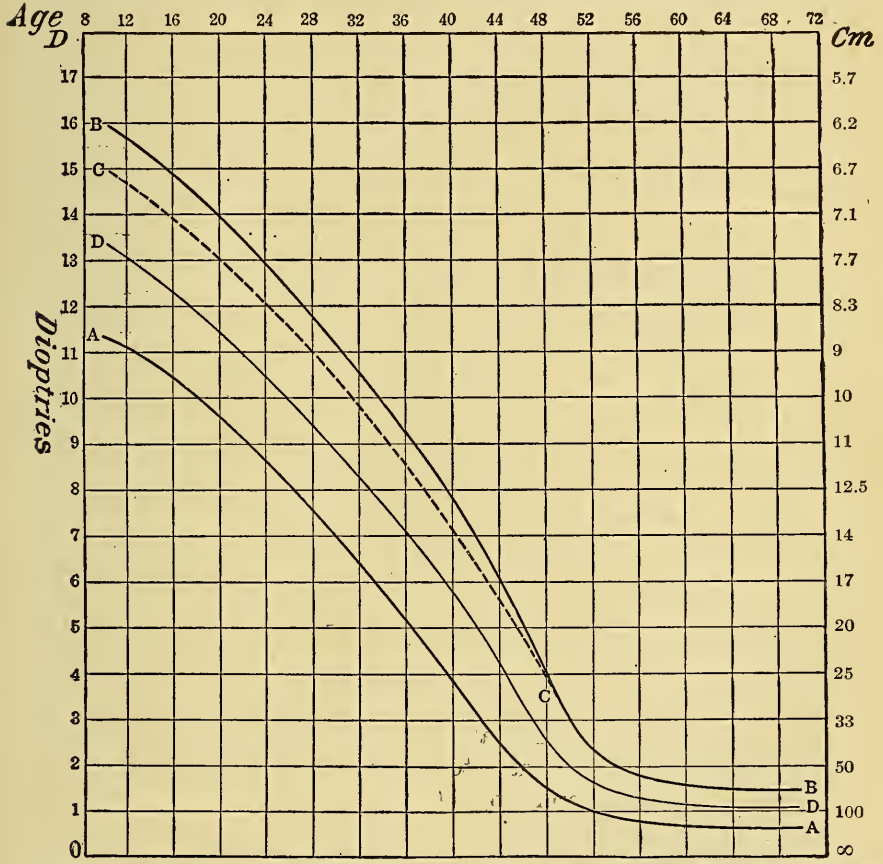


Fig. 377.—Range of Accommodation at Different Ages. After Donders, amended by Duane.

The numbers on the left hand give the strength of the lens, which placed before the emmetropic eye, at a distance of 13 mm. from the apex of the cornea (i.e., placed at the anterior focus of the eye), can replace the accommodation of the eye at the given age, and hence is equivalent to the accommodation, so far as regards the increase of refractivity in the eye which the latter produces. These numbers, therefore, give the range of accommodation of the eye. The right-hand numbers give the focal distances of these lenses in cm. The distance of the near point from the apex of the cornea is found by adding to the focal length of the lens its distance from the cornea (i.e., 13 mm.). Obviously no single observer can follow the progress of the range of accommodation from youth to age, in one and the same individual. The progress of accommodation can, therefore, be found only by determining it in a large number of persons with normal eyes, at different ages and taking the mean of the observations. This is shown by the line DD, which indicates the mean position of the near point from the anterior focus of the eye and also the range of accommodation in D, of an emmetropic eye at different ages. The line AA gives the least values, the line CC the ordinary maximum, and the line BB the extreme maximum values of the accommodation that have been found in the individual cases. They accordingly show the limits within which the position of the near point and hence too the accommodation can still be regarded as normal. [An accommodation which in any given subject persistently falls below the lower limit indicated by AA for his age, must be considered as pathologically weak.—D.]

[5] From πρέσβυς, old man, and ὤψ, sight.

According to Helmholtz accommodation is produced by a passive expansion of the lens due to an active contraction of the ciliary muscle. The more the muscle contracts, the more the lens expands. But after the lens has reached the limit of its expansion, further contraction of the ciliary muscle produces no appreciable effect. Hence we may divide the ciliary energy into two parts—one producing an effect noticeable in accommodative effort (manifest energy), the other producing no effect (latent energy). Obviously, the less able the lens is to expand,—i. e., the older the subject, the less manifest ciliary energy will be required and the greater will be the amount that is latent. Thus suppose we have three persons, aged respectively 15, 25, and 40, having, all three, an amount of ciliary muscle energy, which if unrestricted would produce an accommodative effect of 20 D. Owing now to the differing states of their lenses, the first has an accommodation of 15 D, the second of 10 D, and the third of 5 D, so that the amount of latent or unused energy is 5 D in the first, 10 D in the second, and 15 D in the third. Suppose further that we paralyze the ciliary muscle in all three by a gradually acting poison like homatropine. If the Helmholtz theory is correct, this will have to abolish all the latent ciliary energy before it produces any effect on the accommodation that we can appreciate. It should then have a very different effect on the three persons. In the first it should show its effect early since there are only 5D of latent ciliary energy to be abolished. In the second, it should occur much later, and in the third later still or perhaps hardly be noticeable at all. Now as a matter of fact, repeated experiments show that the paralysis of accommodation will begin to be apparent at about the same time and will advance at the same rate in all three.—D.]

Presbyopia is not a disease, but a physiological process which every eye undergoes. Persons who are presbyopic push the book farther away from them, like to avoid fine print, and pass over the footnotes. Reading at night gives them special trouble, because the pupils dilate owing to the enfeebled illumination, and hence the diffusion circles are larger. They then try the expedient of bringing the light between the book and their eyes, so as to make their pupils contract by having a large amount of light fall upon them. In the subsequent progress of presbyopia reading or fine work at last becomes absolutely impossible without glasses. Pain, however, or asthenopia [usually] does not occur, as it does in hypermetropes.

When an eye is not emmetropic, but has an error of refraction, this error shifts the range of accommodation, and hence also the time when presbyopia begins. Reference in regard to this point must be made to the chapters upon myopia and hypermetropia (pages 868, 885).

763. Correction of Presbyopia.—Presbyopia requires the use of convex glasses for near work. The glass must be strong enough to make the near point come into the place which appears demanded by the work in question. The situation of this depends primarily upon the nature of the work; the finer this is, the closer must the near point be approximated. But in addition to this the visual acuity must be taken into account. If the latter is defective, objects must be brought nearer, so as to make up in size what the retinal image lacks in distinctness.

From the reasons set forth above it follows that it will not do simply to prescribe for each separate age the convex glass that ought to be ordered. On the contrary, we must proceed according to the individual requirements of each case by itself, and determine the glass for it specially. Suppose, for instance, that the subject is sixty

years old, and that his near point is one metre from the eye ($A = 1$ D). The man is a cabinet maker, and in doing his work, which he carries on at arm's length—i. e., about 50 cm.—no longer sees distinctly. His near point, therefore, must be brought up to 50 cm. (=2 D). As he himself can furnish 1 D, it suffices to give him a +1 D glass (or, better still, 1.5 D, so that he may not have to work at his near point, but may have some accommodation in reserve). Perhaps the same man also wishes a glass to read with in the evening when his work is done. For this purpose we must bring his near point up to at least 30 cm. (3.5 D), so that he can read ordinary print with ease, and we would therefore recommend him a glass of +2.5 D or +3 D. [The strength of glass prescribed will also depend somewhat upon the state of the eye muscles. A patient with marked convergence-insufficiency will often read more comfortably with a weak glass than with one which gives him better sight, because the weak glass enables him to carry his book so far off that he has to converge his eyes but little in looking at it. Many people who put on convex glasses for the first time develop a temporary convergence-insufficiency from relaxation of their accommodation (see page 783). This insufficiency may give rise to asthenopia, which has to be relieved by reducing the strength of the reading glasses.—D.]

[The determination of the proper reading glass in presbyopia requires much care and patience. First the full correction for distance must be determined accurately. Then the patient wearing this correction is placed as nearly as can be under his normal working conditions and with his work at the habitual distance. Then different convex glasses are added to the distance correction until an addition is found that gives the most serviceable and comfortable (which is not always the sharpest) vision. With each addition it is well to determine the near point and corresponding amount of accommodation with the accommodation disk (page 857). As stated above the correction will vary greatly with circumstances, but, as a rule, except in the younger presbyopes with a range of 2 or 3 D, a glass which brings the near point within 28 cm. (over 3.5 D) is not well borne, and, other things being equal, the glass that gives the widest and most serviceable range of vision is the best. Sometimes the accommodation test shows a constant difference in the maximum accommodative power of the two eyes, and in that case it may prove best to make a somewhat stronger addition for reading in one eye than in the other (see page 910).—D.]

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764. Ametropia.—Optical variations of the eye from the normal may relate either to its refraction or to its accommodation. The anomalies of refraction are to be rigidly differentiated from those of accommodation, with which they are frequently confounded. An eye whose refraction varies from the normal or emmetropic we call *ametropic*[6]. There are three varieties of ametropia: Myopia, hypermetropia, and astigmatism. When the refraction of the two eyes is different, we speak of anisometropia.

[For the importance not only of correcting ametropia, but also of recognizing its presence and its effect on the visual acuity when we are treating cases of eye disease, see page 847.]

[6][From ἀ, privative, μέτρον, a limit, and ὤψ, sight.]

CHAPTER III

MYOPIA

765. Definition.—Short-sightedness (*myopia, M*) is that refractive condition of the eye in which rays that are parallel to each other when they fall upon the eye come to a focus *in front* of the retina. Hence, when the rays strike the retina, they have already become divergent, and therefore form a diffusion circle upon the latter ($a\ a_1$, Fig. 378). A distinct image is formed upon the retina only when the rays have a certain degree of divergence as they arrive at the eye, which is the case when they emanate from a point R situated close by (Fig. 378). This point is the far point of the myopic eye—i. e., is the point for which the eye is adjusted when in a state of accommodative repose. Accordingly, the far point in this case lies at a finite distance. The greater the myopia, the farther in front of the retina is the point of intersection of parallel rays, and hence the greater is the divergence that rays must have in order to unite upon the retina, and hence, too, the nearer to the eye must the far point (R) lie. Consequently the degree of myopia is determined by the distance of R.

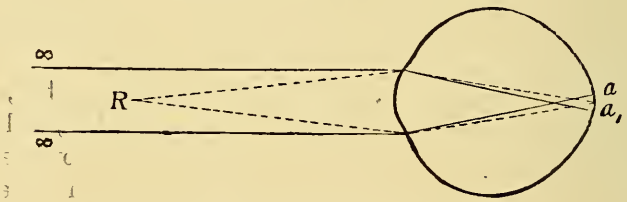


Fig. 378.—Path of the Rays in a Myopic Eye.

766. Determination.—The distance of R from the eye can be measured directly by placing fine print before the eye and gradually withdrawing it until it becomes too indistinct to be read. This method, however, has serious defects, so that we prefer to determine the position of R by means of concave glasses. Let us assume that the eye has such a degree of myopia that its far point is 50 cm. in front of the eye (Fig. 379, F); the rays, then, that emanate from this point come to a focus upon the retina (at f). How can we manage to have this eye see parallel rays distinctly—i. e., have them focused upon the retina? Evidently, by giving them the same direction as if they emanated from the far point. This is effected by placing before the eye a concave glass of 50 cm. focal distance—i. e., of — 2 D. By this glass parallel rays are rendered as divergent as if they emanated from its focus (see page 831). This lies 50 cm. in front of the glass—that is, at the same spot at which the far point of the eye is situated (at f). Accordingly, par-

allel rays acquire the same direction as if they emanated from the far point of the myopic eye, and are hence focused upon the retina of the latter so as to form a distinct image. (In this discussion the distance of the glass from the eye is neglected.)[1]

The deduction made in the foregoing example holds good for all degrees of myopia. The following rule may therefore be enunciated: A myopic eye sees distinctly at infinite distance with that concave glass whose focal length is equal to the distance of the far point from the eye. And conversely: The focal length of that concave glass with which the myopic eye sees remote objects distinctly gives the distance of the far point from the eye—i. e., the degree of myopia. If a man sees remote objects distinctly with—5 D, his far point is 20 cm. (100 ÷ 5 = 20). But in designating the degree of myopia we do not usually give the position of the far point, but give directly the relative power of the correcting glass—writing, therefore, $M = 5$ D.

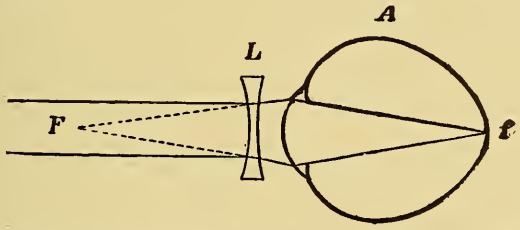


Fig. 379.—Correction of Myopia by a Concave Glass.

If a glass of—6 D is placed before an eye whose $M = 5$ D, such an eye would still see clearly at a distance; the extra strength of 1 D being neutralized by a corresponding effort of the accommodation. Since in myopia there is often a tendency to accommodate, it is by no means rare to find myopes who are wearing glasses that over-correct. In order not to fall into the same mistake in determining myopia, and thus make the latter greater than it really is, we must regard as the correcting glass the *weakest* concave glass with which the myope sees distinctly at a distance. Hence, in determining myopia we proceed as follows: *We place the myope at a distance of* 6 (*or* 5) *metres from the test card, and keep putting concave glasses before his eyes, beginning with the weaker ones and gradually advancing to*

[1] Since we may consider that part of its refractive power has been taken away from the myopic eye by a concave lens, in order to make it equivalent to an emmetropic eye, we say that the myopic eye has the *higher* refractivity of the two; an eye with a myopia of 2 D is so to speak $E + 2$ D. Similarly the hypermetropic eye is known as an eye with *lower* refraction; a hypermetropia of 2 D is, so to speak, $E - 2$ D (Straub). [This conception of the myopic eye as practically equivalent to an emmetropic eye with a convex lens before it, is useful in explaining the symptoms of myopia. Thus to make an emmetrope realize how a myope of 5 D sees, we have simply to put before his eye a + 5 D lens. He will then have the blurred vision for distance, the sharp and enlarged vision for near, and the close near point of one having a myopia of this amount. And like such a myope he will be reduced to an emmetrope again by adding a − 5 D lens to the lens he already has. A myope of 5 D, an emmetrope with a + 5 D before his eye, and an emmetrope using 5 D of his accommodation have for the time being the same sort of vision. So, too, to demonstrate how a hypermetrope of 2 D sees we have simply to put over the emmetrope's eye a − 2 D lens. Such a one will have blurred vision, speedily rendered clear by an often painful and strained effort of accommodation, and a recession of his near point, just as the hypermetrope has.—D.]

those that are stronger, until the best vision is obtained which can possibly be secured in this special case. The WEAKEST *concave glass with which this vision is obtained gives the degree of myopia.*[2]

This method of determining myopia, which was instituted by Donders, is the one generally employed. It is pretty tedious, since we are obliged to go gradually from weaker to stronger glasses, and frequently, therefore, have to place quite a large number of glasses before the eye before we get to the one that corrects. Hence, some have conceived the idea of determining not only myopia, but also the refraction generally, in a more expeditious way, namely, by the use of various apparatus which are called *optometers*. These are constructed upon various principles. Most consist of a test object at which the eye looks through a single lens or through two lenses combined. The rays which enter the eye can be given a parallel, convergent, or divergent direction, and thus adjusted to suit the different refractive errors that may exist, either by altering the place of the test object or by shifting the lenses. The refraction is then simply read off from a scale which is attached to the instrument. In spite of the advantage accruing from the almost instantaneous determination of the refraction which these instruments afford, they have not become popular, because the refraction as found by them is regularly too high; for the person who is looking into the instrument exerts, without being aware of it, some effort of accommodation. Hence, if we desire to find the true refraction—i. e., the refractive state of the eye when the accommodation is relaxed—we are obliged first to paralyze the accommodation with atropine, a procedure which causes the patient considerable discomfort and annoyance. [See page 901.]

767. The determination of myopia by glasses or optometers is called the *subjective* method, because it is dependent upon the statements of the patient. For this reason its results are not always exact. It often happens that, by the patient's straining his accommodation, a higher degree of myopia is simulated than really exists. Moreover, we are dependent upon the patient's good will and intelligence. Frequently we are dealing with malingerers, who purposely try to make their myopia appear too high, in order to escape military service. Again, in small children this method is not applicable. For such cases the *objective* method, which consists in the determination of the refraction by means of the ophthalmoscope [or shadow test], and which is therefore independent of the statements of the patient (pages 103-111) is suitable. And even when the degree of myopia has been already determined by the subjective test, the result should in every instance be verified by the objective test of the refraction.

768. Varieties of Myopia.—The fact of parallel rays coming to a focus in front of the retina, which constitutes the essence of myopia, may in general arise in two ways:

1. The *refractive power of the eye is abnormally great*, so that parallel rays are made too convergent, the retina in this case being in its normal situation (*refractive myopia*). The cause of the increased refractive power may lie in the cornea or in the lens. In the *cornea* it is increased curvature

[2] [This is the rule, provided the accommodation is still active. If the accommodation is completely paralyzed by atropine or homatropine, the rule is the reverse—i. e., the *strongest* concave glass with which the best vision is attained gives the proper correcting glass for the patient to wear when his accommodation regains its power. See page 902—D.]

that leads to myopia. This is found most markedly in ectases of the cornea of the most diverse sort, and to the greatest degree in keratoconus. In this case the myopia is always associated with a considerable degree of astigmatism.

The *lens* can cause abnormal elevation of the refractivity of the eye either through increased curvature or through augmentation of density. The following cases fall under this head: (*a*) In luxation the lens takes on an increased curvature because the tension exerted by the zonula is removed. If the case is one of luxation into the anterior chamber, the forward displacement of the lens contributes to the increase in refractive power, since with the lens the principal focus of the whole dioptric system is shifted forward. (*b*) Accommodation, which increases the curvature of the lens, may be kept permanently in action, and as long as this spasm of accommodation continues myopia will be present. The latter disappears when the accommodation is paralyzed by atropine. (*c*) Myopia due to increase in density of the lens [so-called *index myopia*] not infrequently sets in at the beginning of senile cataract (see pages 528, 538) and also in diabetes. [For the myopia of iritis, see page 406.]

2. The refractive power of the eye may be normal, so that parallel rays come to a focus at the customary spot, but the retina may lie too far back. The cause of this is an elongation of the axis of the eye, for which reason this sort of myopia is called *axial myopia*. The distention of the sclera, to which the elongation of the eye is due, usually affects the posterior division of the sclera, which is bulged out posteriorly, forming the staphyloma posticum of Scarpa. This is the ordinary typical form of myopia, which therefore deserves special mention.

Typical Myopia

769. Symptoms.—Myopes see indistinctly *at a distance*, because of the presence of diffusion circles. To make these smaller, and thus see better, they squeeze the lids together, and in this way produce a stenopæic slit. In fact, it is from this habit of blinking that the name myopia[3] takes its origin. [In myopia and other refractive errors the diffusion circles often cause monocular diplopia—test-types and like objects appearing double.—D.]

Myopes see well *near by*, and, moreover, have the advantage that they need use little or no accommodation for this purpose. The range of accommodation has the same relation in the myopic eye (if the high degrees of myopia are excepted) as in the emmetropic eye. Only, since the far point lies at a finite distance, the whole region of accommodation is placed closer to the eye, as can be seen from Fig. 372 (No. 3), in which the region of accommodation lies between 10 and 5 cm. in front of the eye. In working at close range, therefore, the myope needs to use less accommodation than

[3] From μύειν, to shut, to blink, and ὤψ, sight.

the emmetrope, or even no accommodation at all. Suppose, for instance, that work has to be done at a distance of 33 cm. In this case the emmetrope requires an accommodation of 3 D (100 ÷ 33 = 3). A myope whose myopia equals 1 D needs only 2 D of accommodation, and one having a myopia of 3 D needs none at all, since his far point lies at the working distance. As soon, therefore, as myopia has reached a certain degree, the accommodation ceases to be used (it being presupposed that no glasses are worn). Hence, in myopia of high degree the range of accommodation is, as a rule, not normal, but diminished.

For the same reason, in myopes *presbyopia* sets in later than in emmetropes, or does not set in at all. To be sure, the diminution in elasticity of the lens is produced in a myopic eye just the same as in any other, but practically it does not make itself so perceptible. If a man has a myopia of 3 D, his far point lies at 33 cm., and will always remain there though he be ever so old. During his whole life, therefore, there will be distinct vision at this distance, the only difference from what existed previously being that the patient, when at an advanced age he has lost his power of accommodation, will no longer be able to see closer than 33 cm.—a thing, however, which there is usually no necessity of his doing in any case. Such a myope, therefore, does not become presbyopic at all. Those having myopia of less degree do become presbyopic, but do so later than emmetropes. The point of time when presbyopia sets in—i. e., when the near point recedes beyond 33 cm.—can easily be calculated for each individual case if we know the degree of myopia and the amount of the range of accommodation at different ages.

770. The *troubles* that myopes complain of vary according to the degree of myopia. In the lower grades of myopia distant vision is indistinct, and yet often suffices for ordinary purposes, so that many myopes of this sort do not use glasses. For near work moderately near-sighted eyes are generally regarded as serviceable, because they do their work with less accommodation, and, moreover, either become presbyopic late or do not become so at all.

It is otherwise with the high degrees of myopia. In this case not only is the complaint made of indistinct vision at a distance, but also of inability to keep on with work near by for any length of time; for, owing to the short distance from the eyes at which the far point lies, a considerable effort of convergence is required—an effort which, moreover, is often rendered difficult because the impulse to converge is diminished, owing to the abolition of the accommodation. Hence, a latent divergence and, as a result of it, troubles symptomatic of muscular asthenopia develop. By a transformation of this latent divergence into a manifest one, a strabismus divergens may be set up, a condition, therefore, which is most frequently met with as a result of high myopia.

In myopia of high degree it is often the case that satisfactory distant vision is not attained even by glasses, because morbid changes exist in the fundus. For the same reason, vision close by is frequently defective in spite of the great approximation of the object. Moreover, complaint is made of rapid tiring of the eyes, of great sensitiveness to light, and of muscæ volitantes. The last-named phenomenon is also, to be sure, found in healthy eyes (see page 561); but the myopic eye is more apt to see muscæ volitantes, and sees them in greater number. This arises from the fact that myopic eyes without glasses see everything indistinctly; and upon a hazy background, such as is caused by this indistinct sight, opacities are better projected. Moreover, in the higher degrees of myopia pathological opacities of the vitreous are apt to be present. Muscæ volitantes are not infrequently a source of constant annoyance and worry to myopic patients.

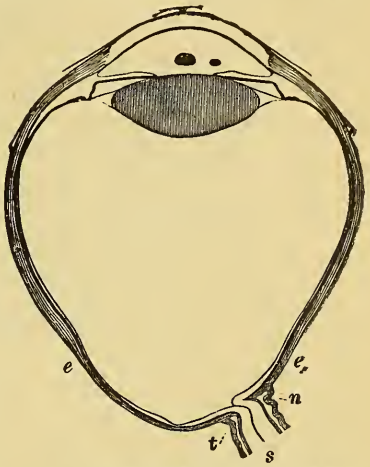


Fig. 380.—Cross Section of a Myopic Eye having an Axial Length of 28 Mm. Magnified 2 × 1.

The ectasia (staphyloma posticum) which occupies the posterior segment of the eye extends from e to e_1. Over the area occupied by the staphyloma the sclera is very much thinned, and at the spot where it is continuous with the sheath of the optic nerve its layers have separated from each other, so that the intervaginal space is dilated at its extremity, and more so at the nasal side, n, than at the temporal side, t. The optic nerve, s, shows within the foramen scleræ an outward bend. The anterior chamber is very deep; the ciliary body projects but slightly.

771. Objective Signs.—Objective examination of a near-sighted eye shows that it is longer than normal (Arlt). The elongation is produced, as dissection of such eyes shows, by the distention of the sclera at the posterior pole (Fig. 380). In marked myopia the enlargement of the eye-ball is recognizable even in the living eye. The eye projects far forward (pop-eye); and when it is turned well in toward the nose, so that the equatorial region appears in the outer portion of the palpebral fissure, this does not, as in the case of the normal eye, make a sharp curve in turning backward, but runs back without much curving—almost straight, in fact. Very myopic eyes are also usually characterized by a deep anterior chamber and a dilated pupil.

The most important changes in myopia are those which are situated in the fundus and can be recognized by ophthalmoscopic examination. These, as a general thing, are the more extensive the higher the degree of the myopia. They affect above all the chorioid and retina, which become atrophic, both in the vicinity of the papilla and in the region of the macula lutea (see page 459). The latter spot, moreover, is the favorite seat of retinal hæmorrhages. Numerous floating opacities are formed in the vitreous, the latter at the same time becoming liquefied. The consequences of

the degeneration of the vitreous make themselves apparent both in the lens and in the retina. In the former there develop opacities, and, as a result of the atrophy of the zonula of Zinn, tremulousness and even luxation may occur; in the retina, detachment may ensue.

The anatomical demonstration of the *enlargement of the myopic eyeball* was first made by Arlt, and thus the nature of myopia was established (1854). Scarpa, to be sure, had already at an earlier date (1807) observed the ectasia of the posterior pole of the eye peculiar to myopia, but did not recognize it as the cause of the latter. The size of this ectasia is in direct ratio to the degree of the myopia. In near-sight of moderate amount the ectasia is limited simply to the posterior pole of the eye; but in the higher degrees of myopia the ectasia ($e\ e_1$, Fig. 380) extends until the optic nerve is implicated, and gets to lie upon the side of the protrusion. The elongation of the eyeball due to the ectasia may be very considerable; there are eyeballs which have an axial length of upward of 35 mm., while the normal eye is only 24 mm. long.

The most striking feature presented by microscopical examination of the ectatic *posterior segment* of the eyeball is the displacement of the sclera with reference to the optic nerve. It looks as though the trunk of the nerve had been pulled away from the foramen scleræ and toward the nasal side; and, as the head of the optic nerve is fixed in the foramen scleræ, the effect of this traction is that the nerve itself is made to bend at its extremity (Figs. 380 and 381). The external sheath is thus drawn away from the trunk of the nerve, particularly at the nasal side. This same displacement is recognizable in the chorioid, from the fact that the latter is drawn away from the border of the optic disk on the temporal side, while at the nasal border the chorioid is drawn up over the optic disk (Fig. 381). The sclera, wherever it happens to lie within the ectasia, becomes attenuated, so that often it is only as thin as paper. The superjacent chorioid and retina present in the main the appearances of atrophy together with slight inflammatory changes; in the later stages both membranes are reduced to a thin pellicle almost destitute of pigment. The vitreous in its posterior division is often liquefied.

The *anterior segment* of a very myopic eye, as far back as the ciliary muscle, is normal (Iwanoff). This muscle has a smaller transverse diameter than in the emmetropic eye, since the circular fibers are less developed, and are sometimes, indeed, almost entirely wanting (Fig. 383). These circular fibers, in fact, are mainly the ones whose function it is to provide for accommodation; and as accommodation is but little employed in a myopic eye, they are not properly developed there. But as the ciliary processes, too, in the myopic eye are not as large as usual, the whole ciliary body appears abnormally flat. [From the researches of several investigators it appears that the non-development of the ciliary body in myopes is probably congenital. Hence A. Wood argues that this non-development is not due to lack of use, and is, in fact, the cause, not the result of the myopia. See page 874.—D.] In hypermetropic eyes the opposite condition exists. In these, Müller's portion of the ciliary muscle is hypertrophied by constant accommodative effort, and thus the whole muscle is increased in size; and hence the entire ciliary body projects farther toward the interior of the eye (Fig. 384). A comparison of Figs. 383 and 384 with each other and with Fig. 382, which represents the ciliary body of an emmetropic eye, shows how the shape of the sinus of the anterior chamber is determined by th form of the ciliary body. In a myopic eye the sinus is deeper, in a hypermetropic eye shallower, than in the emmetropic eye. This relation, which can also be observed macroscopically in the living eye, is held to be of importance in the genesis of glaucoma. We know that in the atter condition, owing both to the swelling of the ciliary processes and also to the thickening of the iris that

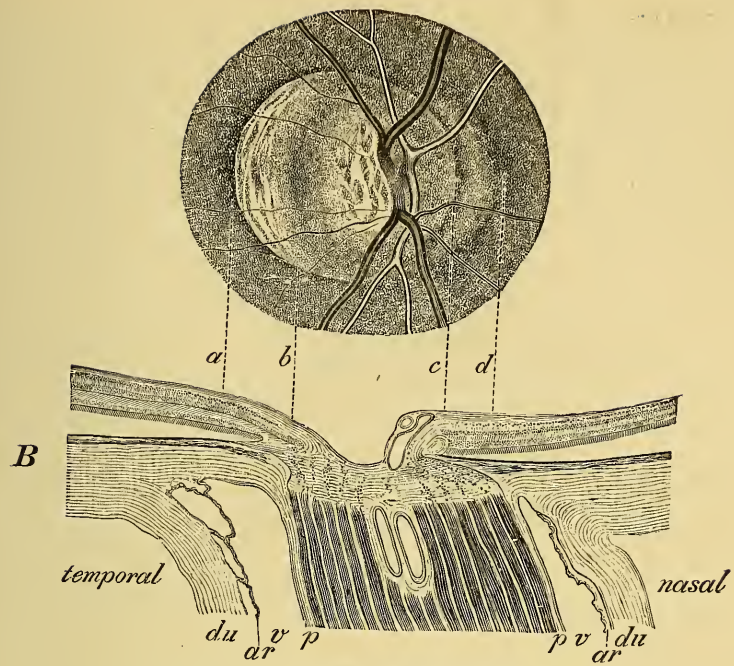


FIG. 381.—OPTIC-NERVE ENTRANCE IN MYOPIA.

A. OPHTHALMOSCOPIC IMAGE OF THE PAPILLA.—The papilla, *b–c*, is of the shape of an ellipse with its long axis vertical. In its outer half it shows the large physiological excavation, upon whose floor are visible, the gray stipplings of the lamina cribrosa, while the central vessels ascend on the inner wall of the excavation. Adjoining the inner border of the papilla and not sharply separated from it, is the bright crescent, *a–b* (distraction crescent). This is of a white color, while the papilla itself is reddish. The crescent is covered with brownish, elongated markings, representing remains of the stroma pigment of the chorioid. The temporal border of the crescent is sharply defined, and the chorioid adjoining it is somewhat more pigmented than usual. On the other hand, the chorioid in the vicinity of the nasal border of the papilla shows a somewhat lighter coloration in the space between *c* and *d*, so that a yellowish crescent, which, to be sure, is not much more than a suggestion of one, is formed on the nasal side of the disk (supertraction crescent).

B. LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE. Magnified 14 × 1. Here the displacement of the optic nerve with relation to the aperture in the sclera and chorioid designed for its passage, is obvious. The optic-nerve funiculi, wherever they consist of medullated fibers, are colored black by Weigert's hæmatoxylin stain; between them can be seen the septa, which remain unstained, and the longitudinal sections of the central artery and central vein. The black staining ceases abruptly at the lamina cribrosa. In front of the lamina cribrosa the head of the optic nerve presents the physiological excavation. This is a depression whose floor at its deepest part is formed by the lamina cribrosa. The temporal wall of the excavation slopes down quite gradually from the retina. The nasal wall declines abruptly, and shows the cross section of the central vessels. The trunk of the optic nerve is inserted obliquely into the eyeball, a fact that is particularly evident when comparison is made with Fig. 264. This displacement is most pronounced where the nerve traverses the sclera and chorioid. The temporal wall of the sclerotico-chorioidal canal therefore, is turned somewhat forward, and hence, since the overlying retina is transparent, comes into view when looked at from in front (with the ophthalmoscope), forming a bright crescent extending from *b* to the point *a*, where the pigment epithelium begins. The stroma pigment of the chorioid extends somewhat further inward than does the pigment epithelium, and is consequently seen under the form of brown spots upon the bright crescent. The nasal wall of the scleral canal is turned partly backward, so that it has to pass in front of the most nasally situated portion of the optic nerve, *c–d*. As the displacement affects not only the aperture in the sclera but also that in the chorioid, the chorioid is also drawn up over the nasal border of the optic nerve as far as the point *c*. Since now this nasal portion of the papilla, being covered by the sclera and chorioid, is not distinctly visible with the ophthalmoscope, the papilla appears contracted in its horizontal diameter. Nevertheless, the portion of the optic nerve that is thus concealed glimmers through its covering, so as to be distinguishable under the form of an ill-defined yellowish crescent at the nasal border of the papilla (A, *c–d*). The displacement of the optic nerve with reference to the sclera is shared in by the sheaths of the nerve. The dural sheath, *du*, and the adjoining arachnoid sheath, *ar*, are separated from the nerve, especially at its temporal side, and the intervaginal space, *v v*, is consequently dilated. On the other hand, the pial sheath, *p* lies in close apposition to the nerve.

occurs when the pupil is dilated, the iris is pushed against the cornea, and the sinus of the anterior chamber is thus shut off (see page 506). Obviously the more prominent the ciliary process and the narrower the sinus of the anterior chamber the more readily will this result take place. In this fact is probably contained, at least in part, the reason why hypermetropic eyes are very frequently, and myopic eyes, on the contrary, are very rarely indeed, attacked by inflammatory glaucoma. [But see page 496.]

The ophthalmoscopic and anatomical changes which make their appearance in the higher grades of myopia render it evident why the *visual acuity* is almost never found to be normal in very marked near-sight.

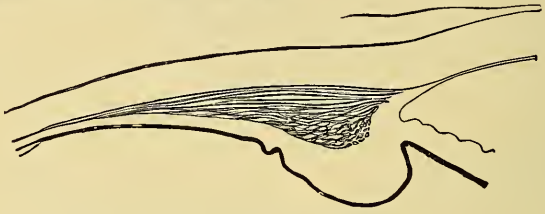


Fig. 382.—Ciliary Body of an Emmetropic Eye.

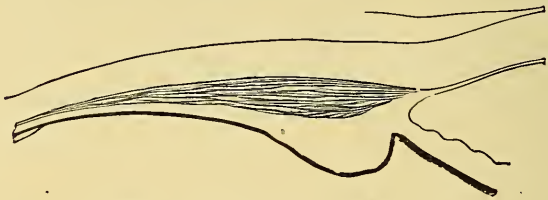


Fig. 383.—Ciliary Body of a Myopic Eye.

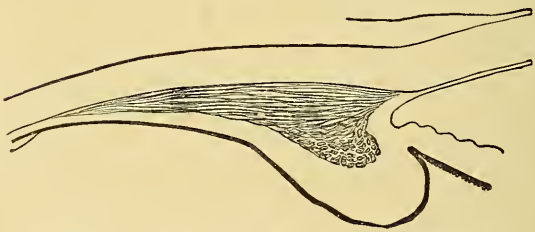


Fig. 384.—Ciliary Body of a Hypermetropic Eye.

772. Course.—Most cases of myopia are those of low degree, which develop during youth and come to a stop after the completion of the body growth (*stationary myopia*). In other cases, however, the myopia attains a considerable height even in youth, and then does not remain stationary, but increases steadily during the whole life, so that finally it reaches the greatest possible degree (*progressive myopia*). It is mainly these cases that lead to destructive changes in the interior of the eye, and that cause myopia to appear in the light of a disease of the eye, and of a serious disease, too, which in advanced age often gives rise to amblyopia or even blindness.

Among the laity there is a belief that near-sight diminishes in age; but this is true only for the lowest degrees of myopia. In advanced age the emmetropic eye becomes hypermetropic (see page 883); consequently, in a myopic eye the myopia must diminish.

[This diminution, however, is usually inconsiderable—not over a dioptry at most.—D.] Myopes, however, often believe that they are becoming less near-sighted because they see better at a distance without glasses than they used to do, and yet testing with glasses shows no diminution in the myopia. This improvement in vision depends upon the fact that in old age the pupils become more contracted, and hence, in looking with the naked eye, the diffusion circles are smaller. But all persons who have a great degree of near-sight see worse and worse as their age increases, because not only is the myopia augmented, but the complications of myopia tend to develop more and more. [In myopia of 10 to 12 D the vision may still be $\frac{20}{20}$, especially in children. Usually, however, it is not better than $\frac{20}{30}$ and may be only $\frac{20}{40}$. In myopia of over 12 D vision is rarely better than $\frac{20}{40}$ even when there are no complications, and when the myopia exceeds 15 D, vision is generally $\frac{20}{70}$ or $\frac{20}{50}$ at best.—D.]

773. Causes of Myopia.—Myopia is only exceptionally congenital, elongation of the eye in that case existing at the time of birth. The rule is that myopia develops in youth at the time when, as the whole body is growing rapidly, considerable demands are coincidently made upon the eyes by school life or by work. It has been established by many observations that acquired near-sightedness is found almost exclusively in those persons who are compelled to strain their eyes with near work. Such are, on the one hand, the members of the cultured classes who apply themselves to study; and, on the other hand, working people, like tailors, seamstresses, compositors, lithographers, etc., who have fine work to do. There is thus no doubt but that the cause of myopia is furnished by near work. Here two factors come into play, namely, the accommodation and the convergence, by the combined action of which the distention of the posterior pole of the eye is effected. [That excessive use of the eyes in near work is the main cause of myopia seems doubtful; and it appears quite unlikely that over-use of the accommodation and convergence is responsible for it. For a contrary view, see page 874.—D.] But, although straining of the eyes in near work is the cause of myopia, not all, but only a fraction of those who are subjected to this strain actually become near-sighted. In this fraction, therefore, special additional factors must be present which favor the development of the myopia due to near work. The following are the factors of this sort that we know of: 1. A *predisposition* to myopia, which doubtless has its seat in definite anatomical conditions, such as too slight resistance of the sclera, peculiarities in the relations of the ocular muscles or of the optic nerve, etc. Since anatomical peculiarities are apt to be inherited, the hereditary character of myopia is also readily explainable. The children of near-sighted parents are not, to be sure, born near-sighted; but if they are exposed to those conditions which favor the development of near-sightedness, they show a greater tendency to become myopic than do the children whose parents have normal sight. 2. Those circumstances which compel *too great approximation of the work*, and thus require an abnormally great accommodation and convergence. This is the case when particularly fine work has to be done, or when work is carried on with insufficient illumination, and also when the visual acuity is diminished (by

maculæ of the cornea, opacities of the lens, astigmatism,[4] etc.), so that it becomes necessary to bring the objects closer than usual (see pages 320, 536).

Near-sightedness is so widespread and important a disease that it has received an amount of investigation which, for extent and for thoroughness along all lines of research, few other diseases can equal. The main thing that these numerous investigations have proved is that *near-sight is an attribute of culture.* In the country, for example, we encounter fewer people with glasses than we do in the city. In the latter, again, it is the schools which are the main hotbeds for the propagation of near-sightedness. Cohn by his extensive researches was the first to direct general attention to this fact. Since then, statistical researches in regard to myopia have been published in almost all countries—researches which extend to all classes of every condition and every age, even including newborn children. It has been proved that among newborn children myopia practically never occurs; in fact, they are almost without exception hypermetropic. Near-sightedness is acquired later in life through straining the eyes, and hence fails to occur when this strain is absent. In savage races near-sightedness no more occurs than it does among children. Again, in the lowest order of schools—the primary schools—there are extremely few near-sighted persons, and the same is true of the rural population, whose education does not, as a rule, get much beyond the primary school. The school most dangerous for the eyes is the intermediate school. It is in this that myopia first develops and then increases, both as regards its intensity and as regards the number of myopes, in proportion as we ascend the classes. In Germany about 20 per cent are myopic in the lowest classes of the high schools and 60 per cent in the highest classes. In going into the higher classes the scholar who is already near-sighted becomes more and more so; and, furthermore, new scholars are constantly being attacked with myopia. In the university the condition of affairs is still more unfavorable. Near-sight acquired as a result of study thus rightly bears the name of "school myopia." [So, too, among 4800 school children in this country, examined under a cycloplegic, only 9 per cent were found to be myopic at the age of 5 and 48 per cent at the age of 16 (Tenner). While, however, these statistics convincingly prove that in each year of the growing period more and more cases of myopia develop, they by no means prove that near work is the cause of the myopia. This latter view, in fact, is contested by many, who argue that there is some congenital anatomical basis for the myopia, which would be effective in causing its progressive increase whether the eyes were used for near work or not. They point out that myopia, even of high degree, is found among those who never use their eyes for near work at all, e. g., among the Bedouins, Egyptians (very frequent), and other illiterates.[5] A. Wood believes that the small size of the ciliary muscle, which is so frequently found in myopia, is not, as stated on page 870, the result, but actually the cause of the near-sightedness. Accepting Thomson's theory (page 15) that the ciliary muscle helps to effect outflow from the eye, he believes that congenital deficiency of this muscle by retarding outflow increases the intra-ocular pressure and hence causes the eye, which is distensible during early life, to expand. When the myopia was once established, the non-action of the ciliary muscle, due to the failure of myopes to use their accommodation, would tend to make it increase, and a contributing factor would be the dilatation of the pupil, which is usually present in myopes and which would also help to impede outflow from the eye. There are a number of clinical observations which seem to favor this theory—one being that, as pointed out on page 876, correction of myopia by glasses, which call the accommodation into play, seems to retard the development of the near-sight.—D.]

[4] [The effect of astigmatism is particularly important. Irregular astigmatism and, according to some authorities inverse and oblique astigmatism are of especial significance in causing myopia.—D.]

[5] [It must be admitted, however, that here the great frequency of corneal opacities among these people may have a good deal to do with causing their myopia.—D.]

Continuous employment of the eyes upon fine work exerts the same influence as do schools. Among lithographers Cohn found 45 per cent and among compositors 51 per cent to be myopic.

While many think that near work produces near-sight, they have not been able to agree as to how it does it. As regards this point different theories have been propounded, each one of which probably contains one or more points that are correct although none is perfectly satisfactory. Those who accuse the *accommodation* of being the cause of myopia assert that during accommodation the intra-ocular pressure is somewhat elevated.[6] If this process is frequently repeated, it may lead to distention of the posterior portion of the sclera where it is most yielding. According to Von Graefe, *inflammatory processes* in the chorioid and sclera (sclerotico-chorioiditis posterior), by which the sclera is rendered softer, are also to be considered in this connection. Others believe that it is not so much the accommodation as the *convergence* that should be made accountable for the development of myopia, inasmuch as in convergence a pressure is exerted upon the eyeball by the external ocular muscles, which leads to its distention. It has been supposed that either the internal and external recti which in the act of convergence are stretched more tightly upon the eyeball, or the two obliques which surround the eyeball, like a sort of noose, may produce this effect (Stilling). The muscles last named are, furthermore, so situated that they press upon the points of exit of some of the venæ vorticosæ from the eyeball, and may thus produce venous congestion in the latter. Convergence might, however, produce distention of the posterior pole of the eye in another way—namely, by the fact that the latter is displaced outward, and consequently is pulled upon by the optic nerve. This action would be particularly looked for when the optic nerve is, relatively speaking, too short (Hasner, Weiss).

[A number of clinical facts seem to show that we cannot consider either excessive accommodation or excessive convergence as the determining cause of myopia. Rather the reverse may be true.—D.] Levinsohn regards the cause of the elongation of the eye to be the traction which *gravity* exerts on the eye, when in close work the head is kept bent forward so that the cornea is directed more or less down. Lastly, some have thought that *diminished rigidity* of the coats of the eye is responsible for myopia, it being assumed that there is a congenital defect in development causing this (Schnabel).

Males and females are equally predisposed to myopia.

774. Treatment.—It is impossible to do away with the elongation of the eyes, that forms the basis of myopia. We must confine ourselves to making provision by means of suitable glasses for distinct vision, and to procedures that will enable close work to be carried on as far as may be without tiring. Moreover, the progress of the near-sight must be checked as far as possible, and any complications present be attacked.

The following principles hold good with reference to the *wearing of glasses* by myopes: In the low degrees of near-sight (up to about 2 D) it is sufficient to order glasses for distance, provided that any desire for them is expressed. For near work, glasses are not necessary, since vision can be carried on without them at a sufficient distance—that is, up to or beyond 50 cm. Hence we may leave it to the patient himself whether he shall use his glasses for his near work or not. But in case troubles referable to a latent divergence [exophoria] are present, the glass should be worn for near also, since by means of it the latent divergence is relieved (page 791). In the medium degrees of near-sight—i. e., from 2 to about 7 D—glasses

[6] [This is almost certainly erroneous (Hess).—D.]

are necessary for distance, and they are often desirable for near work as well; for otherwise the work would have to be held too close, and would thus require great convergence which might give rise both to exhaustion of the eyes and to progressive increase of the near-sight. If the eye is otherwise healthy, and the range of accommodation is large enough, a glass is prescribed which can be worn constantly for both distant and near vision. [Statistics seem to prove unequivocally that the full and early correction of myopia tends to check the progress of the process. It is therefore becoming a more and more prevalent practice here and also in Europe to correct myopia fully, even when of slight degree, and, as far as possible, to get the patient to use the same glass for distance and near. In this way the eyes are placed under more normal conditions both for distant and near vision and also as regards their accommodative function. The result of this practice in individual cases has proved eminently satisfactory. See also page 902.—D.] If the range of accommodation is small, either on account of advanced age or from other reasons, the glass which corrects the myopia will not be borne for near work. Near-sighted persons who have worn the same glass constantly for many years find that, as they become older, reading with this glass becomes more and more difficult. In such cases two sets of glasses must be ordered: a stronger one, which corrects the myopia, for far points; and a weak one, for near work, which removes the working distance to the point desired. The same rule holds good for the high degrees of myopia, in which, likewise, different glasses must be prescribed for far and near. When, owing to complications, the vision is greatly diminished, glasses are in any case of little or no use.

Prescribing glasses for near-sighted persons requires much experience and a careful consideration of all the attendant conditions. In no case should the choice of glasses be left to the optician.

775. In addition to the selection of glasses, the *regulation of the patient's habits*, both as regards his general condition and his eyes, must be attended to. This is the more important the greater the myopia is, and the more reason there is to apprehend its progressive advancement and the development of complications. And, first of all, near work must be restricted as much as possible. Such work as has to be done under any circumstances should be performed at the greatest possible distance from the eyes. To effect this we must see to it that the print of the books is good, that the illumination is sufficient, that the correct position is maintained in reading and writing, etc. Work in the evening by artificial light must be restricted as far as possible. It is very advantageous to interrupt the work at frequent intervals and rest the eyes by looking at a distance. If it is apparent that the near-sight is making rapid progress and threatens to reach a still higher degree, it is advisable to drop all studies for quite a long time. Young people with markedly progressive myopia should be warned to take the condition of their eyes into account in choosing a profession. An occu-

pation, like that of civil-service clerk or one of the learned professions, that requires constant reading and writing, is not suitable for people of this sort.

The great prevalence of near-sight, particularly among the young who are engaged in studies, has justly excited widespread anxiety and led to endeavors to *put a stop to the extension* of the evil. First of all, the excess of work which many scholars have at present to struggle with should be reduced to the proper standard. The way in which scholars are overtasked, both in school and at home, is admitted by most, and is prejudicial not only to the eyes, but also to the child's whole mental and physical development. Instruction ought not to be begun too early (if possible, not before the completion of the sixth year), and more time should be allotted to bodily exercise, especially in the open air, than has hitherto been the case. The hours set apart for this latter purpose should alternate suitably with the hours devoted to sedentary occupations, so as to serve as a rest from both mind work and eye work. That amount of work which absolutely has to be done should be done under the most favorable conditions. To accomplish this, special attention must be paid to the schools, since the work done at home is beyond our control. The requisites, which in many modern schools are already carried out, are: 1. Good illumination—i. e., illumination of sufficient strength and falling upon the work in the proper direction; the light should come mainly from the scholar's left side. [The illumination should be diffuse—never very intense in any one spot,—should present no glaring points of light, and should reach all parts of the room, so as to offer no violent contrasts of light and shade anywhere. By observing these precautions we lessen the tendency to eye strain as well as to the development of myopia.—D.] 2. Well-constructed seats and tables, which, furthermore, should be adapted to the varying size of the scholars, so that they may not be forced to adopt a bad attitude of the body. If, however, the scholars do bend forward too much, especially in writing, the use of some straightening appliance is indicated. 3. A proper method of instruction in writing which will enable the pupil to keep the head and body straight while writing (upright script). 4. Good print. Books having too fine print, and also too fine fancywork for girls, should be banished from schools.

[A very important means of preventing school myopia and checking its advance is the systematic examination of children in the primary schools to determine and rectify refractive errors, especially astigmatism and myopia, and bring the sight as nearly as can be to the normal. As Fuchs says, those children in whom the sight because of opacities in the media of fundus disease can not be brought to the normal should not be allowed to take the full course prescribed for others, and should be trained for some out-of-door avocation. In a beginning near-sightedness, the hygienic rules laid down by Fuchs for myopes in general—restriction of the hours of study, restriction or absolute prohibition of study by artificial light, frequent intervals of rest for the eyes during the day, and insistence on out-of-door exercise—should be followed out with particular stringency. The younger the child, the more important these regulations are. Children with rather high myopia should be segregated so that they can receive instruction specially adapted to their needs and in classes by themselves (Harman).—D.]

In the highest degrees of near-sightedness, we may *remove the lens* by discission, even if it is still transparent Fukala). The operation is a suitable one for young persons, whose myopia amounts to more than 15 D, and who do not show excessive pathological changes in the fundus. The eye may thus be brought nearly to the point of emmetropia, so that it is able to see distinctly at a distance without any glass. We must not, however, overlook the fact that by this operation we sacrifice the accommodation, and that the operation does not act to check the increasing elongation of the eyeball and the consequent changes in the fundus.

CHAPTER IV

HYPERMETROPIA

776. Definition.—Far-sight, hypermetropia[1] (*H*), is that refractive condition of the eye in which parallel rays falling upon the eye come to a focus *behind* the retina (at *f*, Fig. 385). Properly speaking, the rays do not come to a focus at all, since the conical beam of rays has its apex truncated by the retina, and a diffusion circle is formed there. Hypermetropia is the opposite of myopia; in the former the apex of the cone of rays lies behind the retina, in the latter in front of it.

What sort of rays, then, can come to a focus, so as to form a distinct image upon the retina? When we try the experiment of bringing an object from infinite distance closer to the eye, we find that it becomes more and more indistinct; for, the more divergent the rays are when they reach the eye, the farther behind the retina will they be when they come to a focus (thus in Fig. 385 the rays coming from *O* come to a focus at f_1), and hence the larger will be the diffusion circles. Accordingly, the hypermetropic eye cannot, without the exercise of accommodation, see either distant or near objects distinctly. For rays to come to a focus upon the retina of a hypermetropic eye, they must have a certain degree of convergence as they fall upon the eye (*c c*, Fig. 386). How great must this convergence be? To ascertain this we must prolong the rays until they meet. This would occur at a point (*R*, Fig. 386) situated behind the retina. The distance of this point from the eye gives us the measure for the degree of convergence that the rays must have for them to meet upon the retina. This point is, accordingly, the far point, *R*—i. e., the point for which the hypermetropic eye is adjusted when in a state of accommodative repose. It is at a finite distance, just as is the far point in myopia, but is behind the eye and not in front of it, as in myopia. The difference between the two consists in this, that in myopia the rays that are brought to a focus by the eye emanate from the far point, while in hypermetropia they converge to the far point. The far point in hypermetropia, therefore, is not a point from which the rays actually start or at which they actually unite, but is an imaginary point which is simply adopted to indicate the direction of the rays. We say, therefore, that the hypermetropic eye has only a virtual far point, and we designate it by the negative sign:—*R*.[2]

Formerly presbyopia and hypermetropia were confounded with each other. People saw a hypermetropic boy, whose eyes soon grew tired in studying, finally take up his grandfather's glasses and then read well with them and without becoming tired. This boy, they reasoned, must have the same sort of weakness in his eyes that his grand-

[1] [From ὑπερ, in excess of, μετρον, measure, and ὤψ sight.] Also called hyperopia [from ὑπερ and ὤψ].

[2] The points *f* and f_1, in Fig. 385, which likewise lie behind the eye, have nothing to do with the far point, but are simply the foci for parallel or divergent rays incident upon the hypermetropic eye.

father has, only that with him it has already set in in youth, and therefore is very serious. This "hebetudo visus" was attributed to a weakness of the retina, and it was believed that it might possibly go on to blindness. The only thing which might have ameliorated the sufferings of the hypermetrope—namely, the use of glasses—was held to be particularly dangerous.

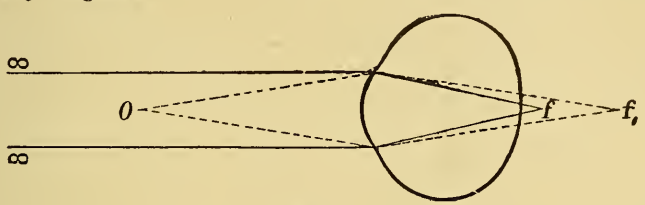


Fig. 385.

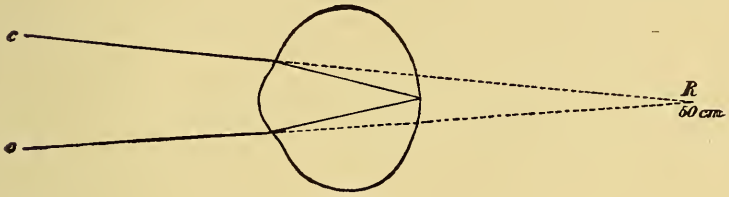


Fig. 386.

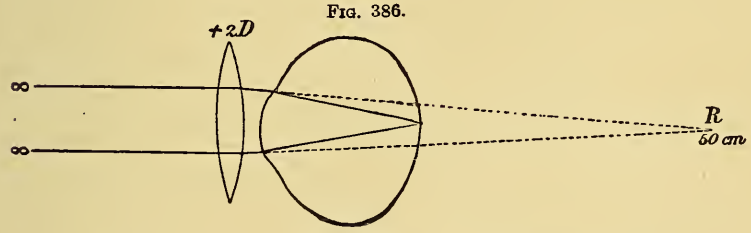


Fig. 387.

Figs. 385 to 387.—Path of the Rays in a Hypermetropic Eye.

Donders deserves the great credit of having discovered the true nature of these conditions. The weak sight of the old man is presbyopia and has reference to the *accommodation*. It is, however, not an anomaly of accommodation, but a physiological state. The boy's bad sight depends upon hypermetropia, which has nothing to do with the accommodation, but is an *error of refraction* that exists in the eye even when destitute of accommodation. The similarity of the two conditions lies in their having one symptom in common—namely, impairment of vision for near points—and yet even in this regard there is an essential difference between the two. In presbyopia, distinct vision inside of a certain distance is simply impossible. In hypermetropia, distinct vision at near points is generally still possible (if the range of accommodation is great enough), but is associated with straining and tiring of the eyes.

Donders also has shown that the asthenopia of hypermetropes is not the symptom of a serious lesion of the eye, but is an evidence of fatigue consequent upon unfavorable optical conditions. By the correction of these conditions with simple optical devices, a countless number of men have since this discovery been rendered once more capable of work and have been freed from the dread of growing blind.

777. Determination.—The greater the hypermetropia, the greater must be the degree of convergence with which the rays impinge upon the

eye if they are to unite upon its retina, and the nearer, therefore, to the posterior pole of the eye will be the point where these rays intersect each other, if they are supposed to be prolonged without undergoing refraction. Now this point where they thus intersect is the far point. The degree of hypermetropia is determined, therefore, just like the degree of myopia, by the distance of the far point from the eye; in both cases the error of refraction is the greater the nearer the far point is to the eye. The only difference is that in myopia the far point lies in front of the eye, and in hypermetropia behind it. For this reason the distance of the far point cannot in hypermetropia be measured directly as it can in myopia. We are forced to determine it indirectly by means of the test with glasses. In doing this we start as in correcting myopia with the attempt to refract parallel rays by means of the lens in such a way that they shall come to a focus upon the retina. Obviously a convex lens is required for this purpose, since this alone is able to render parallel rays convergent. If the far point of the hypermetropic eye lies at −50 cm. (*R*, Fig. 387), we should have to take a lens of 50 cm. focal length (=2 D). Parallel rays (∞ ∞, Fig. 387) falling upon the lens will be so refracted by the latter as to converge toward its focus, which lies 50 cm. behind the eye—i. e., at the same spot as the far point. These rays will therefore be brought to a focus upon the retina. In this discussion, for the sake of simplicity, the distance of the glass from the eye has been neglected.

As the same thing can be proved for hypermetropia of any other degree, the following statement may be enunciated as a general law: To see distinctly at infinite distance the hypermetropic eye requires that convex glass whose focal length is equal to the distance of the far point from the eye. Accordingly, the glass that corrects the hypermetropia gives through its focal length both the position of the far point and the degree of the hypermetropia. We express the latter by the number of dioptries which the correcting glass represents; and hence say a hypermetropia of 2 D, not a hypermetropia with far point at 50 cm.

778. Latent and Manifest Hypermetropia.—While it was said above that hypermetropes can see neither distant nor near objects distinctly, this statement holds good only when no accommodation is made; for by means of his accommodation the hypermetrope is able to increase the refractive power of his eye precisely as if a convex glass had been placed in front of it; he can correct his hypermetropia by accommodation. This fact renders the exact determination of hypermetropia difficult. If we examine the same individual at different times for hypermetropia, we find that it is not always of the same degree. For example, it may happen that in a young hypermetrope the hypermetropia today is found to be 1.5 D, a little while after 1 D, and to-morrow perhaps 2 D. Which of these findings is the correct one? If we instill atropine several times, and again make the

examination, we now find the hypermetropia to be constantly the same but considerably higher than before—e. g., 4 D.

The only possible reason for the fact that the hypermetropia as determined by us before atropinization was too low is that a part of this hypermetropia is concealed all the time by the accommodation. The hypermetrope is so accustomed to accommodate that he is unable to relax his accommodation completely even when convex glasses are placed in front of the eye, which render his accommodation superfluous, or even a disadvantage. Thus, with a glass which completely corrects his hypermetropia, the hypermetrope sees at a distance no less badly, and as a general thing much worse than with his naked eye. If we begin by placing very weak convex glasses before his eyes and then gradually use stronger and stronger ones, he will indeed keep on relaxing his accommodation, but only up to a certain point; he always retains a reserve of accommodation which he is unable to give up. With the glass thus found, combined with the residuum of accommodation, he corrects his hypermetropia and sees distinctly. If still stronger glasses are placed before the eye, these combined with his residuum of accommodation produce an over-correction of his hypermetropia, and vision will once more become indistinct. When, then, we determine the glass with which the hypermetrope sees most distinctly, this does not give us the entire hypermetropia, but only that portion of it which has been set free by the relaxation of the accommodation. This is called the *manifest* hypermetropia (*Hm*). The remaining portion which is concealed by the accommodation all the time is the *latent* hypermetropia (*Hl*). Both together constitute the *total* hypermetropia (*Ht*) —i. e., $Ht = Hm + Hl$. In the example adduced above, $Hm = 1$ to 2 D, $Ht = 4$ D, and hence $Hl = 2$ to 3 D.

The ratio of *Hm* to *Ht* depends upon the range of accommodation, and hence chiefly upon the age. In youth, when the range is large, upward of half of the total hypermetropia is latent. The older a man grows the more the manifest hypermetropia increases at the expense of that which is latent, until in old age $Hm = Ht$. Hence, when we test an old man with convex glasses, we find the whole hypermetropia at once; but in persons who still possess the power of accommodation, a determination of the total hypermetropia is possible only after paralyzing the accommodation with atropine.[3]

In practice we generally abstain from determining the total hypermetropia, because atropinization is accompanied by effects which are disagreeable to the patient and which last several days.[4] We ascertain simply the manifest hypermetropia from which, when we know the age of the subject, a conclusion can be drawn as to the total hypermetropia. However, that

[3] [These statements require considerable qualification. I have repeatedly seen a hypermetropia of 2 and even 3 D latent in persons of between forty-five and fifty, and it is just in people over forty that I have had the greatest difficulty in getting the accommodation to relax. On the other hand many a young hypermetrope will relax his accommodation readily so that nearly or quite all his hypermetropia becomes manifest.—D.]

[4] [See, however, page 901.]

we may come as near as possible to the true value of the hypermetropia, we try to get the patient to relax his accommodation all that he can. For this purpose we proceed as follows:

We place before the eye of the patient, who is stationed at a distance of 6 (or 5) metres from the test card, convex glasses, passing very gradually from weaker to stronger ones, until the best possible vision has been obtained. The STRONGEST *convex glass with which this occurs gives the manifest hypermetropia.*[5]

It very often happens that a hypermetrope has perfect vision with the naked eye, because he corrects his entire hypermetropia by means of the accommodation. In this case it is obviously impossible to improve his visual acuity with convex glasses. For a case of this sort the statement made above may be expressed as follows: The degree of manifest hypermetropia is determined by the *strongest convex glass with which the patient is still able to see as well as with the naked eye.*[6] The fact that any one sees as well at a distance with a convex glass as with the naked eye is of itself enough to prove the existence of hypermetropia, since the emmetrope and still more the myope sees worse with even weak convex glasses; for, while concave glasses can be overcome by a suitable effort of accommodation, there is no way of combating the effect of a convex glass, since the crystalline lens can not be made flatter than it is already when in the state of accommodative repose.

[A hypermetrope will often accept a stronger glass when looking *with both eyes together* than when looking with each separately. Hence, when by adding stronger and stronger glasses in the way above described we have determined the acceptance of each eye by itself, i. e., the strongest convex glass with which it will still see well, it is a good plan to arm each eye with the glass accepted and, making the patient look with both eyes, gradually add more convex glasses to one or both until the vision with both begins to blur.—D.]

[When, as in making a *post-cycloplegic test* (see page 902), we already know the total hypermetropia, but wish to determine how much of it the patient will accept, we proceed in a reverse way. That is, we put on the full correction and, making the patient look with both eyes, ascertain, first, whether after keeping the correction on awhile he will not accept it; if not, then what is the minimum reduction that he will take in order to see clearly.—D.]

In the case of hypermetropia, even more than in that of myopia, it is necessary to confirm the results of the subjective method by an objective determination. It is only exceptionally the case that we get at the true amount of hypermetropia by the former method; but with the objective test

[5] [If the accommodation is completely paralyzed, the rule is the reverse of that given—i. e., the *weakest* convex glass with which a patient gets maximum vision is the measure of the hypermetropia. —D.]

[6] [Or, as we say, "the strongest glass that the patient accepts."—D.]

the total hypermetropia is usually found, the accommodation being entirely relaxed during examination with the ophthalmoscope. [Complete relaxation is not so very frequent, and to determine hypermetropia accurately either by the subjective or objective tests we have to use a cycloplegic (see page 901).—D.]

779. Varieties of Hypermetropia.—The condition, characteristic of hypermetropia, in which parallel incident rays are brought to a focus behind the retina, may in general be produced by two different causes:

1. The *refractive power* of the media is lessened so that parallel rays are not rendered sufficiently convergent to come to a focus upon the retina, although the latter is in the same situation as in the normal eye (*refractive hypermetropia*). The *cornea* may be the part accountable for this state of things—e. g., when congenitally it has a low curvature, or when it is flattened by cicatrices. In the latter case there s always a considerable degree of astigmatism as well. The *lens* gives rise to hypermetropia when it loses some of its refractive power. A high degree of hypermetropia is produced when the lens disappears from the pupillary area, either from being luxated or because it has been removed from the eye altogether (aphakia) [see § 876]. In these cases the eye is not only hypermetropic, but also loses its power of accommodation.

A diminution in refractive power also takes place in advanced age, so that the emmetropic eye becomes moderately hypermetropic (1 to 2 D). Similarly the hypermetropic eye gets to be still more hypermetropic, and the myopic eye less myopic—in fact small degrees of near-sightedness may disappear in this way altogether. The cause of this senile hypermetropia is still a matter of dispute. [It is most probably due to changes in the lens (see page 861).—D.]

2. Hypermetropia also develops when the refractive power of the eye is normal, but the retina lies too far forward (*axial hypermetropia*). This may be produced by a protrusion of the retina due to exudations or tumors. But the most ordinary cause of axial hypermetropia is an abnormal shortness of the entire eye, so that typical hypermetropia constitutes the opposite to typical myopia, which is produced by abnormally great length of the eyeball.

Typical Hypermetropia

780. Symptoms.—Hypermetropes would have indistinct vision both for distance and near points if they possessed no accommodation. In hypermetropes, therefore, this latter plays a particularly important part. The hypermetrope, therefore, in fact, must—in contradistinction to an emmetrope or myope—accommodate even *when looking at a distance*, as in order to see distinctly he must correct his hypermetropia by means of his accommodation. Whether this is possible or not depends upon whether the

accommodation when compared with the amount of hypermetropia is powerful enough to bring the near point within infinite distance (Fig. 388 A). If this is not the case, and the near point lies behind the eye, it is impossible for the hypermetrope to see distinctly at a distance without a glass (*absolute hypermetropia*). If the near point lies within infinity, distinct vision for distance can be attained by an appropriate output of the accommodation (*facultative hypermetropia*).

The extent to which hypermetropia can be concealed by accommodation depends not only upon the degree of the hypermetropia, but also upon the strength of the accommodation. But this latter changes with age,

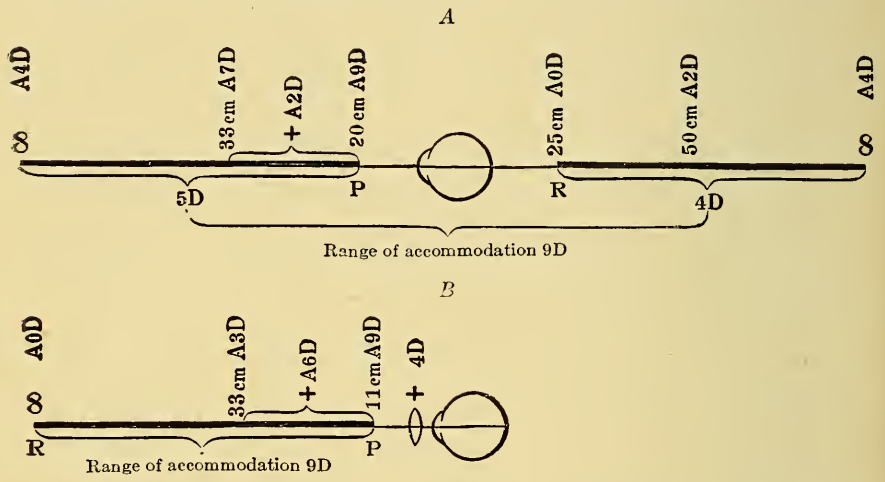


FIG. 388.—REGION OF ACCOMMODATION OF THE EYE OF A MAN 23 YEARS OF AGE WITH A TOTAL HYPERMETROPIA OF 4 D AND A RANGE OF ACCOMMODATION OF 9 D.

A. If the accommodation is completely at rest, the eye is adjusted for its far point, *R*, which lies 25 cm. behind the eye. With an expenditure of accommodation amounting to 2 D the point for which the eye is adjusted shifts to a distance of 50 cm. behind the eye, and if the range of accommodation of this eye amounted to no more than 2 D, the near point of the eye would be situated here (page 853 Note). There would then be an absolute hypermetropia, i. e., the eye would not be able to see distinctly in the distance. It is only by an expenditure of 4 D of accommodation that the eye is adjusted for infinite distance (facultative hypermetropia). By further exertion of the accommodation the point for which the eye is adjusted shifts from infinity to a finite distance from the eye; e. g., with an expenditure of 7 D of accommodation to 33 cm. and with an expenditure of 9 D of accommodation to 20 cm., which, therefore, represents the near point, *P*, when $A = 9$ D. To produce an adjustment for 33 cm. an expenditure of 7 D of accommodation is required, so that of the total range of accommodation but 2 D are left for the positive portion of the range, while 7 D are already put into play—constituting thus the negative portion of the range. In this case, therefore, continuous work at 33 cm. would be impossible.

B. Displacement of the region of accommodation by a correcting convex glass. The far point now lies at infinite distance, the near point at 11 cm., as in the case of an emmetrope in whom $A = 9$ D. For a working distance of 33 cm., 3 D of accommodation are required, so that the positive portion of the range amounts to 6 D, the negative to 3 D.

diminishing steadily with advancing years, and in old age becoming [almost] zero. [See Fig. 377.] Every variety of hypermetropia, therefore, even the slightest, becomes absolute in advanced age.

781. Near Vision.—While the hypermetrope needs accommodation even for distant vision, this is much more the case for *seeing near by*. The amplitude of accommodation, *A*, of the hypermetrope is the same as that of the emmetrope and myope. To be sure, the near point in hypermetropes

lies farther off from the eye, but this is simply because a part of A is employed in the correction of hypermetropia, and only the remainder is left to effect the adjustment for shorter distances. For the same reason the hypermetrope requires for a certain working distance more accommodation than the emmetrope, the excess, in fact, corresponding precisely to the amount of the hypermetropia. Let us assume that work has to be done at a distance of 33 cm. For this purpose the emmetrope must use an accommodation of 3 D. A hypermetrope with $H = 4$ D also uses the same amount of accommodation; but he must employ 4 D more to conceal his hypermetropia, so that altogether he must make an accommodative effort of 7 D (Fig. 388 A). Now, as his range of accommodation is no greater than that of an emmetrope, this great amount of accommodation causes him proportionally more trouble. He may be said to be always dragging about with him a deficit in his accommodation, namely, the quota of the latter necessary for the correction of the hypermetropia—a deficit which causes him to tire quickly when doing near work (asthenopia). At first, vision near by is distinct and the work goes on well; but after a little while the objects, print, near work, etc., begin to grow indistinct and are blurred as though enveloped in a slight haze. This is owing to the fact that the over-strained accommodation gives way, and the eye then ceases to be properly focused. A short period of rest, during which the eyes look at distant objects or are kept closed, enables them to continue the work. But the same obscuration soon sets in again and compels another pause. These periods of enforced rest are the more frequently repeated and are of greater duration the longer the work is kept up. With them are associated pain in the eyes, and more especially pain in the forehead and headaches. The symptoms just described at first make their appearance only after quite prolonged work—i. e., toward evening. But afterward they keep coming on earlier and earlier all the time, so that the work has to be set aside after even a short period of exertion. After quite a prolonged rest—for example, after the repose of Sunday or upon laying work aside for several weeks—the symptoms probably disappear for a number of days in succession, but only to appear again in the old way and in an even more aggravated fashion. They are dependent upon an exhaustion of the ciliary muscle, and are hence comprised under the name of *asthenopia accommodativa* to distinguish them from asthenopia muscularis (see page 790), and asthenopia nervosa (see page 646).

The injurious effect of hypermetropia upon vision at near points furthermore finds expression in the fact that *presbyopia* sets in earlier than in emmetropic eyes. At the same age—i. e., with the same range of accommodation—the near point of the hypermetrope is situated farther from the eye than is that of the emmetrope. In an emmetrope of about forty years with a range of accommodation of 5 D, P lies at 20 cm. ($100 \div 5 = 20$). A hypermetrope having a hypermetropia of 2 D would at the same age and

with the same range of accommodation have his near point at 33 cm. (corresponding to 3 D), since he has to use 2 D of his range of accommodation for correcting his hypermetropia. Such a hypermetrope, therefore, would be already on the threshold of presbyopia at the age of forty, i. e., almost ten years earlier than an emmetrope.

Myopia is a state which is the direct opposite of hypermetropia, and yet there are cases in which these two conditions might be confounded with each other. When the hypermetropia has attained a very high degree, even the strongest effort of accommodation proves insufficient for rendering the vision at near points distinct. Then the hypermetrope gives up altogether the attempt to focus his eye for near points and holds the object as close as possible so as to get large retinal images, just as amblyopic patients do (see page 848). In this way very small print is often read well at a distance of a few centimetres, and, as at the same time distant vision is pretty bad, such a condition may easily be regarded as myopia. Examination with glasses and with the ophthalmoscope, however, will at once rectify the diagnosis.

The difference in the capacity of the emmetropic, the myopic and the hypermetropic eye for close work is explained by the difference in the position of their *region of accommodation* (see page 854). In emmetropia it has its normal position, in myopia it is carried inward (3, Fig. 372), and in hypermetropia it is displaced outward. For example, suppose that an emmetrope twenty-three years old has a range of accommodation of 9 D. His region of accommodation then extends from ∞ to a point 11 cm. from the eye where his near point is situated. Let us compare with this an eye having the same range of accommodation but with a total hypermetropia of 4 D. In this case R lies 25 cm. $(100 \div 4 = 25)$ behind the eye (Fig. 388 A). To change its adjustment from this far point to infinity, the eye must employ 4 D of its accommodation. Hence, out of its whole range of accommodation of 9 D the eye has only 5 D left, and by means of this it is enabled to approach to within 20 cm. of the object. This distance, therefore, is the near point(P) of the eye. Hence the region of accommodation of the latter is displaced in comparison with that of an emmetropic eye having the same range, and in such a way that the near point is made to recede 9 cm. farther away (from 11 to 20 cm.), while at its other end a part of the region of accommodation is made to lie behind the eye. But as this latter portion can not be utilized, and, on the other hand, the recession of the near point hinders the vision of objects close by, the displacement of the region of accommodation is unfavorable to the availability of the eye for working purposes.

The use of a convex glass does not indeed enlarge the region of accommodation, but it nevertheless removes the accommodative troubles, since it brings the region of accommodation into a more available position—it now, in fact, lying entirely in front of the eye (Fig. 388 B).

The calculation of the range of accommodation in hypermetropia is made according to the same rules as in emmetropia. P can be found directly, R is determined by the convex glass which corrects the error. $A = P - R$; hence in the example selected, $A = 5\text{D} - (-4\text{D}) = 9\text{D}$. R must be taken as a negative because it lies behind the eye, i. e., represents a deficiency in refractive power (page 865 Note).

The position of R, and consequently the Ht, can be estimated from the position of P, if we know the range of accommodation of the subject. Since $A = P - R$, $R = P - A$. If in the above example P had been found to be 5 D, and for the age of twenty-three A is assumed to be 9 D, we would have $R = 5\text{D} - 9\text{D} = -4\text{D}$. Hence, $Ht = 4$ D. [Really, owing to the wide variations shown by A at any given age (see Fig. 377) we can not estimate even approximately the hypermetropia in this way. If P falls below the lower

limit for the age (Fig. 377) all that we can say is that either the accommodation is actually subnormal or there is some latent hypermetropia. Which is the case will be determined by the use of a cycloplegic which will show the total amount of hypermetropia present.—D.]

782. Etiology.—The shortness of the eyeball, which is the cause of hypermetropia, is *congenital.* Almost all newborn children are hypermetropic, their eyes being originally constructed too short in proportion to the refractive power of the media. As the child grows, the eyeballs elongate in proportion, so that they acquire their requisite axial length and become emmetropic—indeed, the elongation may even shoot beyond the mark and pass into myopia.[7] On the other hand, the elongation of the eye may fail to take place to a sufficient degree, so that a certain amount of hypermetropia remains during the whole life. This is the typical hypermetropia of which we are speaking. Slight degrees of it are so frequent that hypermetropia must be regarded as the most frequent refractive state, being indeed [much] more frequent than emmetropia itself. Higher degrees of hypermetropia, on the other hand, are pretty rare—much rarer, in fact, than the high degrees of myopia. They can be recognized at once by external examination of the eye, which shows that the eyeball is distinctly diminished in size, and that the anterior chamber is shallower and the pupil more contracted than usual. If the eye is turned strongly inward, we see that the equatorial region of the ball, which comes into view in the outer part of the palpebral fissure, presents a particularly sharp curve as it turns backward, and thus gives evidence of the shortness of the axis of the eyeball. The ophthalmoscope shows that the interior of the eye is sound. The hypermetropic eye, accordingly, is an optically defective but otherwise healthy eye, as opposed to the myopic eye, which is diseased, and is hence threatened by dangers of various kinds.

In the *extreme degrees of hypermetropia,* however, the eye is no longer normal as a whole. It is abnormally small even from the time of birth (slight degree of microphthalmus), and many of these eyes show other signs of disordered development, such as a strikingly small cornea, marked astigmatism, deficient visual acuity due to incomplete formation of the retina, and other congenital anomalies.

Hypermetropia does not change in its amount [materially after childhood]; it remains *stationary.* It is true that to the laity it appears as if it increased with years, because vision at near points becomes steadily worse; but this is not due to an increase in the error of refraction, but to a diminution in the power of accommodation, the result of which is that less and less of the hypermetropia is concealed all the time. [In advanced life it is apt to increase somewhat (see page 883).—D.]

[7] [The proportion of hypermetropes, therefore, diminishes during the growing period. In 4800 school children in New York examined under a cycloplegic, 91 per cent of those at the age of 5 and only 48 per cent of those at 16 were hypermetropic (Tenner).—D.]

783. Treatment.—There is no way of curing hypermetropia—i. e., of transforming it into emmetropia. All that we can do is to make vision distinct by means of properly selected glasses, and enable it to be performed without asthenopia.

When the hypermetropia is not great and the range of accommodation is good, glasses for distant vision are not as a general thing required. In the contrary event, convex glasses are given which correct the manifest hypermetropia. [See, however, page 901.]

Of more importance than the glasses for distance are those for near points or for working. A priori it would seem best to have the hypermetrope start at once by wearing that glass which corrects the total hypermetropia and so convert him into an emmetrope; but in most cases this would not be borne, owing to the tendency that he has to render his hypermetropia partly latent by his accommodation; he would soon quite forget altogether how to correct his hypermetropia by his accommodation in case of necessity. We therefore confine ourselves to correcting the hypermetropia by glasses only as far as is required to relieve the asthenopia. For this purpose it is generally sufficient to give for working purposes a glass which is somewhat stronger than the manifest hypermetropia. Inasmuch as the latter increases with the age, the hypermetrope must keep on getting stronger and stronger glasses. It is only when he has reached an age at which his range of accommodation = 0 [or at least has reached its minimum], and hence his whole hypermetropia has become manifest, that he can keep on using the same glasses.

Full correction of the total hypermetropia, and constant wearing of the glasses for distance and near, are indicated only in those cases in which we have to combat a strabismus convergens which has developed in consequence of hypermetropia. In this case we must sometimes first paralyze the accommodation with atropine in order to have the correcting glass tolerated.[8]

[8] [For this whole question of glasses in hypermetropia, see also page 901.—D.]

CHAPTER V

ASTIGMATISM

784. Definition.—By astigmatism[1] (*As*) we understand that refractive condition of the eye in which parallel rays falling upon the eye are *not at any spot* brought to a common focus. This is the case when the curvature of the refracting media is irregular. We distinguish two kinds of astigmatism: regular and irregular.

(*a*) *Regular Astigmatism*

This is present when the curvature of the refracting media is regular in each meridian considered by itself, but the separate meridians are distinguished from each other by differences in curvature. The ordinary site of regular astigmatism is in the cornea. In Fig. 389[2], let $v\ h\ v_1\ h_1$ represent the circumference of the cornea, and $v\ v_1$ its vertical meridian, which has a curvature such that the rays passing through it come to a focus at f. In the meridian immediately adjoining the curvature becomes a little greater, and in the succeeding meridians it increases still more, so that it reaches its maximum value in the horizontal meridian, $h\ h_1$. The rays passing through the latter may be supposed to intersect at a point as near as f_1. In this case we would have one meridian (the horizontal one) which refracts rays most strongly, and one perpendicular to it (the vertical meridian) which refracts most feebly; and corresponding to these are the most anterior and posterior foci, f_1 and f. These two meridians thus distinguished from the others are called principal meridians; those meridians lying between them represent all intermediate stages of curvature and refractive power. We see that when the refracting surface is of this character there is no point at all at which all the rays passing through the surface will unite. The image of a point cast by such a surface upon a receptive screen is therefore not a point, but a diffusion circle. In reality, however, the image does not always have a circular shape. On the contrary, its shape depends upon the spot where the retina is situated and cuts the conical beam of rays. Let us assume that the retina is at the point, marked 1. Here the rays passing through the horizontal meridian are already brought closer together than those incident upon the vertical meridian; hence the section of the cone of rays is an

[1] From ἀ, privative, and *στίγμα* point. [Called also *astigmia*.—D.]

[2] The representation of diffusion images given in the figure is not precisely but only approximately correct, and that for the following reason. In a cylindrical lens (Fig. 355) the meridian, *bbb*, which is perpendicular to the axis is the only one that has a spherical curvature. The curvature of the intermediate meridians is not spherical but elliptical, for which reason the rays that pass through them are not brought to a focus at all. [The idea here is that, while the non-astigmatic cornea is practically a spherical convex lens, the astigmatic cornea is such a lens with a cylinder superadded. This conception itself is not quite precise, yet serves to show how an astigmatic sees. Just as a myope sees like an emmetrope wearing a convex sphere and a hypermetrope like an emmetrope wearing a concave sphere (page 865, Note), so an astigmatic sees like an emmetrope wearing a cylinder with or without a sphere.—D.]

erect ellipse. At 2, where the rays of the horizontal meridian come exactly to a focus, the image of the point is a vertical line. In the same way the shape of the cross section of the beam—i. e., the shape of the diffusion image of the point—can be ascertained for the more posteriorly situated points from 3 to 7. This cross section is sometimes an erect or horizontal ellipse, sometimes a vertical or horizontal line, according as it is more or less distant from the refracting surface. It is only at 4 that there is really a diffusion circle, because here the rays passing through the horizontal meridian diverge to the same extent that those of the vertical meridian converge.

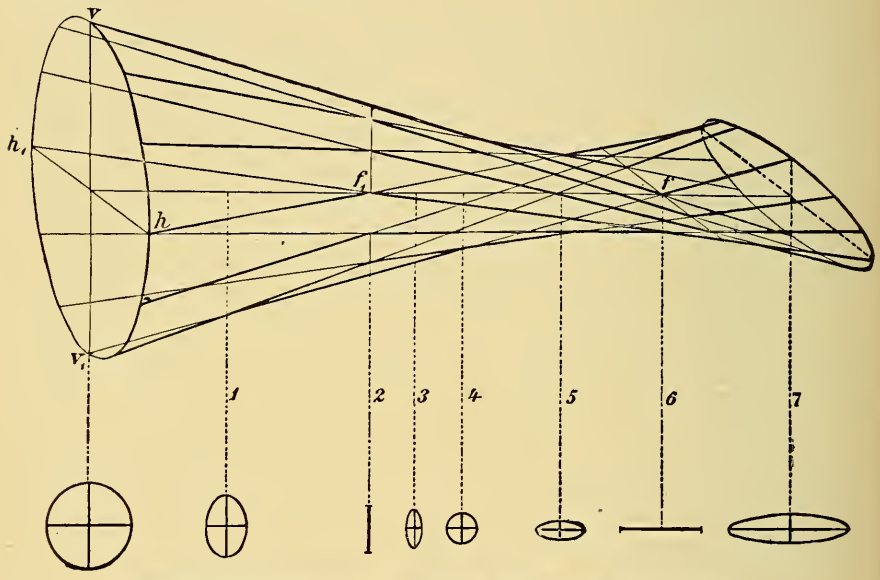


Fig. 389.—Refraction of the Rays in Regular Astigmatism.

785. Symptoms.—The *vision* of an astigmatic person is not simply indistinct, like that of one who is near-sighted or far-sighted, but presents special peculiarities on account of the elongated form of the diffusion images. Circular surfaces—e. g., the full moon—appear elliptical. Straight lines sometimes look distinct, sometimes indistinct, according to the direction that they take. Let us assume that we have before us an astigmatic who sees the diffusion image of a point under the form of a vertical line (2, Fig. 389). If this man looks at two lines standing perpendicular to each other (Fig. 390 A) the horizontal line appears broadened and indistinct but the vertical line seems sharply defined. For, we may imagine these two lines to be composed of an infinite number of points. Each one of these points appears upon the retina of the astigmatic patient under the form of a short vertical stroke, and the horizontal line therefore appears under the form of a series of such vertical strokes,

which coalesce and constitute a band of a certain degree of breadth (Fig. 390 B). In the vertical line the vertical strokes are superimposed and cover each other, so that the line appears sharply defined. Only the uppermost and lowermost diffusion lines extend beyond the terminal points of the vertical line and make it seem somewhat longer than it is. Thus, for every astigmatic person there is one direction in which straight lines appear most distinct, and one, perpendicular to it, in which they appear most confused. Most people looking attentively at Fig. 391 will find that, of the radii of the star, two situated opposite to each other are distinguished by being particularly black, while the radii which are placed perpendicular to them are the ones that look most pale and hazy. If one is unable to perceive this phenomenon with the naked eye, he can readily do so if

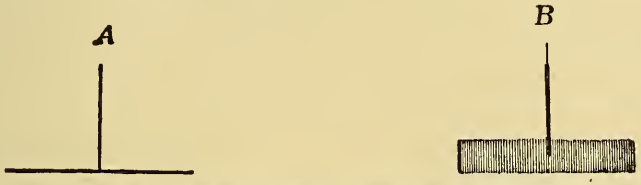


Fig. 390.—Retinal Images in Regular Astigmatism.
A, two lines placed perpendicular to each other; B, their image upon the retina of an astigmatic person.

he makes himself artificially astigmatic by placing a cylindrical glass before his eye.[3]

[As the example above given shows, vertical lines appear distinct when the horizontal meridian is emmetropic. At the same time horizontal lines appear indistinct. These lines, on the other hand, would be distinct if the vertical meridian were emmetropic. But the vertical meridian can be rendered emmetropic by adding the glass that corrects its refractive error. In other words, the glass with which a given set of lines is seen distinctly measures the refraction of the meridian at right angles. (For the application of this principle in ophthalmoscopy see page 106 and for its application in the correction of astigmatism see page 893.)—D.]

The *vision* in regular astigmatism is distinguished from the vision in other errors of refraction by the fact that objects are distorted, and that not all their parts are seen with the same indistinctness. If the principal meridians are respectively vertical and horizontal, the horizontal strokes of the letter *E* will appear distinct, the vertical ones indistinct, or vice versa. The astigmatic subject then tries from the parts which he does see to guess the rest. If we test the visual acuity of a myope at a distance of 6 metres, he will read Snellen's test types well down to a certain line, and then stops because he does no see any farther down. The astigmatic patient, on the contrary, often reads the whole card down to the bottom, but tells almost every letter wrong. In fact, he takes refuge in guessing—an attempt, however, which gives rise to a peculiar and very unpleasant form of asthenopia. [Another cause of disturbed vision in astigmatics is the apparent motion of objects—wheel-like figures appearing to revolve and check patterns seeming to dance. This causes asthenopia and vertigo.—D.]

[3] In default of this, an ordinary convex or concave glass may be used, which is held obliquely before the eye [see page 835].

786. Varieties.—The principal meridians usually intersect at a right angle, and the cross formed by them is generally vertical, more rarely oblique. The rule is, that the vertical meridian has a greater curvature than the horizontal (astigmatism "with the rule," or direct astigmatism); but the reverse condition (selected for Fig. 389 because of being more readily represented) also occurs, and is then known as astigmatism "against the rule" or inverse astigmatism. The degree of astigmatism is expressed by the difference between the meridians of greatest and of least refraction. As long as this difference remains below 1 D the astigmatism may be regarded as physiological, since most eyes are affected with a slight error of curvature of this sort; but as soon as the astigmatism amounts to 1 D or over it must be regarded as pathological. It then affects the visual acuity, and in many cases causes asthenopic troubles. [An astigmatism of even 0.25 D may do this, particularly if the eyes are used much for fine work, and an astigmatism of 0.75 D so often gives rise to trouble that it can scarcely be regarded as physiological.—D.]

Various *kinds* of astigmatism are distinguished, according to the character of the refraction of the principal meridians. If one meridian is emmetropic and the other hypermetropic, the condition is called simple hypermetropic astigmatism; but if both meridians are hypermetropic, it is compound hypermetropic astigmatism. In analogous fashion we speak of simple and compound myopic astigmatism. If one meridian is hypermetropic and the other myopic, the condition is known as mixed astigmatism.

The *sort of regular astigmatism* that is present, whether hypermetropic, myopic, or mixed, does not depend upon the curvature of the cornea, but upon the situation of the retina. If the latter is situated at point *2* (Fig. 389), where the rays passing through the horizontal meridian come to a focus, this meridian has an emmetropic refraction. But the vertical meridian is hypermetropic, since the rays passing through it would meet behind the retina. In this case, then, there would be simple hypermetropic astigmatism. If the position of the retina were farther forward—e. g., at *1*—both meridians would be hypermetropic; that is, compound hypermetropic astigmatism would be present. If the retina was situated at any point between *2* and *6*, the rays passing through the horizontal meridian would have their focus in front of the retina, those passing through the vertical meridian behind it, and mixed astigmatism would exist. If the retina is situated at *6*, simple myopic astigmatism is present, because there is emmetropia for the vertical meridian and myopia for the horizontal meridian. Finally, if the retina should be situated still farther back—that is, behind the foci of both meridians—myopia would be present in both the latter or there would be compound myopic astigmatism. Hypermetropic astigmatism is the kind most frequently occurring; mixed astigmatism is the rarest.

The astigmatism is frequently found to be greater after atropinization than before (Dobrowolski). [Sometimes it is less.—D.]

787. Subjective Determination.—How astigmatism is determined and corrected may be illustrated by the following example: We first make the astigmatic patient look at Fig. 391, or some similar stellate figure, and thus, for instance, determine that the horizontal radii of the star appear blackest. From this we conclude that vertical lines are seen distinctly, because the horizontal radii are composed of vertical lines. If

vertical lines appear distinct, the diffusion lines or diffusion ellipses must be vertical (Fig. 390)—i. e., the adjustment for the horizontal meridian must be correct, or at least be better than the adjustment for the vertical meridian. We now place a stenopæic slit before the eye, in the horizontal meridian first, and determine the refraction of the latter by means of spherical glasses. Suppose that this refraction is $M = 1$ D. Now making a test of the refraction with the slit in the vertical position a myopia of 3 D is found. Accordingly, myopic astigmatism (*Am*) is present, and one, namely, of 2 D, since the degree of astigmatism is given by the difference in refraction of the two meridians. The correction of this astigmatism would have to be made by two concave cylindrical glasses, the axes of which run vertical and horizontal. The cylindrical glass refracts most strongly in the direction perpendicular to its axis (see page 835). Hence, to correct the horizontal meridian, we must place a cylindrical glass of —1 D with the axis vertical, and for the vertical meridian a glass of—3 D with the axis horizontal. This is written as follows:

—1 D cyl. vert. ⊂ —3 D cyl. horiz.[4]

In cases where the sign of both cylinders is the same, a simplification of the combination is obtained in the following way: If, in the example chosen, we give a spherical glass of—1 D, this will correct the horizontal meridian to the point of emmetropia,

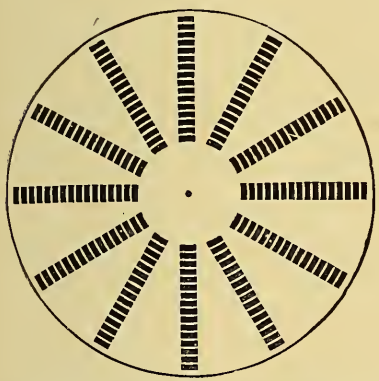
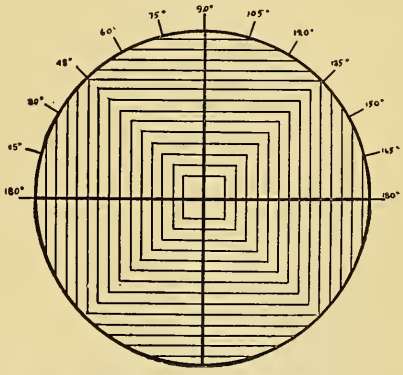


Fig. 391.—Test Object for determining the Position of the Principal Meridians in Regular Astigmatism.



[Fig. 392.—Verhoeff's Chart.—D.]

and the vertical meridian to 2 D. To get the complete correction, therefore, we only need to add to the spherical glass a —2 D cyl. horiz. We would therefore prescribe

—1 D sph. ⊂ —2 D cyl. horiz.

If the two principal meridians are not vertical and horizontal, but oblique, their direction must be denoted in degrees of arc. In consonance with international agreement, this is done by stating the position of the upper end of the meridian. To define this position, the trial frame carries the upper half of a circular arc divided into degrees, the numbering of which begins with 0° at the nasal end of the horizontal meridian and extends to 180° at the temporal end of the horizontal meridian. [See Fig. 358, and for other methods of indicating the principal meridians or axis of astigmatism, see Figs. 356 and 357. Fig. 356 represents the method which still prevails throughout America.—D.]

As can be seen from the example above given cylindrical glasses may be combined both with spherical glasses and with cylinders [see page 835], and they may likewise be

[4] [According to the notation prevailing in this country (see page 835), this would be written: —1.00 cyl. axis 90° ⊂—3.00 cyl. axis 180° So also the equivalent formula mentioned just afterward would be written:—1.00 sph. ⊂—2.00 cyl. axis 180°.—D.]

combined with prisms. Cylinders are generally prescribed in spectacles in order to insure the axis of the glass being in proper position.

[The astigmatism test-card shown in Fig. 391 is usually replaced in this country by one in which each radiating bar is composed of three parallel lines; the horizontal bar, for instance, being made up of three long horizontal lines instead of a series of short vertical ones. In this case, of course, if the horizontal bar appeared the blackest it would show that the patient saw horizontal lines most distinctly, i. e., that his vertical meridian was most nearly emmetropic, and we should, therefore, place the axis of the correcting cylinder vertical. Many modifications of this chart have been devised. One of the best is that of Verhoeff (Fig. 392) which is used as follows:—The chart is rotated until one set of lines comes out most distinctly, making the two corresponding segments stand out black and sharp, while the other two segments look gray. Cylinders are then applied with their axes parallel to the less distinct lines of the chart. The cylinder which, with its axis in this direction makes all the lines look alike is the one that corrects the astigmatism. Other devices, such as disks or letters (Pray's letters) composed of lines running in different directions are used for the same purpose.—D.]

The result which we obtain by the methodical method of determining the astigmatism above described we can get at more quickly in the following way: If we suspect astigmatism, we put on a weak cylindrical glass and rotate it before the eye. If there is no astigmatism worth mentioning, the patient sees worse through the cylindrical glass, no matter what direction it occupies in front of the eye. But if astigmatism is present, the sight will become better when the glass is in a certain position, worse when it is in another. In this way we find the direction of the principal meridians. Then, convex or concave cylinders of varying strength, and either alone or combined with spherical glasses, are successively placed before the eye in a direction corresponding to the principal meridian, until the best combination has been found. [See also page 900.]

788. Objective Determination.—The objective determination of astigmatism can be made in different ways. Astigmatism manifests itself with the *ophthalmoscope* by the alteration in the shape of the papilla, which in regular astigmatism appears elongated either lengthwise or laterally (see page 111); in irregular astigmatism it appears irregularly distorted. In the erect image, in cases of regular astigmatism, the horizontal and vertical vessels are not seen distinctly at the same time, as, owing to the difference in their refraction, they require different correcting glasses. It is on account of this fact that it is possible to determine the astigmatism with the erect image by finding for each of the two principal meridians the correcting glass with which the vessels of this meridian are seen most distinctly [page 106]. Astigmatism can also be made out and measured [most accurately] by means of the *shadow test*, [see page 110.]

Regular corneal astigmatism can be determined by measuring the radii directly. This is done by means of the *ophthalmometer* which was first constructed by Helmholtz; Javal and Schiötz then modified the instrument so that it could be applied practically, and it is now in general use. It is the task of the ophthalmometer to measure the reflected images on the cornea. From this the curvature of the latter can be deduced; for, the greater the curvature of a convex mirror, such as the cornea represents, the smaller are the reflected images. The reflected images which serve for making the measurement should be quite large, because then the changes in their size which take place when the radius of the cornea alters have also large absolute values and hence are more readily demonstrated by measurement. For producing the reflected image, therefore, an object is selected whose image on the cornea is so large as to be equivalent to at least a fourth of a meridian of the cornea. If we should take for such an object an upright white cross, the reflected images of the two arms of the cross would be equally long only in case the cornea had a precisely spherical curvature. If, as is generally the case, the vertical meridian is more curved than the horizontal, the verti-

cal arm of the cross would look shorter in the reflected image than the horizontal; and from the difference between the two we might compute the difference between the radii of curvature of the two principal meridians. It is an easier matter to take, instead of the whole cross, only one arm of it, which is first placed horizontally then vertically, and is measured in both instances. We, accordingly, give the object a considerable elongation, so that its reflected image shall extend mainly over only one meridian of the cornea; e. g., if the object is horizontal its reflected image will belong to the horizontal meridian (Fig. 394). In order to be able to vary the size of the object, we take what may be called simply two opposite edges of it. These are represented by the two white plates α and β which are attached to the circular arc B, and are movable on it (Fig. 393). By carrying them nearer together or further apart, the object as

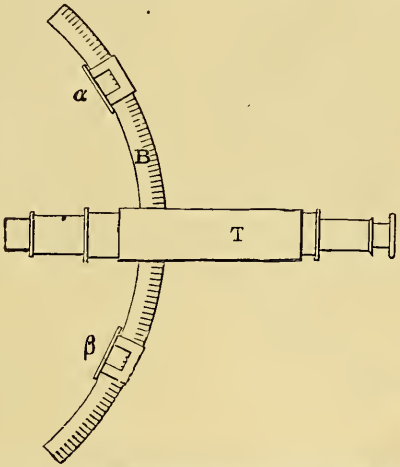


Fig. 393.—Ophthalmometer of Javal-Schiötz

Fig. 394.

Fig. 395.

Fig. 396.

Figs. 394 to 396.—Reflected Images on the Cornea as Seen by the Ophthalmometer.

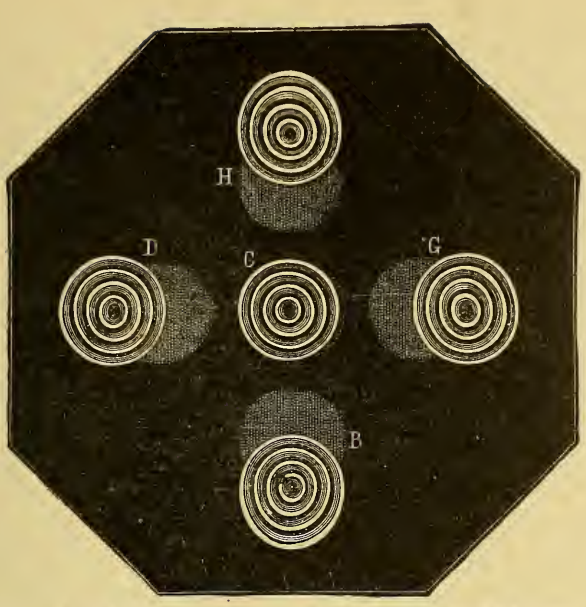
a whole is diminished or enlarged and so too is its reflected image on the cornea. If the size of the object is constant and its distance from the eye that is being tested is also constant, the size of the reflected image will vary only with the degree of the corneal curvature. Hence the latter can be computed if the size of the reflected image is known. *Helmholtz* was the first to measure this precisely, using the method of duplication of the reflected image. The apparatus designed to effect this is placed in the telescope which passes through the center of the arc B, and shows the reflected image in a magnified form. In the ophthalmometer of *Javal and Schiötz*, which is now generally used, the doubling of the image is accomplished by a prism of double-refracting Iceland spar, and in the instrument made by Kagenaar by means of two glass prisms whose refracting angles meet in the center. Hence, if we look through the telescope at the cornea we see the latter and also the images upon it double. The doubling is

so contrived that the two images partly overlap (in Fig. 395 the image a_1 b_1 partially overlaps the image ab). If now by approximating the two plates α and β on the arc B we make the object smaller, its reflected image also becomes smaller; a advances toward b, and a_1, toward b_1. At the same time a_1 also comes closer and closer to b, until at last when the plate α has been brought sufficiently close to plate β, their inner edges which face each other come in contact (Fig. 395). We now read off on the arc the distance between the two plates, i. e., the size of the object; and since the distance of the latter from the cornea on which the reflection is cast and also the amount of the doubling are known, we can compute the size of the corneal image and from this the curvature of the cornea. For convenience the arc B is so graduated that from the position of the plates the radius of the cornea in millimetres or the refractive power of the cornea in dioptries can be read off directly on it.

[Fig. 397.—Placido's Disk.—D.]

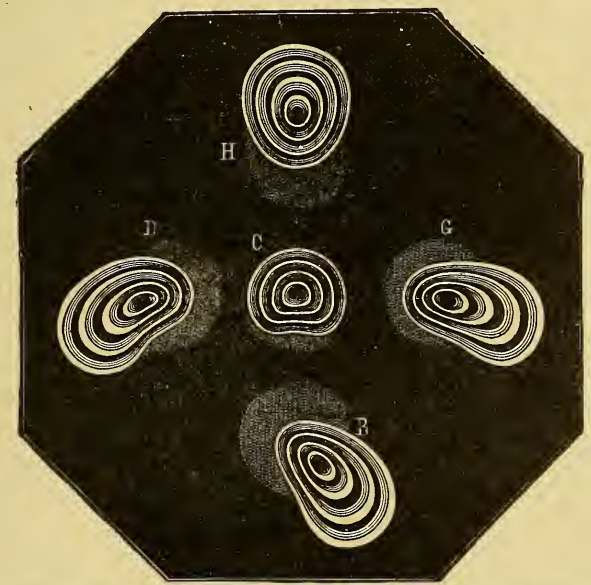
If now the arc is placed vertical its reflected image will lie in the vertical meridian of the cornea. If this has the same curvature as the horizontal meridian the reflected image will remain unaltered. If, however, there is a greater curvature in this meridian as is the case in astigmatism with the rule, the reflected image becomes smaller. a and b come closer together, and so do a_1 and b_1, whence it follows that now a_1 slides over on b and partly overlaps it (Fig. 396). By enlarging the object, i. e., by separating the plates α and β from each other, we can now enlarge the double reflected image to such an extent that a_1 and b again come just in contact. If then we again read off on the arc the curvature of the cornea, we shall ascertain how much the latter has increased in comparison with the horizontal meridian, i. e., we shall ascertain the amount of the astigmatism. But we can also deduce this directly from the amount of overlapping of a_1 and b. For this purpose plate α does not constitute a rectangle but a stair figure with six steps so measured off that an overlapping of one step corresponds to an increase of refractive power of one dioptry. Thus in the case represented in Figs. 395 and 396 the vertical meridian would be 3 D more refractive than the horizontal.

[In using the ophthalmometer the arm B is rotated until the meridian is found in which the images of α and β are furthest apart. This is the meridian of least corneal curvature or first *principal meridian*. The direction of this is read off on a scale attached to the instrument, and then the arm is rotated until the images are closest together (meridian of greatest corneal curvature or second principal meridian). The difference in refraction between the two meridians is then measured in the way described. The two principal meridians are almost always at right angles to each other.—D.]



[Fig. 398.—Keratoscopic Images in a case of Regular Inverse Astigmatism. After Javal (from Norris and Oliver).

C, reflex, from center of cornea The reflex is an ellipse with the vertical diameter the longer. *B*, *D*, *H*, *G*, reflexes from outlying portions of the cornea where the astigmatism is more marked and is also slightly irregular.—D.]



[Fig. 399.—Keratoscopic Images in a Case of Irregular Astigmatism. After Javal (from Norris and Oliver).—D.]

The determination of the corneal astigmatism with the ophthalmometer facilitates very greatly the determination of the total astigmatism present, but does not alone suffice for this purpose; in every case the examination of the eye with glasses must also be made. For in the first place the ophthalmometer gives only the difference in refraction between the two principal meridians, but not their absolute refraction; it does not tell us whether either one of these meridians is hypermetropic or myopic or to what extent they are so. In the second place the astigmatism that is determined with the ophthalmometer does not agree precisely with that which is found with tests by glasses, for the ophthalmometer shows only the astigmatism of the cornea, but the glasses give the astigmatism of the whole eye (functional *As*). Yet the difference between one kind of astigmatism and the other is almost always in the same sense, there being added to the corneal astigmatism an astigmatism of 0.5—1 D against the rule, the seat of which is probably in the lens. Hence the functional astigmatism appears to be 0.5—1 D less than the corneal astigmatism in case the latter is with the rule, and as much greater than the corneal astigmatism when the latter is against the rule. [This statement has many exceptions. The corneal astigmatism determined by the ophthalmometer differs from the total astigmatism by a variable amount. Moreover, the axis indicated by the ophthalmometer does not always coincide with the true axis. —D.]

A very useful instrument for determining whether there is actually any great amount of corneal astigmatism present is *Placido's keratoscope* [see Fig. 397]. This consists of a disk of cardboard, one surface of which bears a number of black concentric rings upon a white ground. An aperture in the center of the disk, and corresponding to the center of the rings, allows the observer to look through the disk. The disk is held so that the side with the rings upon it is turned toward the eye that is being examined, and so that the plane of the disk is parallel to the base of the cornea. If now we look through the central aperture at the eye, we see the rings mirrored upon its cornea. If the cornea has the normal curvature, these rings appear perfectly circular; if not, the rings are transformed into ellipses, or show irregular bulgings, according as regular or irregular astigmatism is present. [See Figs. 398, 399.]

789. Etiology and Treatment.—The cause of regular astigmatism in the great majority of cases is a *congenital* irregularity of the curvature of the cornea—a condition which is apt to be transmitted by heredity. In high degrees of congenital astigmatism it is generally impossible, even with perfect correction of the astigmatism, to bring the visual acuity up to the normal pitch; and these cases are associated not infrequently with other defects in the development of the eye. Congenital astigmatism is often present in both eyes, although not always in the same degree, and the direction of the principal meridians is apt to be symmetrical in the two eyes. *Acquired* astigmatism may have its cause in the cornea or in the lens. The former variety occurs when the curvature of the cornea has been altered, either because of diseases affecting it, or still more frequently because of operations. After every cataract operation, and in fact even after an iridectomy, a certain degree of corneal astigmatism develops, which indeed diminishes with the consolidation of the cicatrix, but seldom disappears entirely. The lens gives rise to regular astigmatism when it is obliquely placed, as, for example, in case of subluxation. This state

of things can readily be imitated experimentally if we look through a spherical lens obliquely. The print then looks as if in a state of astigmatic distortion, and the separate radii of Fig. 391 seem to differ in distinctness. Accordingly, an obliquely placed spherical lens acts also as a cylindrical one (pages 835, 891, Note). Many astigmatic patients who wear spherical glasses hit upon this fact themselves; to see better, they place their spherical glasses in such a way as to look obliquely through them.

The *treatment* of astigmatism consists in correcting it as precisely as possible by means of cylindrical glasses [page 902]. In this way distinct vision can be secured, and at the same time the asthenopia is relieved.

(*b*) *Irregular Astigmatism*

790. Irregular astigmatism occurs when the curvature in any one single meridian is not everywhere alike, so that the rays passing through the same meridian are never united in one point [or when successive meridians differ irregularly in refraction.—D]. A certain degree of irregular astigmatism must be regarded as physiological, as it is present in every eye, its location being the lens. The individual sectors composing the latter do not all have the same refracting power, this being probably due to unequal curvature of their surfaces. Accordingly, the images of a point which these sectors cast do not all fall upon the same spot in the retina, although they come so close together that for the most part they overlap.

Under pathological conditions—e. g., in beginning opacity of the lens—this lenticular astigmatism is so much increased as to give noticeable trouble. As the refractive power of the separate sectors of the lens becomes more and more different, the images produced by them recede farther and farther apart, so that ultimately they appear entirely distinct from each other. It is in this way that monocular polyopia develops in incipient cataract (see page 527). A very high degree of irregular astigmatism occurs in subluxation of the lens when the lenticular displacement is so considerable that part of the pupil still has the lens in it and part is aphakic.

Pathological irregular astigmatism originates from the cornea even more frequently than it does from the lens. It is found in this situation as an accompaniment of marked regular astigmatism, and still more often in consequence of pathological processes—e. g., in faceting of the cornea after ulceration, or in flattening or ectases of the entire cornea.

Irregular astigmatism makes objects appear irregularly distorted and sometimes also look multiple [polyopia], and in this way diminishes the visual acuity. It is impossible to correct it by glasses. In some cases of irregular corneal astigmatism a stenopæic slit is of service for making out minute objects (see page 839).

[APPENDIX

[791. Determination of Refraction with Glasses.—In determining the refraction with the trial case, it is important to proceed according to some systematic plan. A good way is as follows: Unless we know the patient is myopic, we begin with a convex spherical glass (one of 0.50 D if the vision is already good, one of 1 or 2 D, if the vision is poor). We change this rapidly until no further improvement in sight is secured.[5] Then we begin adding to this strongest convex glass accepted cylinders, weak if the vision is good, stronger if the vision is poor, and at axes of 90°, 180°, 45°, and 135°, respectively. We try convex cylinders first, and if these do not help, concave cylinders. If any cylinder improves, we leave it on in the axis indicated. If neither convex spheres nor cylinders improve, we try concave cylinders or put in concave spheres, and when the maximum vision attainable has been secured with these, add cylinders to them. Thus, in one way or another, we get an approximate spherical or sphero-cylindrical correction. Or, we may get our first approximation with skiascopy or with the ophthalmometer. Suppose this approximate correction to be + 2.50 sph. ⊃ + 1.25 cyl. 90°. To this we add in turn—

+ 0.50 sph.		making	+ 3.00 sph. ⊃ + 1.25 cyl. 90°
+ 0.50 cyl.	90°	"	+ 2.50 sph. ⊃ + 1.75 cyl. 90°
+ 0.50 cyl.	180°	"	+ 3.00 sph. ⊃ + 0.75 cyl. 90°
− 0.50 cyl.	90°	"	+ 2.50 sph. ⊃ + 0.75 cyl. 90°
− 0.50 cyl.	180°	"	+ 2.00 sph. ⊃ + 1.75 cyl. 90°
− 0.50 sph.		"	+ 2.00 sph. ⊃ + 1.25 cyl. 90°

If any of these additions improves the sight we make the corresponding substitution and start with this as a new basis. Thus, if the addition of −0.50 cyl. 180° was an improvement (and the other additions were not), we substitute + 2.00 sph. ⊃ +1.75 cyl. 90° for our first trial combination, and then to this new combination make systematic additions as before. Pretty soon we come to a combination which is not improved by any addition (even of a 0.25 D sph. or cyl.). Then we try shifting the axis of the cylinder a little one way or the other. If this does not help, then we can be fairly sure that the combination last found is the best that can be had. We confirm by skiascopy (page 107). To do this we add a + 1D to the sphero-cylindrical combination found and determine if this produces reversal at one metre in all meridians. If not, slight changes are made in sphere, cylinder, and axis of cylinder until uniform reversal occurs.

If with our first combination we get comparatively poor sight (less than $\frac{20}{40}$) we would make additions of 0.75 or 1.00 D exchanging this for a 0.50 D, when the vision had been improved to $\frac{20}{40}$ or better. When the vision is about $\frac{20}{20}$ we can make additions of 0.25 D or even 0.12 D.

[5] Or, if we are not making our examination under a cycloplegic, until the added convex glass causes blurring. For the reason of this and the method of determining the highest acceptance in this case see page 882.

If no addition brings the vision above $\frac{20}{40}$ or $\frac{20}{50}$, we re-examine the eye carefully for pathological changes (faint opacities of the cornea or lens, disease of the fundus) and if these are absent, examine the patient for a central scotoma, especially a color scotoma.

In making the subjective test it is important that the glasses in the trial frame should be at the same distance from the eyes as the glasses worn by the patient. If the glasses in the trial frame are at a wrong distance they exert an effect stronger or weaker than that indicated by their number.—D.]

[**792. Cycloplegics and the Correction of Refractive Errors.**—In this country a cycloplegic is very frequently used in determining the refraction whether the case is one of hypermetropia, myopia, or astigmatism. *Homatropine* is the agent mainly in use, although some prefer *scopolamine* (in $\frac{1}{5}$-per-cent solution). Homatropine in 2-per-cent solution, instilled three or four times at intervals of ten or fifteen minutes suffices in almost all cases to produce in an hour or an hour and a half a complete relaxation of the accommodation. As the cycloplegia proceeds, a convex glass of 3 D or more should be added to the manifest correction and then the far and near points and the range determined with a Prince's rule (see page 857). When the range becomes less than 1 D, the cycloplegia may be regarded as fairly complete. The effect begins to abate pretty soon, but does not disappear completely until two or three days at least have elapsed. Some measurable effect, indeed, is often apparent five or six days after the instillation. The period of blurred sight may be materially abridged by repeated instillations of $\frac{1}{4}$-per-cent eserine with 1-per-cent pilocarpine.

The *practice in regard to the use of a cycloplegic* varies, although oculists here may be said to employ one in from 40 to 90 per cent of their refraction cases. The translator's own practice is to use homatropine whenever practicable, especially insisting upon its employment in children and where there is a suspicion of spasm of accommodation (difference between subjective and objective tests) or where there are evidences of convergence-excess (see page 780). Contrary to the statements generally expressed he has found it advantageous to paralyze the accommodation in patients between forty and fifty. It has a number of times been his experience to find patients of this age in whom the refraction could not have been determined accurately without a cycloplegic. He has found the latter particularly serviceable at the time between forty and forty-six when the accommodation is changing fast and when the patient, in struggling to use his failing accommodation, often over-exerts the latter for distance and conceals some part of his ametropia. Of course, in using homatropine for these elderly cases, we must exclude any suspicion of glaucoma, and be particularly careful to avoid using a cycloplegic when the pupil in a non-myopic patient is unnaturally dilated.

The gain [6] in certainty both for the physician and the patient that we get by using a cycloplegic is so great in comparison with the moderate

[6] A gain, be it noted, that obtains for the objective as well as the subjective tests.

inconvenience[7] produced, that it seems proper to employ it in all cases except in the very old and in those who are likely to develop glaucoma.

Occasionally homatropine fails to produce complete cycloplegia. This will be evident from the failure to obtain satisfactory or consistent results by subjective testing, from a continued discrepancy between the results of the objective and subjective tests (particularly between the test with the trial case and the shadow test) and from the persistence of over 1 D residual accommodation (page 901). This *failure of homatropine* to produce complete paralysis occurs especially in cases of spasm of accommodation. Here, as well as in convergent strabismus, *atropine* (1, 1½, or 2 per cent) should be instilled two or three times a day for one, two, or many days. Even with this we may not be able to secure complete relaxation.

In hyperopia and presbyopia it is usually necessary to make a *post-cycloplegic test* (page 882) five to seven days after the homatropine [if made earlier the test often misleads, because the accommodation has not returned completely.—D].

793. The *glass prescribed* after the refraction has been determined under a cycloplegic will depend upon various factors. Each case must be judged by itself and in accordance with the effect we wish to produce. In general we may say:

1. We correct the total amount of *astigmatism* found under a cycloplegic. The only exception is when the astigmatism is very high (5 D, or over), in which case a partial correction is occasionally less annoying to the patient than is a complete correction which gives only slightly better vision. With properly adjusted glasses, however, it will usually be found that even very strong cylinders will be worn with comfort and advantage. Slight degrees of astigmatism (0.25–0.50 D) may be left uncorrected if they do not appear to be giving trouble. They should be corrected (*a*) whenever a glass has to be used anyhow (as in presbyopia); (*b*) in most cases also when the patient has to use his eyes excessively for near work; (*c*) when there are well-marked symptoms of eye strain, such as headache, asthenopia, and obstinate blepharitis or conjunctival irritation.

2. We correct the total amount of *myopia* found under a cycloplegic. The main exception will be when the myopia is excessive and the patient has not worn very strong glasses hitherto, as in this case the sudden change may cause discomfort. Moreover, if the patient is under the presbyopic age, we try, as far as can be, to make him use the same glass for distance and near (see page 876).

3. We under-correct the total *hypermetropia* by an amount which depends upon—(*a*) The age of the patient. The younger he is the more we leave for his accommodation to do. (*b*) The amount of manifest

[7] In a myope of 3 D or more the inconvenience, except for the dazzling produced by the mydriasis, is practically nil, as such a one, having a far point at thirteen inches or less, can still read when his accommodation is paralyzed.

hypermetropia. The less this is in proportion to the total hypermetropia, the more we usually have to under-correct the latter in our prescription. In general, we give a glass somewhere between the manifest and the total hypermetropia and as near the latter as the patient will accept on a post-cycloplegic test (page 882). (*c*) The patient's requirements. If he uses his eyes excessively for near work, we correct more of the hypermetropia than if he is leading an out-of-door life. (*d*) The symptoms. In the presence of marked asthenopia, headache apparently due to eye strain, neurasthenia, and general muscular weakness (particularly the accommodative weakness after exhausting diseases), and especially in the presence of a tendency to excessive convergence, we correct more and more or even the whole of the hypermetropia. When there is an actual tendency to convergent squint or when there is a spasm of accommodation, we correct the whole of the hypermetropia and insist upon the continuous use of the glasses combined if need be with the use of atropine (see pages 791, and 910). On the other hand, if well-marked exophoria is present, we may under-correct the hypermetropia somewhat, so as to stimulate the accommodation and with it the convergence. Generally, however, the more completely a hypermetropia is corrected, the better. Many hyperopes, indeed, do not secure relief from their symptoms until a full correction is applied. As a rule, children accept a full correction very readily, and adults generally can be got to do so too, if they are willing to put up with some temporary blurring of sight. But with many patients, particularly those of middle age, the blurring produced by a full correction often causes great annoyance and even asthenopia; and much relief is experienced when the correction is reduced by even 0.25 D. It must be remembered that a convex glass may seem too strong and a concave glass too weak because too far from the eyes (see page 832). This is one reason why toric glasses and automobile goggles sometimes cause blurring and have to be reduced in strength.

As a general thing, unless he is presbyopic or his accommodation is otherwise subnormal, a hyperope, if he wears a glass at all, should wear the same one for distance and near.

[For the determination of *reading glasses* see page 863.—D.]

794. Anisometropia.[8]—By anisometropia is meant a difference in the refraction of the two eyes. One eye may be emmetropic and the other myopic, hypermetropic, or astigmatic, or both eyes may be ametropic, but in a different way. In this regard all possible combinations occur.

Anisometropia not infrequently is congenital, and then, at least in the higher degrees of it, often manifests itself even upon external inspection by an asymmetrical formation of the face and of the skull. Acquired anisometropia most frequently originates from the circumstance that the change taking place in the refraction during life—that is, the decrease in the hypermetropia or the development of a myopia—does not advance

[8] From ἀ, privative, ἴσος, equal, and μέτρον, measure, and ὤψ, sight.

at the same pace in both eyes. [The right eye usually has the higher refraction.—D.] Very high degrees of anisometropia develop when one eye is normal, but the other, in consequence of a cataract operation, has become very hypermetropic.

A correction of anisometropia without the aid of glasses would be conceivable only as the result of an effort of the accommodation differing in the two eyes; but this the eyes are incapable of doing, at least to any noteworthy extent. Accordingly, the anisometrope never sees distinctly with both eyes at once. This, however, gives him so little inconvenience that many persons do not become aware of the fact that they are not seeing equally well with both eyes until the tests of vision which the physician institutes are made. Moreover, if the difference in the refraction is not too great, binocular vision is not disturbed by it. Both images, even though they are of unequal distinctness, are superimposed and made to coalesce. In the high degrees of anisometropia, however, strabismus very frequently sets in. This may be either divergent or convergent, and is often alternating, particularly when one eye is hypermetropic, the other myopic (see page 762). [Anisometropia, particularly when of high degree is rather frequently associated with hyperphoria.—D.]

The obvious course to pursue would seem to be to correct the anisometropia by ordering the correcting glasses for the two eyes. And in slight degrees of anisometropia the unequal glasses are well borne. But if the difference between the two glasses is somewhat great (more than 1.0—1.5 D), the patients complain of an unpleasant sensation in the eyes, of vertigo, headache, etc., and when they try to use their glasses cannot get accustomed to them.[9] We are then obliged to refrain from correcting the anisometropia fully, and give glasses the difference between which is less than the amount of the anisometropia or actually give the same glass for the two eyes. Sometimes, again, the best plan is to correct only one eye and place a plane glass before the other. In doing this we always have regard to the better eye—namely, the one which appears more efficient for the purpose in view (distant or near vision). [In the large majority of cases, the patient will readily tolerate and find satisfaction in glasses fully correcting each eye, even when the difference in refraction is very great. There may be a period of temporary discomfort but even this is absent in many cases, and the final outcome is more satisfactory than when an incomplete correction is used. Moreover, by the use of this full correction combined with exercise of the poorer-sighted eye, the vision of the latter may be materially improved (see pages 633, 792). In anisometropia it is particularly important that the glasses should be so centred as to produce the minimum of prismatic deviation (cf. page 839).—D.]

[9] [In many cases the obstacle that opposes our attempts at correction of both eyes is a muscular error. This produces diplopia, which, as long as the image of one eye is indistinct, is not obtrusive and hence can be neglected, but which becomes annoying as soon as both images are made clear by the use of correcting glasses. In a few cases the trouble lies in the unequal prismatic effect of the unequally strong glasses. This causes diplopia when the patient looks through the periphery of the glasses and can generally be obviated by careful adjustment.—D.]

CHAPTER VI

ANOMALIES OF ACCOMMODATION

[**795. Varieties of Accommodative Anomalies.**—A necessary preliminary to the consideration of the anomalies of accommodation is a knowledge of the limits of the normal accommodation. These are shown in Fig. 377 and the table on page 860. From the latter it can, for example, be deduced that at 40 the average man has a range of 5.8 D, while in others who still can be regarded as normal the range varies between 3.9 and 7.1 D and in extreme cases may reach 7.8 D. A range lying persistently outside of the limits given must be regarded as abnormal. The accommodation in this case may be either too low (*insufficiency* and *paralysis* of accommodation) or too high (*excess* and *spasm* of accommodation). It may also be unduly *inert*; and it may be *unequal* in the two eyes.

When measuring the accommodation in order to ascertain whether these anomalies are present, we must first provide the patient with the full correction of his refraction as found under a cycloplegic. Otherwise we might, for example, be unable to tell whether a remote near point meant weakness of accommodation or latent hyperopia (cf. page 887). Neglect of this precaution doubtless occasioned some errors in the past, e. g., in the presbyopic curve as originally plotted by Donders.—D.]

[**796. Insufficiency of Accommodation.**—(*a*) *Ill-sustained Accommodation.* In many cases the range of accommodation is normal for the age, i. e., above the minimum shown in Fig. 377, but soon gives out so that it drops below the normal when the patient tries to use his eyes. This occurs in conditions of weakness in general (convalescence from disease, etc.) and in the other conditions noted below as the causes of accommodative insufficiency—of which, indeed, this ill-sustained accommodation is often but an initial or a terminal stage.

(*b*) In true *accommodative insufficiency*, the patient's accommodative power remains persistently below the normal limit for his age.[1] It is a fairly frequent condition.

It may be *caused* either by undue rigidity (accelerated sclerosis) of the lens or by weakness of the ciliary muscle. In the former type, which may properly be called premature presbyopia, the accommodation drops in much the normal fashion from year to year, but in any one year is always below the normal standard.

[1] [Obviously, also, a patient must be held to have at least a *relative* insufficiency of accommodation if his accommodative power, although above the minimum normal limit for his age, is persistently below his own lower normal limit, as shown by later tests.—D.]

In the type due to anomalies of ciliary action, the insufficiency varies from time to time, often in a quite haphazard manner.

The causes producing simple premature presbyopia are unknown. The causes of ciliary weakness are as follows:

1. Toxic conditions due to infectious disorders (intestinal toxæmia, tuberculosis, influenza, whooping-cough, measles, and tonsillar and probably dental infection).
2. Nasal obstruction. This appears to be a frequent cause.
3. Disorders of the internal secretions.
4. Neurasthenia and anæmia, brought on by overwork, impaired nutrition, and similar causes.
5. Vascular hypertension:
6. Probably excessive action of light.
7. Glaucoma in the prodromal stage.

In nearly half the cases, especially in those due to nasal obstruction and neurasthenia, accommodative insufficiency is associated with convergence-insufficiency and frequently with retinal hyperæmia and persistent dense injection of the conjunctiva.

The *symptoms*, which in part are caused by the associated conditions, are asthenopia—either simple tiring of the eyes or tiring associated with eyeache (sometimes of great severity), with marked irritation and burning of the eyes or with headache; blurred vision, especially for near work; vertigo; aprosexia; photophobia (quite a marked symptom in a number of cases). An occasional finding, especially in the cases with marked convergence-insufficiency, is a concentric contraction of the visual field.

The intensity of the symptoms varies greatly. In many cases they are such as to render eye work almost impossible. On the other hand, especially in simple premature presbyopia, the only complaint may be of impaired vision for near, which is remedied at once by suitable reading glasses.

The *course* and *duration* naturally depend on the cause. Recurrences may take place, sometimes at long intervals. In simple premature presbyopia the course goes on much as in an ordinary presbyopia, but the necessity for the use of an additional glass for reading begins much earlier —at forty or before.

The *treatment* is directed to the cause, whenever we can discover the latter. The removal of toxic conditions by remedying an intestinal indigestion or a dental or tonsillar infection; the relief of anæmia and malnutrition by the use of tonics and proper diet; the regulation of vascular anomalies; the removal of nasal obstructions; treatment addressed to abnormal glandular conditions—all these are to be employed when required. Eye-strain should be relieved by proper glasses. An associated conjunctival injection should be treated with zinc or other astringents.

For the insufficiency itself the following means are used:

1. For the blurred vision at near, the distance glass should be supplemented by a proper addition for reading (presbyopic correction for an unnatural presbyopia).

2. Even when the vision is not much blurred, this presbyopic addition may be necessary, especially if there is an associated convergence-excess.

3. The accommodation may be stimulated directly by means of reading exercises, or exercise in focusing on the fine test-object (line) used in measuring the accommodation (Fig. 375).

4. Especially in cases associated with convergence-insufficiency it is useful to stimulate both accommodation and convergence by exercise with prisms, bases out, when the eyes are directed at a near object. This is supplemented by practice in converging both eyes on a dot which is brought closer and closer to the eyes until it doubles.—D.]

[**797. Paralysis of Accommodation.**[2]—Paralysis of accommodation is simply an insufficiency carried to an extreme degree. The term, however, is particularly applied to the cases due to the more serious toxic and organic causes. It produces an extreme reduction of the accommodation below the normal (see Fig. 377), as shown by tests made with reading or with the accommodation disk (Fig. 375).—D.]

The disturbance which paralysis of the accommodation causes varies greatly according to the refractive condition of the eyes. If an emmetrope is affected with paralysis of the accommodation, reading and writing become perfectly impossible, or at least, in case of incomplete paralysis (paresis) of accommodation, very difficult and possible for only a few moments at a time. Distant vision, for which the emmetrope does not require to use the accommodation, is not affected. In the hypermetrope paralysis of accommodation makes itself still more noticeable, since without accommodation he sees poorly even at the distance. The reverse is true of the myope, to whom the abolition of accommodation causes little or no inconvenience; indeed, in the higher degrees of myopia a paralysis of accommodation is often discovered only accidentally at the time when a careful examination is being made. And in old persons in whom accommodation for near has become impossible owing to sclerosis of the lens, paralysis of accommodation would also necessarily remain undetected—in fact, we could never succeed in demonstrating it. [Yet until the patient has reached the age when the minimum normal accommodation is less than 1.5 D—i. e. until he is 48—paralysis of the accommodation can be made out by the tests, and at 40 and 45 is almost as apparent as in youth (see page 862).—D.]

In paralysis of the accommodation, whether produced by disease or artificially by a cycloplegic, the statement is frequently made that objects appear smaller then usual (*micropsia*).[3] This phenomenon is explained in the following way: We estimate

[2] [Also called *cycloplegia*, from κύκλος circle, and πληγή, stroke. Hence *cycloplegic*, an agent—e. g., atropine—paralyzing the accommodation.—D.]

[3] [From μικρός, small, and ὤψ, sight.—D.]

the size of an object from the size of its image upon the retina taken in connection with the distance at which we judge the object to be situated. An object of certain size seen at a certain distance gives us a retinal image of certain size. If the object is approximated to one-half the distance, its retinal image becomes twice as great. If this were not the case, and the retinal image remained of the same size when the object was approximated, we would infer that the object itself had been reduced to one-half its former size. It is this mistake that we fall into in case of paralysis of the accommodation. Since in this condition the act of accommodation for any given distance of the object costs us a greater effort than under other circumstances, we estimate the accommodation at too high a figure, and hence believe the object to be nearer than it really is; but as the retinal image is no larger, we think that the object itself has diminished in size. The same phenomenon manifests itself when an emmetrope looks through concave glasses [held close to the eyes]; these make objects look smaller to him: for, to overcome the concave glasses, he must strain his accommodation. Now, without being distinctly aware of this strain, he yet infers from it that objects are nearer than they are, and thus the latter, since their retinal images are not any larger, seem smaller to him. The converse phenomenon, by virtue of which objects appear larger than normal—*macropsia*[4]—is observed in spasm of the accommodation. This, too, originates in a delusion as to distances, resulting from the disturbance of the accommodation. [For retinal micropsia and macropsia, see page 126—D.]

Paralysis of the accommodation arises from a paralysis of the ciliary muscle or of the oculo-motor nerve, which supplies this muscle. It may be simply one of the symptoms of a complete oculo-motor paralysis, in which cases its etiology agrees with that of oculo-motor paralysis in general (see page 756). But in many cases the paralysis of accommodation exists alone, or is at most associated with a coincident paralysis of the sphincter pupillæ. These two intrinsic muscles, which under physiological conditions act in conjunction, are also commonly paralyzed together, so that the paralysis of accommodation is combined with mydriasis paralytica (ophthalmoplegia interna [or interior]). The *causes* of paralysis of accommodation (with or without paralysis of the pupil) with which we are acquainted are as follows:

1. *Diphtheria*.—Paralysis of accommodation belongs among the post-diphtherial paralyses—i. e., those which usually develop in the stage of convalescence. The most common of these, besides the paralysis of accommodation, is paralysis of the soft palate, which manifests itself in the nasal character of the speech and also by the fact that food and liquids readily get from the choanæ into the nose when the patient tries to swallow. Isolated paralysis of the sphincter pupillæ or paralysis of the exterior eye muscles, of the muscles of the extremities, or of the trunk itself, occurs less often. Diphtherial paralysis of the accommodation [affects both eyes, and] is usually not associated with paralysis of the sphincter pupillæ. It generally passes away of itself in one or two months as the patient gains in strength, and hence affords a good prognosis. Cases of paralysis of accommodation consequent upon *influenza*, which were

[4] [From μακρός long, and ὤψ, sight.—D.]

analogous in their behavior to post-diphtherial paralyses, occur pretty frequently during some epidemics. [Paralysis of accommodation may also be caused by bacterial infection springing from the *tonsils* or other parts (Veasey, Johnston).—D.]

2. *Poisoning.*—The most complete paralysis of accommodation, combined with paralysis of the pupil, is produced by atropine and the other cycloplegics. These act not only when administered internally, but also locally when introduced into the conjunctival sac. The cases in which paralysis of the accommodation, together with symptoms of general posioning, have been observed after eating spoiled meat, sausages, fish, etc., likewise depend upon poisoning, which in this case is poisoning by *toxins*. As regards post-diphtherial paralyses also it is assumed that they are caused by a diphtheria toxin, and a toxic origin perhaps holds for the paralysis of accommodation that occurs in consequence of *diabetes*. In all cases in which the paralysis depends on internal poisoning, it is bilateral. [The paralysis that occasionally occurs in disorders of metabolism is presumably toxic.—D.]

3. Brain *syphilis* and tabes and progressive paresis, which are connected with syphilis. In these cases the paralysis of accommodation is frequently unilateral, is usually associated with paralysis of the pupil [and generally resists all treatment. It may also occur with or without paralysis of the pupil in non-syphilitic and in functional brain disease. Paralysis of the accommodation, due to brain disease, may be associated with paralysis of convergence (see page 784).—D.]

4. In *contusions of the eyeball*, being then a purely local affection.

It is evident that the accommodation is completely abolished when the lens is luxated, or is removed altogether from the eye, but such cases are not properly denoted as paralysis of the accommodation.

The *treatment* of paralysis of the accommodation must first of all be regulated according to the lesion which lies at the bottom of it, and must seek to effect the cure of this latter by appropriate means. In post-diphtherial paralysis we institute corroborative measures, giving hearty nourishment, wine, iron, quinine, etc. For local treatment, pilocarpine is employed. This, besides contracting the pupil, also produces a spasm of the accommodation by contracting the ciliary muscle. This, however, is not of long duration, any more than the miosis is; after some hours the muscle relaxes again and the paralysis returns. Nevertheless, the contraction of the muscle produced by the miotic appears sometimes to exert a favorable influence upon the paralysis itself, perhaps acting in the same way as faradization does when it produces a good effect in paralyses. Pilocarpine, however, has a really beneficial effect only in those paralyses which in themselves give a good prognosis, i.e., in the post-diphtherial paralyses and those dependent on poisoning. In other paralyses mercury and potassium iodide are more in order [but rarely do good]. If the paralysis presumably will last a long time the indication is to render work possible by means of proper convex glasses.

[798. **Accommodative Inertia.**—Accommodative inertia is the condition in which the patient changes from one accommodative state to another sluggishly or with difficulty. It is analogous to the slow adaptation that some eyes show in passing from

light to darkness, or vice versa. It seems sometimes to be a source of real trouble. In such cases exercise of the accommodation and particularly of the convergence may be tried.—D.]

[799. **Excessive Accommodation.**—Excessive accommodation may be due to ciliary overaction (*spasm of accommodation*). Apart from what is called the normal tone of the ciliary muscle, which is abolished by atropine and which at most equals 0.25 D, and apart from the continuous tension of the ciliary muscles in hypermetropes when overcoming their hypermetropia so as to see distinctly] a state of permanent excessive tension of the ciliary muscle does occur, which is directly prejudicial to vision since it mainly affects myopic eyes, which by it are made to appear more myopic than they really are. Such spasm of accommodation may be revealed by our finding the myopia higher when we make the subjective test (with glasses) than when we determine it objectively with the ophthalmoscope [or shadow test]. For during the examination with the ophthalmoscope it is usual for the accommodation to relax [partially or] entirely, so that the eye shows [more nearly] its true refraction. The confirmatory test is made by instilling atropine which paralyzes the accommodation and thus abolishes the spasm so that now on making the tests with glasses the true refraction is found. Genuine spasm of accommodation is rare and occurs only in young persons,[5] and the degree of tension put on the accommodation is usually pretty considerable. Formerly the frequency of accommodative spasm as well as its significance for the development of myopia was much overrated.

Spasm of accommodation is combated by the instillation of atropine,[6] which must be kept up for quite a long time (four weeks and more). Unfortunately, in most cases when the atropin has been discontinued, the spasm returns after a shorter or longer interval.

High degrees of spasm of accommodation, sometimes combined with spasm of convergence (see page 781), are found in hysterical subjects.

An artificial spasm of accommodation of high degree combined with contraction of the pupil develops after the instillation of a miotic (eserine and pilocarpine).

[*Lenticular accommodative excess* is the condition in which the accommodation is above the usual limit, because the lens is less rigid than ordinary. It is the opposite of premature presbyopia, and may be called delayed presbyopia.—D.]

[800. **Unequal Accommodation.**—We sometimes find the accommodation unequal in the two eyes. Sometimes this is due to the fact that the ciliary muscle is weaker in one eye than in the other. This occurs quite frequently in ophthalmoplegia interior due to syphilis, tabes, traumatism, or poisons. It can be readily understood that these conditions may affect one eye more than the other, or may affect one to the exclusion of the other. Since in these cases the sphincter iridis and the ciliary muscle are usually affected simultaneously, the inequality in accommodation is generally associated with inequality of the pupils—the eye with weaker ciliary muscle having the larger pupil.

When not due to the causes above mentioned, unequal accommodation is usually not associated with inequality of the pupils. In this case, too, it may be caused by unequal action of the ciliary muscle, but most of the cases of this sort seem to be due to an unequal rigidity of he lenses in the two eyes. In other words, it is a form of presbyopia in which the sclerosis of the lens has advanced faster in one eye than in the other. It produces no symptoms until the accommodation in one eye or the other has sunk so low as to require the addition of a glass for near work. When this occurs, it is important to take account of the inequality and to determine by experiment whether we shall or shall not make an unequal addition for reading to the distance correction.

In some cases such unequal addition is quite helpful (see page 863).—D.]

[5] [Some of the most marked and obstinate cases of spasm of accommodation I have seen have been in persons of forty-five and even forty-eight years of age.—D.]

[6] [And by precise correction of the refraction, determined under complete atropinization.—D.]

PART V
OPERATIONS

OPERATIONS

CHAPTER I

EYE OPERATIONS IN GENERAL

801. Antisepsis.—The *antiseptic method*, which represents the greatest progress made in surgery during recent times, has also produced an essential improvement and greater certainty as to results in the special domain of operations upon the eye. It is therefore the first duty of every operator upon the eye to proceed in a perfectly aseptic and antiseptic manner. In operations upon the eye we have less to do with antisepsis than with asepsis; we do not have to disinfect a contaminated wound, but to make a wound that is clean and keep it from contamination.

In former times much more importance was attached than now to the shape and position of the section, especially in cataract operations, the hope of a happy result being based solely upon the proper performance of the section. Starting with this view, observers devised a great number of different methods of operating which have already, in part at least, fallen into oblivion. At the present time we know that the rigorous carrying out of asepsis and antisepsis in the operation and after-treatment is of much greater significance than the choice of a method of operating. Any section that is of the necessary size and is suitable in position gives good results, if in other respects we proceed with the most scrupulous cleanliness. In eye operations this is doubly important, since the result that we seek is attained only if healing by first intention is secured. When an amputation wound does not heal by first intention, but by suppuration, this accident usually does the patient no harm beyond prolonging his stay in bed; but if suppuration ensues instead of primary union after an iridectomy or cataract operation, the eye is lost, which for an operator upon the eye is the same thing as the death of a patient would be for the surgeon.

802. Preparation for the Operation.—Contamination of the wound may either be effected by means of the operator and his instruments or it may take its origin from the eye itself or its vicinity. To avoid the former, the hands of the operator must be well cleansed and then disinfected with a solution of corrosive sublimate (1:2,000) or mercury oxycyanide (1:1,000). As the operator has to talk to the patient, it is advisable for him to wear a wire frame covered with sterilized gauze in front of his mouth to avoid infection by drops falling from the mouth. The delicate instruments which are used for operating upon the eyeball itself are disinfected by boiling in a 1-per-cent solution of sodium carbonate, in which they do not rust. To prevent infection of the wound by the adjacent parts (lids and conjunctival sac), the vicinity of the eye, and especially the skin of the lids and the edges of the lids, should first be wiped off with a little benzine in order to remove the grease from the skin, then thoroughly washed with soap, and finally bathed with the above-mentioned sublimate or oxycyanide solution. For washing out the conjunctival sac we use a solution of one of the mercury salts, of half the strength above given, or

a physiological (0.6 per cent) salt solution which has been sterilized by boiling. The conjunctival sac, however, affords special danger of infection only when decomposed secretion is present in it as a result of disease of the conjunctiva or lachrymal sac. Hence, before every operation we ought to examine carefully the conjunctiva, and more especially the lachrymal sac, and before proceeding to perform the operation we ought to try first to relieve by appropriate treatment any lesion of these structures that may be present. Those operations in which the eyeball is opened require special care as regards asepsis. In such cases, therefore, if the conjunctival sac is not clean, its secretion should be examined for any germs that it may contain, and the operation is to be performed only after the pathogenic bacteria (especially pneumococci and streptococci) have disappeared from the secretion.

So far as concerns the special case of chronic dacryocystitis, its complete cure, unfortunately, requires a very long time. Hence, in order to accomplish my purpose more quickly, I generally extirpate the lachrymal sac one or two weeks before the operation. If, however, there is no abnormal secretion in the lachrymal sac, I content myself with splitting the anterior wall of the sac directly before the operation, and after suitable cleansing, fill it with iodoform powder or seal the canaliculi by searing them with the galvano-cautery.[1] [The same precautions should be used if the nose has any condition (ozæna, ethmoiditis) likely to give rise to infection (Lancaster).—D.]

[To make sure whether the eye is fit for operation many take smears from the conjunctival sac, in every case, and if dangerous germs are found postpone operation until the conjunctiva is sterile. To render it sterile, Elschnig applies mercury oxycyanide every half hour, and if this does not suffice applies one-per-cent nitrate of silver once a day. Others apply a bandage before operating, and if this causes secretion to develop in the eye, postpone operation until the conjunctiva is in healthier condition.—D.]

In *cleansing the eye* before the operation, we may employ quite strong antiseptic solutions for the parts surrounding the eye. For the conjunctiva the stronger antiseptic solutions are contraindicated; obviously so, because they produce a marked irritation, or actually a traumatic conjunctivitis associated with considerable secretion. But if we employ the antiseptics in a degree of concentration that is well borne by the eye, they have, owing to the brief period of their action, no bactericidal effect. All investigators have arrived at the same result, namely, that by these agents we can simply diminish the number of the germs present, but cannot annihilate them completely. We get the same result by employing indifferent sterile liquids, particularly if at the same time we remove the adherent mucus from the surface of the conjunctiva [and cornea] mechanically—i. e., by wiping it with moistened pledgets of cotton. Hence, if the conjunctiva is normal, I use simply a physiological salt solution which has been sterilized by boiling, and employ a sublimate solution (1:4000) only in those cases in which the conjunctiva is diseased (catarrh, trachoma).

[More thoroughgoing in its antisepsis is the procedure of Herbert and Elliot. Fifteen minutes before operating they irrigate the everted lids with a 1:3000 mercury b chloride solution delivered from a container 3 feet above the patient's head, and at the same time move the lids vertically, so that the sclution shall reach all the folds of the conjunctiva. This irrigation is done for a minute at least and may be repeated if

[1] [Some advocate passing a temporary ligature round each canaliculus just before the operation (Buller, Quackenboss). See page 934.—D.]

the conjunctiva does not give a satisfactory reaction (secretion of grayish mucus, cloudiness of membrane). Then epinephrine (adrenaline) and cocaine are instilled, and the eye is irrigated with normal salt solution. In addition Elliot wipes off the conjunctiva with sterile cotton. He and other operators also trim off the lashes, because it is practically impossible to sterilize these, and they may come into contact with the instruments during the operation. It cannot be denied that, so far as the prevention of infection is concerned, the method of Herbert and Elliot greatly surpasses the less energetic procedures. In a series of several thousand cases thus operated on not one case of suppuration and very few cases of non-suppurative inflammation occurred (Lancaster).—D.]

As the conjunctiva even after careful cleansing often still contains germs, we might think that the infection of recent wounds would necessarily be very frequent. Fortunately, however, at the present time infection of wounds is only of exceptional occurrence, so that evidently the germs of the conjunctival sac are not to be greatly dreaded. [Cf. page 31.]

Any disease of the lachrymal sac, on the other hand, is very dangerous, and many a cataract operation has gone wrong because such a disease has been overlooked. Hence, before every cataract operation or iridectomy, we ought to examine the tear passages carefully, and if there is anything in the least suspicious, test their perviousness by passing liquid through them (page 930).

The infection of wounds, furthermore, as experiment also has proved, often occurs from contaminated instruments; and it is to the sterilization of these that our attention ought, above all, to be directed.

803. Anæsthesia.—Anæsthesia for operations upon the eyeball is effected by cocaine, a 5-per-cent solution of which is instilled several times into the conjunctival sac at intervals of a few minutes. [For some purposes holocaine, novocaine, and alypine are preferred (see pages 63, 64).—D.] The solution should be freshly prepared and sterilized by boiling. After instilling it we must take care that the patient keeps the eye shut, because the frequency of winking is diminished as a result of the cocaine anæsthesia, and hence the cornea, if uncovered, is apt to become dry upon its surface. [Dryness of the cornea may be prevented and the surface of the eye kept from contamination by dust, etc., if, as is often done in this country, the eye is irrigated from time to time, before and during the operation, with a sterile boric acid or salt solution.—D.] Cocaine anæsthesia lasts about ten minutes. It affects only the superficial parts, like the cornea and conjunctiva, while the iris remains sensitive. In iridectomy, for example, the grasping of the eyeball and the incision are not felt, but the excision of the iris is painful.[2] In operations upon the lids, several drops of the cocaine solution may be injected beneath the skin of the lids, in operations on the eye muscles [and in iridectomy for acute glaucoma] beneath the conjunctiva. [Such subconjunctival injections, however, are sometimes disadvantageous in muscle operations because they may puff up the tissues enough to obscure the relations of the parts.—D.] For enucleation cocaine is most effective when it is injected in the neighborhood of the ciliary ganglion. A long cannula is

[2] [If, however, the performance of the operation is delayed long enough (ten or fifteen minutes) after the first instillation of cocaine, for the drugs to pass through the cornea, the iris also may often be completely anæsthetized, and iridectomy is then performed absolutely without pain.—D.]

plunged 4.5 cm. in, at the temporal side of the optic nerve and nearly to the apex of the orbit. [Guttman showed that by infiltration-anæsthesia with very weak solutions of cocaine injected into the retrotarsal folds the operations on the tarsus, e. g., the expression of trachoma granules, can be rendered painless.—D.] For injection into the tissues, in order not to

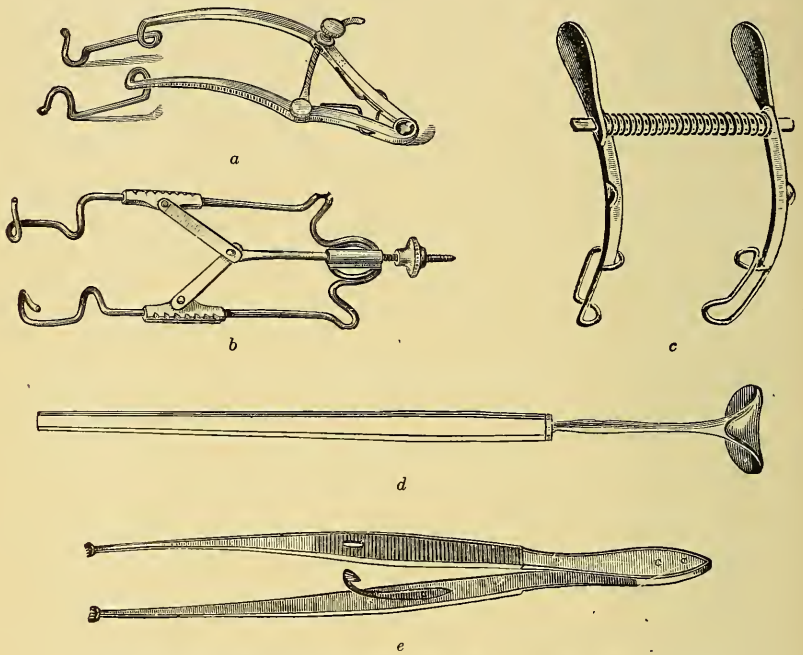


Fig. 400.—Instruments for opening the Eye and keeping it in Place.

a, *b*, *c*, various forms of spring specula for holding the eye open in operations. *d*, Desmarre's elevator or lid retractor, for holding the lids apart in examinations of the eye and in operations. *e*, fixation forceps for holding the eyeball steady in operations.—D.]

produce poisoning, we use a weaker—i. e., a 1-per-cent—solution, to which with advantage may be added some adrenaline (two to three drops of the commercial 1:1,000 solution [to each c.c of the cocaine solution).[3] Adrenaline is also used if we have to operate on highly injected eyes. If these are previously rendered pale by adrenaline, the cocaine acts better. [Adrenaline, moreover, is a useful adjunct to cocaine in muscle operations, since here it checks the bleeding which in the tiny field of operation interferes greatly with the view. On the other hand, it is better not to use it in operating on a pterygium or other vascular growths, which may be so contracted by it that the normal outlines are obscured.—D.] In case of very great sensitiveness of the eyeball (severe irido-cyclitis, acute glaucoma) cocaine even with the assistance of adrenaline cannot make the

[3][Cocaine or alypine, preferably combined with adrenaline, may be introduced into the tissues by cataphoresis (see page 57) especially to produce anæsthesia in operations on the lid and lachrymal sac.—D.]

eye insensitive enough for security in the performance of the operation. In that case general narcosis with chloroform or ether is requisite, and it is likewise usually employed in major operations such as enucleation, etc., and for children.

804. Fixation of Eye.—In regard to *operations upon the eyeball itself* the following principles hold good:

The separation of the lids is effected by means of lid specula (blepharostats, elevators, or écarteurs). There are some which hold both lids open at once, keeping them apart by the elastic force of a spring (spring specula; Fig. 400, *a*, *b*, *c*); others which are designed for one lid only and must be held with the hand (Desmarre's lid retractor, Fig. 400, *d*). The eyeball itself is fixed by grasping a fold of conjunctiva close to the corneal margin with a toothed forceps (Waldau's fixation forceps, Fig. 400, *e*) and holding it in place. [Angelucci followed by many operators fixes and controls the eyeball by firmly grasping the tendon of the superior rectus with the fixation forceps. Elschnig, Maddox, and others pass a stitch through the tendon of the superior rectus, and control the eye with this.—D.]

Since the metal arms of the speculum exert a pressure on the lids that is unpleasant to the patient, we may, if we have a skilled assistant, get him to hold the lids apart with his fingers. This is advisable most particularly in cases in which success depends on our not exerting any pressure on the eyeball (e. g., in order to avoid escape of vitreous), and we then too abstain as far as possible from grasping the eyeball with the fixation forceps.

805. Incisions.—The incision which lays the eyeball open is made, as a rule, within the limits of the anterior chamber. As this latter is bounded by the cornea and at its periphery by the most anterior portion of the sclera, the section may lie either in the cornea or in the sclera. We therefore distinguish sections with respect to—

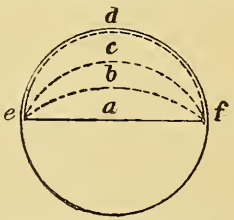


Fig. 401.—Different Forms of Sections in the Cornea.

(*a*) Their *position*, into corneal and scleral. These differ from each other mainly in the following points: 1. In scleral incisions there is more tendency to prolapse of the iris than in incisions of the cornea (for explanation of this see page 922). 2. The sclera is covered by conjunctiva, and a wound in the sclera can therefore be provided with a conjunctival flap—a thing which is not [usually] possible in corneal sections. 3. Scleral wounds are less apt to become infected than those in the cornea, because of the slighter tendency that the sclera exhibits toward purulent inflammations. Hence, before the introduction of antiseptic methods, scleral incisions gave better results than did those in the cornea. At present, when infection is avoided in every possible way, this distinction is no longer of so very much weight.

(*b*) In *shape*, incisions may be linear or curved. The former lie in a great circle of a sphere, and hence form upon the surface of the eyeball a line which is the shortest that can be made to connect the terminal points of the section (*eaf*, Fig. 401). The curved or flap incisions correspond to a small circle of a sphere. Between the largest curved section (*edf*, Fig.401) and the linear section an infinite number of sections (*ecf*, *ebf*) may be conceived to exist, constituting the transition forms between the two. h se are curved sections of varying altitude; the linear section forms their inferior limit, and may therefore be regarded as a curved section whose altitude =0. Most of the sections in general use are curved ones, with an arch of greater or less altitude. An example of a pure linear section would be the one devised by Saemisch for laying open an ulcus serpens, in which the latter is divided with a Graefe knife from behind forward (page 968). A flap incision, having the ends of its section at the same distance apart as a linear incision, will make a much longer wound than the latter, and, by the lifting up of the flap, the wound can be made to gape more.

In *making* the section, care must be taken that the knife is withdrawn from the wound slowly, so that the aqueous may escape as gradually as possible. In this way we avoid the evil results which too rapid escape of the aqueous often entails, such as extensive prolapse of the iris, subluxation of the lens, prolapse of the vitreous, and intra-ocular hæmorrhage. Slow escape of the aqueous is particularly important if an operation is done when there is increase of tension.

In judging of the size and position of any particular form of section, not only the *outer and visible wound* but also the *internal* one must be taken into consideration. That the latter is of a different size, shape, and position from the external wound arises from the fact that in most methods of performing the section the knife divides the tunics of the eye obliquely (Fig. 408, *I* and *P*). This is particularly the case with the wounds made by the lance-shaped knife. Even if at first we plunge the lance knife in perpendicularly, we must still, as soon as its point has entered the anterior chamber, change its position so that it shall be pushed along parallel with the iris, as otherwise we should get into the iris and lens. The inner orifice of the section (Fig. 402, *i i*) therefore lies nearer the center of the cornea than does its outer orifice (Fig. 402, *a a*). For this reason sections whose outer orifice lies in the sclera, and which hence are usually regarded as scleral sections, nevertheless by their inner segment belong to the cornea (*I*, Fig. 408). A further fact contributing to this relation between the external and internal sections is that the sclera overlaps the cornea externally, and the latter the efore in ts inner layers extends farther toward the periphery than can be seen from the outside. Hence, even sections that are fairly perpendicular, like those made in performing cataract extraction with a Graefe knife (Fig. 411), are in great part located in corneal tissue. This fact that the internal wound is less peripherally situated than the external, must be taken into account in making the section. If, for example, we desire to excise the iris up to a certain spot, we must not perforate the cornea at a point directly opposite the latter, but must place the corneal section farther toward the periphery, so that the internal wound may lie at the spot at which the iris is to be cut off.

The internal wound is also of less length than the external (Fig. 402, *a a* and *i i*). This fact also must be taken into consideration, particularly in cataract operations, in which care must be taken that not only the external but also the internal wound shall be large enough to allow the cataract to pass through.

The *oblique course* of the wound through the tunics of the eye, furthermore, affects the tendency of the wound to gape. It was said above that flap wounds gape more than linear wounds; but the tendency toward gaping depends still more upon whether the wound traverses the tunics of the eye perpendicularly or obliquely. The former is more particularly the case in sections made with Graefe's knife, in which the knife passes through the cornea or sclera from within outward; the latter is the case in sections made with the lance knife. Sections of the former sort gape, owing to the elastic retraction of the edges of the wound. Wounds, on the other hand, which are made with the lance knife, and pass obliquely through the tunics of the eye, do not gape, because the lips of the wound close upon one another like a valve. The closure is effected by the intra-ocular pressure. This latter is exerted to the same extent on every point of the internal surface of the eyeball. It presses as strongly upon the posterior lip of the wound (*a*, Fig. 408) as upon the anterior (*b*), and hence pushes the former against the latter. To this valve-like closure of the wound is to be attributed the fact that the aqueous does not escape after paracentesis of the cornea if the lance knife is withdrawn from the wound cautiously, and without pressure or rotation. The wound must be made to gape before the aqueous (or, in simple linear extraction, the soft masses of lens matter) can be expelled. It would be a mistake to try to effect this by pressure either upon the center of the cornea or upon the sclera, as in so doing we should only increase the intra-ocular pressure by that which is exerted from the outside, and thus push the posterior lip of the wound still more strongly against the anterior. Only upon very strong pressure, by which the lips of the wound would be shoved past one another, would the wound gape open. The proper procedure, rather, is to depress the peripheral lip of the wound (*c*, Fig. 408) with a Daviel's scoop, and thus open the valve.

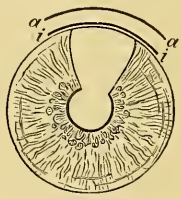


Fig. 402.—Iridectomy in a Case of Increase of Tension. Magnified 2×1. *a a*, external orifice of the wound situated in the sclera; *i i*, internal orifice situated at the sclero-corneal junction. In order to represent these relations a correctly performed iridectomy was made upon the eye of a cadaver, and the exact position of the orifices of the wound, properly magnified, was transferred to the drawing.

In small children quietude of behavior after the operation is not to be expected, and hence the large sections, such as those made for iridectomy or cataract extraction, have their healing interfered with. Accordingly, for small children we ought to choose only those methods of operating, such as discission, which produce very small wounds.

[In order to avoid postoperative infection many try to close in operation wounds with conjunctiva wherever they can. Thus in performing discission, whether of soft or membranous cataracts (see pages 987 and 990), they enter through the conjunctiva beyond the corneo-scleral margin and then through the sclera at the periphery of the anterior chamber, instead of going through the cornea directly (Byers). So, too, when doing a posterior sclerotomy (see page 1005), especially one for removing foreign bodies from the vitreous chamber, they fashion a quadrangular conjunctival flap in such a way that the cuts in the conjunctiva are distant from the incision to be made in the sclera. This flap is reflected, the scleral incision made, and the flap then replaced and sutured so as to cover the wound in the sclera. In a quite similar way a conjunctival flap is used by Kuhnt after keratectomy for partial staphyloma of the cornea (page 971), by Lagrange for covering in the wound in sclerectomy, by Elliot for covering in the artificial corneal fistula made by the trephine (see page 984), and by van Lint and others for covering in the corneal incision of a cataract extraction (see page 995).—D.]

806. Reposition and Prolapse of Iris.—In completing the operation the greatest attention must be paid to having *the iris in proper position.* Under no circumstances should the iris be left incarcerated in the wound [except in iridotasis]. In cases in which an iridectomy has not been made, incar-

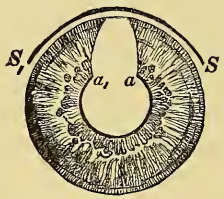


Fig. 403

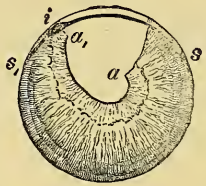


Fig. 404

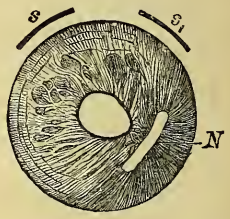


Fig. 405

Fig. 403.—Normal Position of the Iris. Magnified 2 × 1. The flap incision for the extraction, *S S*1, lies in the sclera. The angles of the sphincter, *a* and *a*1, are both low down.

Fig. 404.—Incarceration of the Iris in the Wound (after a Corneal Flap Extraction). Magnified 2 × 1. The flap incision for the extraction, *s s*1, lies in the cornea. The iris is visible as a dark nodule, *i*, in the wound, and the angle, *a*1, of the sphincter on the same side appears drawn up in comparison with that of the other side, which is in its proper position.

Fig. 405.—Incarceration of the Iris (after Linear Extraction). Magnified 2 × 1. The pupil is drawn toward the cicatrix, *N*.

ceration takes place because either the out-gushing aqueous or the lens that is pushed out by the pressure forces the iris into the wound. If an iridectomy is made, the iris, which is grasped by the forceps in front of the wound (Fig. 406, *aa*), is cut off flush with the latter. When this is done, of course

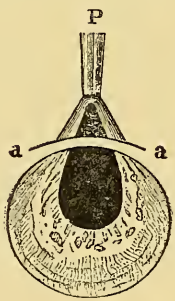


Fig. 406

Fig. 407

Fig. 406.—Position of the Iris During Iridectomy. Magnified 2 × 1. The forceps has grasped the iris near the pupillary margin and has drawn it out of the wound, *a a*. The portion of the iris lying outside of the eye is tent-shaped. The pupillary margin forms the two sides of the entrance to the tent, and between can be seen the posterior aspect of the iris—the retinal pigment of the latter—lining the inside of the tent.

Fig. 407.—Inclusion of Both Limbs of the Coloboma in the Scar. Magnified 2 × 1. The operation scar is solid and smooth and nowhere shows any included iris. The inclusion of the iris is recognized only from the drawing up of the pupil. The angles of the sphincter are not to be seen on either side, but the pupillary portion of the iris extends like a garland from one side to the other above, and the fibers of the ciliary portion are put greatly on the stretch in a vertical direction.

the part of the iris that lay in the tract of the wound at the moment of the excision is left behind in the wound. If during the excision some traction is made on the iris, the portion of the iris that still lies inside of the anterior chamber is put on the stretch. This tension ceases when the iris is excised,

so that the iris that is left behind can contract and thus the portion lying in the channel of the wound can retract into the anterior chamber. This retraction takes place particularly well if a contraction of the sphincter pupillæ aids it, although the sphincter has naturally lost much of its strength on account of the excision of a piece involving a solution of continuity. Very frequently, however, these agencies do not suffice to draw the iris back from the wound into the anterior chamber. If then the iris is not pushed back by the operator into the interior of the eye, it remains in the wound.

Incarceration of the iris in the wound is manifested by different signs, according to the extent to which the iris is protruded. If the iris has pushed its way through the wound to the outside, it becomes visible as a dark swelling or nodule either in the middle of the section, or, if some of the iris has been excised, at one or both ends of it (*i*, Fig. 404). The condition when the iris does not protrude from the wound, but is merely jammed in between the internal lips of the latter, is recognized by the displacement of the pupil. The latter in case no iris has been excised is pear-shaped with its apex directed toward the scar (Fig. 405) just as after perforating ulcers of the cornea with inclusion of the iris (Figs. 92 and 118). After operations of this sort, in which the iris has been excised, the boundary between the pupil and the coloboma is marked by two projecting angles, forming what are called the angles of the sphincter (Fig. 403). These represent the spot where the margin of the pupil passes into the lateral limits ("limbs") of the coloboma. When the iris is free, the angles of the sphincter are directly opposite one another, and, moreover, lie in the circular line which the margin of the pupil would form if still intact ("the angles of the sphincter are low down," Fig. 403). But if the iris is incarcerated in the wound, the corresponding limb of the coloboma is thereby shortened, and the angle of the sphincter looks as if drawn up ("the angle of the sphincter is higher," Fig. 404, a_1). The angle of the sphincter may be pushed up so far that it is not visible at all and if this is the case with both sphincter angles the entire pupil is markedly displaced toward the margin of the cornea (Fig. 407).

Inclusion of the iris in the wound is accompanied by evil consequences of many kinds. The healing of the wound is interfered with by inflammatory irritation, and is protracted. The cicatrix is less solid and regular in its formation, and may become ectatic, and later on the inclusion of the iris may give rise to increase of tension, to inflammation, and even to sympathetic disease of the other eye. To avert these results every attempt must be made, after completing the operation, to release the iris from its condition of incarceration, and to put it in the proper position. This is done by entering the wound with a spatula, and stroking the iris with it back into the anterior chamber. Should this attempt be unsuccessful, or should the iris after replacement again prolapse into the wound, the incarcerated portion of the iris must be pulled out, grasped and excised.

The firm closure of the wounds made with a lance knife diminishes the danger of prolapse of the iris. How, then, does *prolapse of the iris* occur at all? When the cornea has been perforated at any spot the aqueous flows from every direction toward this point, because here the ocular tension has sunk to nothing (i. e., has become equal to that of the external air). The fluid of the anterior chamber can flow toward the opening without obstruction; but the fluid of the posterior chamber, to get at the opening, must first pass through the pupil. Let us assume that the opening lies at the nasal margin of the cornea (*I*, Fig. 408). In this case the liquid from the temporal portion of the posterior chamber would flow toward the opening directly through the pupil, because this is its shortest way. The case is otherwise with the nasal portion of the chamber, which lies immediately opposite the opening. Here going through the pupil means taking a roundabout way, which is the more circuitous the farther the opening lies toward the periphery. The aqueous will therefore tend to take the shortest way and rush straight forward toward the opening, pushing the iris before it. In the language of physics, the posterior surface of the iris is subjected to the pressure of that portion of the aqueous which has not yet escaped (*d*, Fig. 408). On the other hand, the pressure upon the anterior surface of the iris has become zero, and the iris consequently is pushed against and into the opening. This is the way in which a prolapse of the iris originates, the prolapse being nothing but a sac consisting of iris and filled with the liquid of the posterior chamber.

The *danger of a prolapse of the iris* occurring is greater—1. The greater the rapidity with which the aqueous escapes, because then proportionately less time is allowed the liquid of the posterior chamber to make the circuit by way of the pupil. Hence the rule that in making the section the aqueous should be allowed to flow off as slowly as possible. 2. The higher the ocular tension, because then the difference between the pressure in the anterior chamber which has been emptied and the posterior chamber which is full becomes so much the greater. When an iridectomy is made in glaucoma, a considerable extent of the iris usually at once protrudes from the wound. 3. The further the wound lies toward the periphery, for then the route which the aqueous has to take through the pupil is just so much the more circuitous, and, there is a proportionate increase in the depth of the posterior chamber at the spot corresponding to the section and in the quantity of aqueous that acts to propel the iris forward. 4. According to the size and shape of the aperture, since these conditions likewise have an effect in producing prolapse of the iris. It is evident that the opening must be of a certain size for the iris to enter it at all. Again, if the aperture is circular—as is the case, for example, after perforation of an ulcer of the cornea has taken place—prolapse of the iris will assuredly not fail to develop. On the other hand, wounds with a valvular closure, such as those made with the lance knife, display a comparatively slight liability to inclusion of the iris. We therefore try to anticipate the occurrence of perforation of an ulcer by making paracentesis with the lance knife, in order to avoid prolapse of the iris and the anterior synechia that results from it.

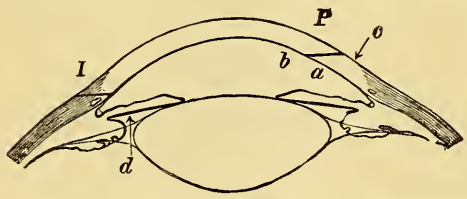


FIG. 408.—OBLIQUE COURSE OF THE SECTION THROUGH THE TUNICS OF THE EYE (SCHEMATIC). *I*, section for iridectomy, lying externally in the sclera, internally in the cornea. *P*, section for paracentesis; *a*, peripheral; *b*, central lip of the wound.

Prolapse of the iris may occur not only during the operation but afterward also. We may, for example, have succeeded in avoiding the development of prolapse during the operation, or have removed it if it has developed; and yet on the next day, when

we change the dressing, we may find the iris prolapsed into the wound. This occurrence is due to the fact that the recently agglutinated wound has reopened, and the moment this took place the same conditions favoring prolapse of the iris were supplied as at the instant of the operation itself.

Prolapse of the iris and its incarceration in the operation wound must be avoided at any cost. If we are dealing with wounds in which there is but little tendency to prolapse, it is sufficient to replace carefully any iris that may have prolapsed during the operation. But if the section is of such a character that the iris is apt to be pushed into it (as, for instance, is the case with a large scleral section), the reposition of the iris affords no security against prolapse; this may, and very often will, take place subsequently. In these cases the only resource is excision of the iris. How does this avert prolapse? Is it perhaps because all the iris is removed that could possibly prolapse into the wound? If that were the case, the iris would have to be excised through the entire extent of the wound—that is, often for a considerable width. But this is not by any means necessary. As a matter of fact, iridectomy prevents prolapse of the iris because it puts the posterior into direct communication with the anterior chamber at the site of the wound, so that the fluid which reaccumulates in the posterior chamber can flow directly toward the opening of the wound without forcing the iris before it. For this purpose a narrow iridectomy or even a very small opening in the periphery of the iris is all that is necessary [see page 996].

807. Escape of Aqueous, Vitreous, and Blood.—[The escape of *aqueous* which follows an incision into the anterior chamber should be made as gradual as possible (see page 918). In certain operations, as in discission (page 987) no aqueous escapes. Escape of *vitreous* presupposes a breach in the sclera or in the diaphragm separating the aqueous and vitreous chambers. It occurs, therefore, particularly in cataract operations (see page 998).—D.] *Hæmorrhage* into the anterior chamber occurs in those operations that cause injury to vascular tissues like the sclera and iris. If the iris is healthy, it hardly bleeds at all upon being cut through, since its vessels close very rapidly from contraction of their walls. But in those cases in which an operation is done upon a diseased iris (as in iritis, glaucoma, and atrophy of the iris), copious bleeding often takes place from the iris, so that the whole anterior chamber fills with blood. [General disturbances of metabolism also tend to provoke bleeding. Thus in a large number of cases of cataract-extraction it was found that hæmorrhage into the anterior chamber occurred in 29 per cent of the diabetic patients, in 13 per cent of those affected with albuminuria, and only 4 per cent of those having neither albumin nor sugar in the urine (Wheeler).—D.] The bleeding is disagreeable, because it prevents the operator from inspecting the interior of the eye; but in otherwise healthy eyes it generally produces no other disadvantage, as the blood is absorbed again within a few days. But in eyes where the iris is diseased not only is the hæmorrhage more extensive, but in addition the blood takes a longer time—sometimes in fact months—to disappear by resorption; for, it is precisely in such cases as these that the whole metabolism of the eye is seriously affected.

Hæmorrhage from divided vessels should not be confounded with those intra-ocular hæmorrhages which are caused by the sudden and very great reduction of ocular tension in consequence of the operation—either from escape of the aqueous or removal of the lens—so that now blood pours in greater quantity into the vessels of the uvea and retina and makes them rupture. Such hæmorrhages are especially to be anticipated when operations are done in the presence of increase of tension, in which case the vessel walls are often degenerated. In fact in glaucoma small retinal hæmorrhages after an iridectomy are the rule (see page 511). In rare cases, blood escapes from the vessels in such quantities that it pours out into the vitreous or beneath the chorioid. In the latter case the chorioid is detached from the sclera by the blood, and indeed when the hæmorrhage is very great it may even happen that the contents of the eyeball are expelled from the wound by the blood ("expulsive" hæmorrhage) and then the blood itself oozes from the wound. Such an eye, of course, is lost.

An escape of *aqueous* after the cornea has been opened presupposes a proportionate contraction upon the part of the capsule of the eyeball. If the walls of the eyeball were perfectly rigid like a metallic capsule, for instance, not a drop of liquid would escape from the orifice which had been made; a counter-opening would have to be made in another spot before it could do so. In addition to the elastic contraction of the tunics of the eye, the pressure of the external ocular muscles, and also the pressure of the lids upon the eye, contribute to reduce the volume of the capsule of the eyeball. Another factor contributing to the same result is added where the diaphragm formed by the lens and zonula is yielding enough to push forward after the escape of the aqueous. In old persons, the capsule of whose eyeball is rigid and whose eyes lie deep in the socket, so that the lids and muscles have but little power over them, the cornea after the escape of the aqueous (especially if the lens is removed at the same time) is often pushed in by the external atmospheric pressure (*collapsus corneæ*). This occurrence is favored by the diminished thickness of the cornea in old age, and also by the use, during the operation, of cocaine, which reduces the ocular tension. Collapse of the cornea was formerly looked upon as an evil event, because it prevents the precise apposition of the lips of the wound, and it was supposed that suppuration of the wound was thus produced. We now know that the process of healing is in no respect affected by a collapse of the cornea. The collapse disappears as soon as the aqueous reaccumulates, which is generally the case as early as a few minutes after the operation. [If this is not the case, the collapse may be relieved by introducing warm sterile salt solution into the anterior chamber. This can be done by means of a bulb irrigator, a slender-tipped syringe, or any like device, introduced between the lips of the wound. The same manœuvre is called for in case of the much more serious collapse of the eyeball caused by great *escape of vitreous*.—D.] When the cornea on account of its elasticity tends to resume its shape after collapse has taken place, a negative pressure develops in the anterior chamber precisely as when the rubber ball of a syringe, after being compressed with the hand, is allowed to expand again. Air may be sucked in by means of this negative pressure, so that an air bubble enters the anterior chamber. This does no sort of harm to the eye. A more unpleasant effect of this aspiration action occurs when the *blood* is sucked out by it from the divided vessels of the iris, so that the chamber is filled with blood. This is particularly apt to take place when the cavity of the chambers is separated from the cavity of the

vitreous by a more solid diaphragm than usual (exudation membranes), which is not able to advance adequately after the escape of the aqueous. Particularly profuse hæmorrhage is thus encountered in those iridectomies and iridotomies which are made in eyes with an old irido-cyclitis. The blood in this case is unpleasant for two reasons: first, because it is very slow in being absorbed; and, second, because it may in part become organized and close the new-made pupil up again. To prevent this hæmorrhage ex vacuo, I apply in such cases a pressure bandage to the eye as soon as possible after the pupil has been formed. This bandage by external pressure diminishes the volume of the capsule of the eyeball, and presses the vitreous against the cornea.

808. Dressing.—After the operation an aseptic dressing is applied. If the operation was upon the eyeball itself, directly after it has been com-

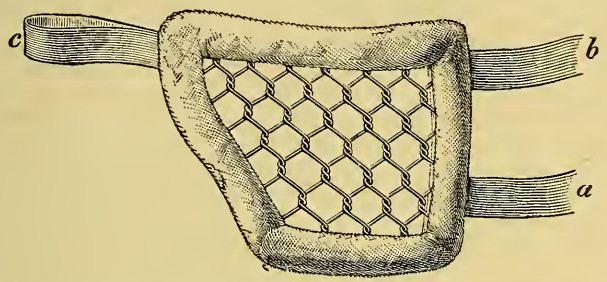
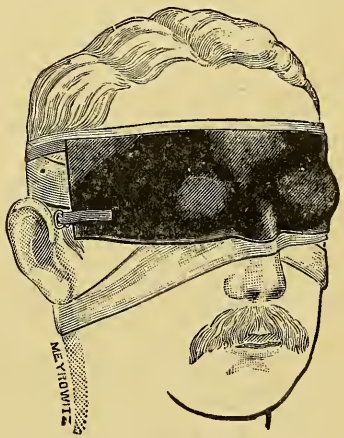


Fig. 409.—Latticed Frame Used for Dressing the Left Eye.

This is padded on its edges so as not to exert any pressure. From the two temporal extremities pass the bands, *a* and *b*, which are carried respectively below and above the left ear and across the occiput to the right side of the head. Here the two bands are tied together after one of them has been passed through the loop *c*.

pleted we close the lids and place upon them first a pledget of sterilized gauze, and upon this lay a dressing of cotton, which is held in place by a bandage. After an operation in which the eyeball is freely opened (as in iridectomy or cataract operations), I fix the mass of cotton in place upon the eye by means of a strip of linen 4 or 5 cm. broad, whose two ends are attached to the cheek and forehead respectively by soap plaster. [In operations of all kinds it is well to fasten down the gauze-cotton dressing securely in some such way as this, and then, except when we wish to produce pressure on the eyeball, the bandage may be omitted—preferably so, according to many, in cataract-extractions.—D.] In operations for cataract, etc., I fasten over the dressing a light latticed frame of wire (Fig. 409), which prevents the patient from getting at his eye with his finger and doing harm by making the wound burst open. Snellen uses for this purpose a plate of aluminum,



[Fig. 410.—Ring's Mask.—D.]

shaped like a mussel shell and fastened over the eye with strips of plaster. [The same end is accomplished very satisfactorily by Ring's light pasteboard mask (Fig. 410). This completely covers both eyes, but, as a window can readily be cut in it, either eye may be left open when desired (see also page 54).—D.]

[Some prefer to dispense with a dressing altogether after operations, believing that it does not really prevent movements of the lids or the eye that would interfere with the healing of the wound, and that it does favor the damming up of secretions and the multiplication of germs in the conjunctival sac. And, as a matter of fact, if the conjunctiva is predisposed to inflammation, bandaging the eye will cause a profuse accumulation of secretion in the sac, so that the dressing has to be removed, and the eye irrigated several times a day with sterile salt solution and argyrol or protargol. So far as statistics go, the results of the open method seem to be as good as when a dressing is employed. Yet the consensus of opinion among the best and most experienced operators is that the dressing should be used in all operations in which the eyeball is opened (cataract, iridectomy). It is also the custom with most to bandage the eyes for several (two to ten) days after an advancement, the intention here being not so much to protect the eye from infection as to keep it from seeing and hence from moving about and so making traction upon the sutures. If this is the intention, both eyes ought to be bandaged, since if this is not done the uncovered eye will move about freely, and in that case the covered eye will necessarily move too.

Even if it is thought wise to omit a dressing in the kind of cases just cited, the eye ought to be protected by a mask from the risk of accidental blows, especially at night.

The open method is suitable for tenotomies. Here, contrary to what obtains in the case of advancement, we wish to have the eyes move under the modifying influence of binocular vision and to be subjected to the fusion impulse which will help them to assume the proper position. As the danger of infection in these cases is very slight and after the first few hours when the external wound is sealed is practically nil, a bandage is used for at most the day of the operation. After this both eyes are left open, and the patient is encouraged to use them for distant vision.

Most prefer to inspect the eye twenty-four hours after the operation, to ascertain the character of the wound and particularly to see whether a prolapse of the iris is present or not; also in the case of an advancement or tenotomy to determine whether the position of the eyes is correct. If the condition is found to be satisfactory the dressing may then be left undisturbed for forty-eight hours, although many still prefer to examine the eye daily.

Under a bandage a secretion often forms which glues the lids together, and makes it somewhat difficult to open the eye for inspection. This tendency to agglutination can be obviated by putting sterile vaselin into the conjunctival sac (with or without argyrol) before applying the dressing. The often painful adhesion of the lashes to the dressing may be prevented by smearing the lids and lashes with sterile vaselin or a boric-acid ointment (see page 54).—D.]

809. After-treatment.—The *after-treatment* of an operation in which the eyeball has been opened must be mainly directed to the prevention of anything that might interfere with the rapid and permanent closure of the wound. For this purpose the eye operated upon is bandaged, in order to put a stop to the movements of the lids; and in operations of any magnitude the eye not operated upon should also be kept shut for the first

day. Furthermore, the patient ought to avoid all physical exertion, as by this the ocular tension is increased, and the recently agglutinated wound might be forced open again. Hence, after major operations (iridectomy or cataract extraction), the patient is made to stay flat on his back in bed for the first day, and on the following days is permitted to be up in an arm-chair. For the first few days he is allowed only soft diet, so as to obviate the exertion of chewing. [For other precautions to be observed

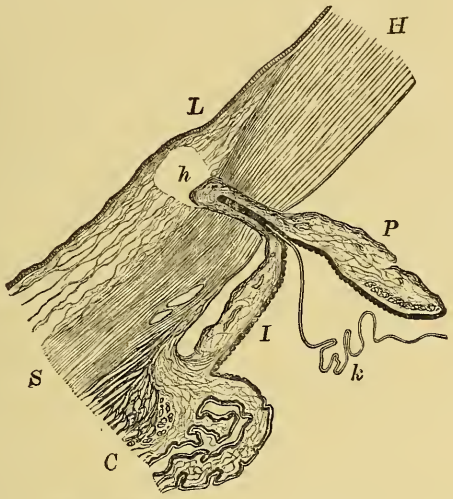


FIG. 411.—INCLUSION OF THE IRIS WITH CYSTOID CICATRIZATION AFTER THE EXTRACTION OF A SENILE CATARACT. Magnified 13 × 1. The iris, *I*, extends from its origin in the ciliary body, *C*, to the inner orifice of the wound, so that here the anterior chamber is contracted into a narrow slit. The iris within the tract of the wound is folded upon itself, the point of flexion corresponding to the exterior surface of the sclera. The pupillary portion, *P*, of the iris extends from the site of the constriction into the anterior chamber, where it lies free. On its anterior surface may be seen the entrance of a crypt, near its posterior surface the cross section of the sphincter pupillæ. Besides the iris, the capsule, *k*, of the lens, is also drawn up to the cicatrix, and has become adherent to it. The section by which the extraction was made cuts in two the line of junction between the sclera, *S*, and the cornea, *H*, so that by its anterior half it lies in the sclera, by its posterior half in the cornea. On account of the interposition of the iris, the lips of the wound have not united, in fact, the tract of the wound extends as an open cavity, *h*, even into the tissue of the conjunctiva of the limbus, *L*, so that the wound is closed only by a very thin layer of tissue.

in post-operative treatment, see page 52.] When these precautions are observed the course of healing regularly takes place as follows: The edges of the wound become agglutinated soon after the operation, and the anterior chamber is restored. It very often happens that the recently agglutinated wound can not at once withstand the pressure of the accumulated aqueous, and in the course of the first day opens once or twice and allows the aqueous to escape before a permanent closure of the wound has taken place. The edges of the wound then heal by indirect union, so that a fine linear cicatrix is formed. If this lies in the cornea, it remains visible always as a narrow gray line, while cicatrices in the sclera are as a usual thing scarcely discoverable after some time has elapsed. It always takes quite a long time for a cicatrix to become sufficiently firm to be able to resist exter-

nal injuries. Until this has occurred (that is, for several weeks or months, according to the size of the wound), the patient must refrain from all severe physical exertion, avoid making any pressure upon the eye, etc.

In old people, especially if they are drinkers, delirium not infrequently occurs, particularly when both eyes are bandaged. In this case the eye that has not been operated upon must be opened at once. [Sometimes the delirium can be relieved only by sending the patients home. Drinkers, moreover, must not be deprived of their alcohol, see page 52. Atropine must be stopped, and hyoscine (0.3 mgr.) may be given (De Schweinitz).—D.]

Old people suffering from marasmus are apt, if they lie quiet upon their backs for several days after an operation, to get hypostases in the lungs, which may cause the patient's death. Hence, persons that are weak from old age ought to be taken out of bed very soon—if necessary, directly after the operation. There are other accidents, too, by which the course of the healing may be interfered with. As these cannot usually be foreseen, it is advisable never to operate upon both eyes at one sitting; for from the operation and after-treatment in the case of one eye we learn what we are to expect when operating upon the second.

Variations from the course of healing just described not infrequently occur. Disturbances of healing most frequently observed are—

1. *Irregular Healing.*—The closure of the wound may be delayed, and the anterior chamber remain obliterated for days. [This is due usually to inclusion of iris, etc., in the lips of the wound (see infra), sometimes to detachment of the chorioid (see page 473).—D.] Still more frequently it happens that the wound, after it has already been closed, is burst open again by an external injury, such as pressure of the hand upon the eye, coughing, sneezing, spasmodic tremor in sleep, and the like. This "bursting of the wound" is ordinarily followed by extravasation of blood into the anterior chamber. The iris also may be swept into the wound and incarcerated there, or inflammation (irido-cyclitis) may set in. Another way in which healing of the wound may be interfered with consists in the fact that the edges of the wound do not adhere to one another directly, but are united by an interposed cicatricial mass of recent formation. This is most frequently the case when direct contact of the edges of the wound is prevented by the presence of an incarcerated iris or lens capsule [or conjunctiva], but it may also occur when there is an increase of tension by means of which the wound is made to gape and is thus kept open. In these cases the scar that is formed is less firm, and in fact a small portion of it may remain entirely open, so that the aqueous keeps oozing through it beneath the conjunctiva and makes the latter œdematous (*cystoid cicatrization*, Fig. 411). The scars which have not sufficient firmness frequently become ectatic. This has as its immediate consequence an irregular bulging of the adjoining parts of the cornea, so that the results of the operation, so far as vision is concerned, are impaired by the presence of high astigmatism. Later on, cystoid or ectatic scars may give rise to elevation of tension or to inflammation.

2. *Suppuration of the Wound.*—This usually begins in one or two days after the operation, rarely later. If the germs have made their way into the lips of the wound itself, the inflammation shows itself by a yellow infiltration of the edges of the wound; but, if the germs have been introduced into the deeper parts—anterior chamber or vitreous,—the inflammation begins with an exudation, which is poured out into the interior of the eye and which is first fibrinous, but very soon becomes purulent. It is only in the rarest cases that such an inflammation is arrested so quickly that the eye retains some visual power. The rule is that the eye is destroyed either by suppuration of the cornea or by a severe plastic irido-cyclitis or by an actual panophthalmitis. The outcome is atrophy, or, if panophthalmitis is superadded, phthisis of the eyeball. [The lighter cases may sometimes be checked by cauterizing the wound with the cautery or carbolic acid or by repeated paracentesis of the cornea with injection of formalin or other antiseptic into the anterior chamber (De Schweinitz). The subcutaneous injection of vaccines has saved some cases.—D.] Suppuration of the wound is most apt to set in after cataract extractions, and formerly was the most frequent cause of blindness in an eye which had been operated upon for cataract. We now know that suppuration of the wound is the consequence of infection of the wound; and by the application of antiseptic methods the number of cases in which suppuration takes place can now be reduced to a minimum.

3. *Non-Suppurative Inflammation of the Uvea.*—Iritis and irido-cyclitis occur very frequently after operations in which the eyeball has been opened. In most cases there is simply a slight iritis, which does no harm beyond that induced by the persistence of a few posterior synechiæ. But in the severe cases the inflammation leads to occlusion of the pupil, and either necessitates a secondary operation, or actually terminates in incurable blindness due to atrophy of the eyeball. In cases of the latter sort there is also a danger of sympathetic disease of the other eye. Slight inflammations of the iris may be produced by infection of a particularly mild character (see page 36); but in other cases they are to be regarded as purely traumatic, being caused by the way in which the iris has been grasped and pulled upon or by the fact that portions of the lens which are left behind, or similar substances, produce mechanical or chemical irritation of the iris. Severe inflammations depend either upon infection or upon a lighting up of old inflammation, as when an operation is done in an eye which was formerly the seat of an iridocyclitis.

(For *striate opacity* of the cornea after operations, see page 303.)

CHAPTER II

OPERATIONS ON THE CONJUNCTIVA AND LACHRYMAL ORGANS

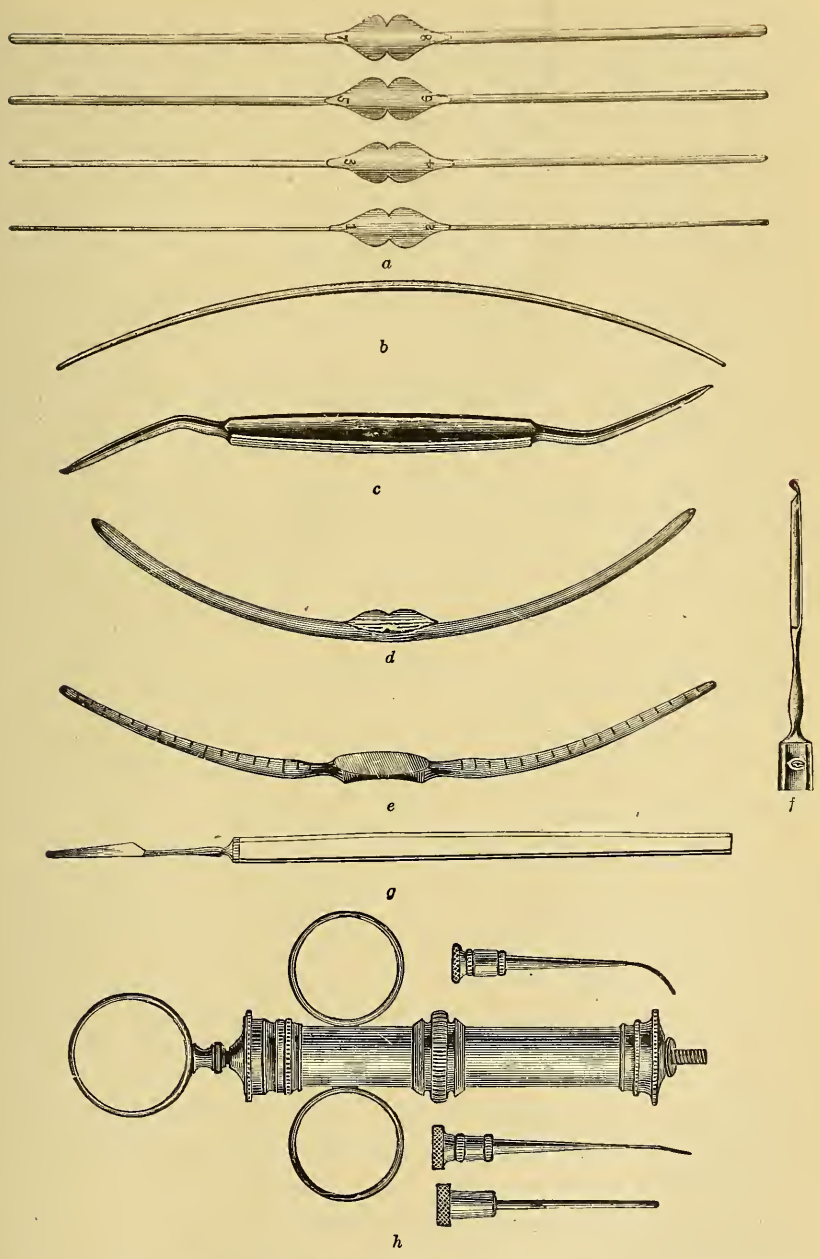
I Dilatation of the Tear Passages

811. [Dilatation of the canaliculi and the nasal duct is effected either with Anel's syringe (Fig. 412, *h*) or with sounds.

(*a*). **Syringing the Duct.**—Before using the syringe, the punctum and canaliculus should be dilated with the conical sound (Fig. 412, *b* and *c*). A little cocaine-adrenaline solution is dropped on the punctum, and the tip of the sound introduced with gradually increasing pressure. When well engaged it is turned horizontally inward and pushed gently toward the sac. Immediately afterward the tip of the syringe, properly bent, is introduced and gently shoved into the sac, and then 5 to 10 drops of a 2-per-cent solution of cocaine with a few drops of adrenaline solution added are injected. The cocaine and adrenaline not only make the passages insensitive, but also shrink up the mucous membrane and so make the passages wider. When the passages are well opened by this means we may inject disinfectant solutions (1-4,000 sublimate solution, 3-per-cent solution of boric acid), which can afterward be replaced by astringent solutions. [Or, even better, we may instill 30-per-cent argyrol just before the injection of the cocaine-adrenaline. When the latter has made its way through to the nose the argyrol will generally follow. (It is better not to inject the argyrol, as argyrosis may be produced in this way).] Syringing may be done twice a week or oftener, the patient in the intervals using instillatims of adrenaline followed by argyrol.—D.]

812. Slitting of Canaliculus.—[This is done as a preliminary to the use of sounds. We dilate the lower canaliculus with a probe, and syringe in a little adrenaline-cocaine solution as directed in the preceding section, then] we introduce a Weber's knife (see Fig. 412, *f*) into the dilated canaliculus, in such a way that the edge of the knife looks up and a little backward. By rapidly raising the knife, the probe-pointed extremity of which rests in the lachrymal sac, the canaliculus is slit open, and is thus converted into an open groove, looking upward and a little backward. [Through this groove we can introduce sounds much too large to pass through the uncut canaliculus (see page 933).—D.]

Bowman has the credit of showing that the conduction of tears is not injuriously affected by the slitting of the canaliculus. If this operation is performed with Weber's knife in the way described above, it affects only the external two-thirds of the canaliculus; the inner third of the latter lies beneath (behind) the caruncle, and remains unopened. Slitting of this most internal part would be possible only if the caruncle



[Fig. 412.—Instruments for Lachrymal Stricture.

a, Bowman's sounds. *b*, Ziegler's needle dilator for dilating the canaliculus. *c*, Ziegler's dilator for use after the needle-dilator. *d*, Theobald's probe. *e*, Weber's thick sound for graduated dilatation. *f*, Weber's canaliculus knife for slitting the canaliculus. *g*, Stilling's knife, for division of lachrymal strictures. *h*, Anel's syringe.—D.]

were divided at the same time; and if this were done, the cut surfaces that were made would be pretty broad, and it would be quite hard to prevent their reunion. Even in the ordinary method of performing the operation of slitting, the div ded canaliculus is usually found to have closed up again on the following day, so that we have to introduce the conical sound and thus tear apart the slightly agglutinated edges of the wound. If in this way readhesion is prevented at the outset, the cut surfaces become clothed with epithelium, so that afterward adhesion is no longer to be apprehended.

813. Sounding the Duct.—For *sounding* the nasal duct we make use of Bowman's sounds, [Fig. 412, *a*] which are kept on hand in different sizes, numbered from one to six [or eight. In this country many prefer Theobald's probes, which are larger (Fig. 412, *d*; see also page 933).—D.]

The sound is first introduced through the inferior canaliculus, after this has been slit open, and is passed in until it strikes against the inner wall of the lachrymal sac; the sound taking the direction of the canaliculus—that is, passing from below outward in a direction upward and inward. Then we tilt the sound—that is, we direct it so as to be about perpendicular by lifting its free extremity until its point, which is in the lachrymal sac, points to the furrow between the ala of the nose and the cheek, this giving the position of the inferior orifice of the nasal duct. The sound, being thus placed in an upright position, is now slowly and cautiously pushed downward until it rests upon the floor of the nasal fossa. In so doing we necessarily pass the contracted point, the favorite seat of which is either the point where the lachrymal sac enters the nasal duct, or the inferior extremity of the latter—the former spot, because it is normally the narrowest point in the lachrymal passages; the latter, because it is affected sooner than is the rest of the lachrymal tract by diseases of the nasal mucous membrane. We begin with the slenderest sounds. If we cannot pass the contracted spot with one of these, we must not push the sound forcibly forward, but must keep trying again and again, on succeeding days to introduce the sound, until at length we succeed in carrying it down into the nose. The sound after its introduction is allowed to remain for about a quarter of an hour. We repeat the sounding every day, or every other day, gradually passing to larger and larger sounds, until at length the nasal duct is readily pervious and the epiphora has ceased. Even then the sounding should not be at once discontinued, as in that case the spots which have been dilated would soon close up again, owing to renewed contraction of the cicatrices. The sounding must therefore be repeated again and again at longer intervals (of a week to a month).

The operation of sounding can be performed through *either the upper* or *lower* canaliculus. The former is narrower, but, as an offset to this, we have to turn the sound but little after its introduction in order to place it upright. In sounding through the lower canaliculus, the sound must be tilted so as to be turned through more than a right angle, but the canaliculus is wider. Accordingly, the lower canaliculus is usu-

ally selected because the mucous membrane of the narrow canal would be lacerated in passing sounds of a higher number. The consequence of this would be contraction or obliteration of the canal, which would set in as soon as the sounding was stopped. The sound can also be introduced and carried into the nose through a canaliculus which has not been slit up, but this is inadvisable, because of the likelihood of producing injuries to the mucous membrane like those just mentioned. I usually do it only for diagnostic purposes (for demonstrating the presence of a stricture) and then employ only the sounds of the lowest number, which can be readily introduced even through a canaliculus that has not been slit open. Preliminary slitting of the canaliculus is always indicated when the treatment by sounds is to be kept up for any length of time.

The act of sounding itself requires a deft hand and much experience, for which reason it ought first to be practised very industriously upon the cadaver. Beginners in sounding frequently make the mistake of tilting the sound before its point has got into the lachrymal sac. We then feel an obstacle opposing the downward progress of the sound, and if we should try to overcome this obstacle by force we would make a false passage. That the sound has been tilted up too soon is recognized from the fact that when it is set vertical, the skin beneath the canaliculus is drawn along with it and is thrown into wrinkles. We shall not commit this mistake if we avoid tilting the sound until we distinctly feel through its point the firm resistance offered by the inner osseous wall of the lachrymal sac (the lachrymal bone). The obstacles opposing the passage of the sound in the nasal duct may be actual constrictions of the duct, but are often nothing but projecting folds in which the sound catches. We accordingly try to push our way forward by sliding the point of the sound sometimes along one, sometimes the other, wall of the lachrymal sac in order to smooth out the folds. Sometimes we can pass a rather thicker sound (No. 3) more readily than we can the thinnest ones; the latter, moreover, are more apt to injure the mucous membrane, so that we get beneath it and make a false passage. Bleeding from the nose after sounding points to an injury of the mucous membrane, as does also the striking of the apex of the sound upon bare bone. The latter, however, can also happen without the mucous membrane's being injured, if, for example, the bone has been already denuded by ulcerative disintegration of the mucous membrane of the nasal duct. In cases of this kind a cure cannot usually be obtained by treatment with sounds.

When finally the sound has been passed all the way through, it is felt to rest upon the floor of the nasal cavity. In most persons the lamina in the middle of the sound then lies upon the inner end of the eyebrow. To ascertain the position of the sound more precisely, we may place on the outside a second sound of the same length in the direction of the lachrymal tract and in such a way that the laminæ of the two sounds are superimposed; the exterior sound then shows at what height the lower extremity of the sound that we have introduced stands.

Treatment by sounds must be kept up until at least No. 4 of Bowman's sounds passes with ease. Then the patient may be instructed how to pass the sound upon himself with the aid of a looking-glass, so that the sounding may be performed from time to time subsequently in order to prevent a recurrence of the contraction.

The long duration of the treatment by sounds has excited a desire to accomplish the dilatation of the strictures rapidly instead of gradually, and thus shorten the treatment. This can be done either by Weber's [or Theobald's] method of introducing very thick sounds [Fig. 412, *d* and *e*] or by Stilling's method of incising the strictures with a knife designed for the purpose [Fig. 412, *g*], or by a combination of both methods. By these methods of treatment, however, solutions of continuity are made in the mucous membrane, which lead to the formation of fresh cicatrices, and which hence,

after a period of apparent cure, entail even quicker recurrences than usual. Most ophthalmologists therefore prefer the gradual dilatation of the strictures.

814. [Artificial Closure of Puncta.]—A procedure for shutting off the connection between the lachrymal sac and the conjunctival sac before an operation on the eyeball and thus preventing infection of the wound consists in searing the canaliculi by cauterization. For this purpose a fine galvano-cautery point is carried into the canaliculus. The latter is at once obliterated by the eschar and by further action is completely seared. If, however, the cauterization is performed lightly, the permeability of the canaliculus can be restored [after the operation wound has healed]. Even when we extirpate the tear sac before operating on the eyeball we ought at the same time to sear the canaliculi, since otherwise the latter may become the source of infection. [Quackenboss and others accomplish the same end by passing a ligature round the canaliculi before an operation. When the operation wound is healed the ligature can be removed and the lumen of the canaliculus restored.—D.]

II. Operations on Lachrymal Sac and Glands

815. [Incision of Lachrymal Sac (Dacryocystotomy).—In *acute dacryocystitis*] as soon as fluctuation is apparent we incise the anterior wall of the lachrymal sac, or that portion of the skin beneath which the presence of pus can be made out. A lachrymal fistula is thus artificially produced, through which the contents of the abscess and of the lachrymal sac itself are discharged externally. This is kept open by the introduction of a strip of iodoform gauze every day, until all inflammatory symptoms have disappeared and the secretion that exudes has lost its purulent character. But even then we ought not to allow the fistula to close at once, for we must recollect that a chronic dacryocystitis has preceded the acute attack, and that consequently there is a stricture present in the nasal duct. So long as this is present the fistula will not become permanently closed, and its temporary closure might result in a new attack of acute dacryocystitis. Hence, the permeability of the nasal duct must first be restored by treatment with sounds. When we have succeeded in doing this the fistula usually closes of itself. If this should not be the case, we can effect a closure of the fistula by either refreshing and uniting its edges or by cauterizing them. If the conditions are such that a permanent state of perviousness of the lachrymal channels is unattainable, or if the patient cannot undergo the protracted treatment with sounds, we proceed to extirpate the lachrymal sac [or perform West's operation. See pages 935, 936. To the translator this radical treatment seems the better procedure, and he has even found it of distinct advantage to extirpate the sac in the acute stage of dacryocystitis right after incision.—D.]

In *chronic dacryocystitis* incision is done by Petit's method, for the performance of which Arlt has given the following guides: By drawing the lids to the outer side the internal palpebral ligament is put on the stretch, so that it is seen through the skin of the inner angle of the eye as a prominent projection. The point of a sharp scalpel is introduced exactly beneath the center of the ligament. The back of the knife looks upward and the knife itself is held so that its handle passes through the middle point

of an imaginary line drawn from the apex of the nose to the outer margin of the orbit. The knife held in this direction is thrust vertically in, thus penetrating through the skin and the anterior wall of the lachrymal sac. As soon as we feel the point of the knife striking against the posterior wall of the lachrymal sac (lachrymal bone) we no longer push it forward, but depress its point by raising the handle as high as the forehead. If now the knife is pushed forward, its point enters the upper part of the nasal duct, the wound in the anterior wall of the lachrymal sac being at the same time enlarged. After withdrawing the knife we enlarge the wound upward and downward so as to have a view of the mucous membrane of the sac throughout its whole extent.

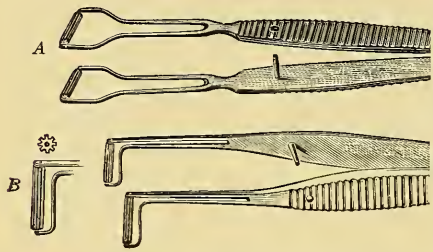
Incision of the sac was formerly done in order to introduce corrosive agents into it and thus destroy it. This procedure is now replaced by that of extirpation of the sac. Typical incision of the sac I scarcely even do now except as a preliminary to a cataract operation or to an iridectomy. If, before such an operation, I find an evident dacryocystitis with abnormal contents in the sac, I extirpate the latter. If, however, it cannot be demonstrated that the secretion from the mucous membrane of the sac is abnormal, but there is only the suspicion that it is so, I content myself with opening the sac, washing it out and packing it off with iodoform gauze directly before the operation on the eyeball. When after the wound in the eyeball heals the gauze is taken out of the sac, the wound in the latter readheres and the sac performs its function as before.

816. Extirpation of Sac [Dacryocystectomy].—For this purpose we first inject some cocaine-adrenalin solution beneath the skin over the tear sac, and also through the canaliculi into the tear sac itself. Then we make a curved incision through the skin 3 mm. to the nasal side of the inner canthus, beginning a little above the internal palpebral ligament and dividing the latter[1] and running 2 cm. downward and somewhat outward. [We distend the lips of the wound with a small spring speculum (Müller's), which not only exposes the field of operation but also checks the bleeding.] Then beginning at the temporal side of the tear sac and using a blunt instrument, we dissect the sac out without opening it. When the sac has been isolated completely, it is divided with the scissors close to the point where it passes into the nasal duct, i. e., at the upper end of the bony canal. The extirpation must be very carefully done since, if the smallest bit of mucous membrane is left behind, suppuration will recur from the canaliculi or from a fistula. If, owing to the great friability of the mucous membrane, it is impossible to dissect it out completely, we must curette the remaining portions out with a sharp spoon. Then we sew up the outer wound and by means of a pressure bandage make sure that the walls of the cavity are in apposition. As a rule, primary union occurs and healing is complete in a few days. [In Verhoeff's operation, which exposes the parts well and is attended with little bleeding, a curved incision, 15 mm. long and extending rather more above than below the canthal ligament, is made with its apex directed toward the eye, and the skin flap thus formed is laid back over the nose.—D.]

[1] [The operation can also be done without dividing the internal palpebral ligament, the cupola of the sac which lies behind the ligament being simply drawn down and dissected out. The extirpation may be facilitated by injecting the sac with paraffin (its lower end being tied off before this is done); or if the sac has already been opened it may be packed with gauze.—D.]

817. Operations Effecting Permanent Drainage.—Since by extirpation of the lachrymal sac the normal drainage of tears is made forever impossible Toti has proposed as a substitute for this operation that of *dacryocystorrhinostomy*. The medial wall of the sac is removed, and a correspondingly large gap is also made in its bony substratum by the resection of a piece of the lachrymal bone and of the frontal process of the superior maxilla. Thus the lumen of the lachrymal sac is brought into direct connection with the nasal fossa, into which the contents of the sac can now discharge, though passing outside of the nasal duct. The direct result of the operation is good so far as the epiphora is concerned, but further experience is required to determine the permanence of the result and the protection that the operation affords against infection of the cornea by bacteria from the lachrymal sac. The same is true of *West's operation*, by which the tear sac is opened from the nasal cavity and to such an extent that a permanent communication is effected. [Other operations for the same purpose are those of Clark and Yankauer.—D.]

818. Extirpation of Lachrymal Glands.—Extirpation of the *superior lachrymal gland* is performed through an incision made in the outer part of the previously shaved eyebrow. (It is made here in order that the scar shall be invisible afterward.) This operation is done particularly in cases of degeneration of the lachrymal gland.



[Fig. 413.—Roller Forceps for Trachoma. *A*. Knapp's forceps. *B*. Rust's modification. Similar forceps have been devised by Noyes and others.—D.]

The *inferior lachrymal gland* is extirpated through the conjunctival sac. After everting the upper lid we draw down the retrotarsal fold with the forceps and inject some cocaine solution beneath the temporal half of the fold. Then we cut the latter longitudinally with the scissors as far as the outer commissure. If now we separate the edges of the wound we see the acini of the gland presenting, and can readily dissect them out. Since the acini are grouped about the excretory ducts of the upper gland these ducts are divided at the same time. This explains the marked effect of the operation on the secretion of tears.

III. Expression and Grattage

819. In performing *expression* for trachoma we attempt to remove the granules without destroying the conjunctiva. This may be done either by puncturing the granulations individually with a sharp knife and then squeezing them out (Sattler), or by passing the retrotarsal folds through the blades of Knapp's roller forceps. In the latter instrument each blade carries a fluted roller, and [when the lids are everted] the conjunctiva is drawn in between the two rollers, which fit closely into each other, and the trachoma granules are squeezed out.

[Many scarify the retrotarsal folds with a small triple-bladed scarifier before expressing the granulations. Kuhnt uses an "expressor," which has smooth blades and simply squeezes the granules out without making traction on the conjunctiva or tearing it. Others prefer *brossage*, which consists in brushing the everted lids vigorously with horizontal sweeps of a small sterile toothbrush. *Grattage* is brossage with

preliminary scarification, the toothbrush being soaked in a 1:500 or 1:2000 sublimate solution. All these operations are quite painful and in children require general anæsthesia. In others they may be performed with local anæsthesia, particularly if, following Guttman's suggestion, we inject a very weak solution of cocaine directly into the retrotarsal folds. The translator's experience, agreeing with that of others, is that expression with the roller forceps is a satisfactory method, which, if properly performed, is rarely attended with undue reaction or untoward symptoms. The expression may be applied not only to the retrotarsal folds but to the tarsus as well. In this case one branch of the forceps is placed on the cutaneous, the other on the tarsal surface of the lids, and the latter is squeezed by the forceps, which is rolled in different directions.—D.]

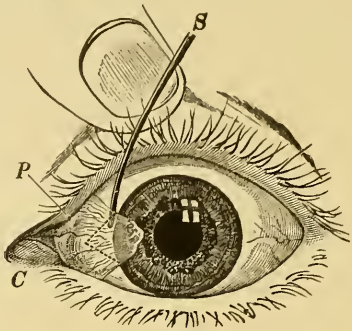


Fig. 414.—Operation for Pterygium.—Dotted lines indicate incisions.

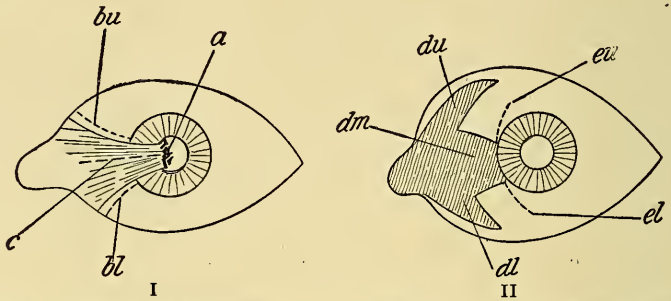
IV. Operations for Pterygium and Superficial Growths on Eye

820. A pterygium may be ablated by Arlt's method. With a toothed forceps we grasp the pterygium at its neck, where the incurving of its margin permits it to be partially lifted from its bed. Starting from this point, we carefully dissect off the head from the cornea, upon which it lies, keeping strictly to the line of division between the tissue of the pterygium and the cornea. We must be particularly careful to remove thoroughly the gray, gelatinous zone at the apex of the pterygium, scraping it away if necessary. When the whole of the head as far as the limbus has been separated from the cornea we circumscribe this segment by carrying from the upper and lower margins of the neck two converging incisions into the body of the pterygium (the dotted line, Fig. 414). In this way a rhomboidal piece, containing the head and part of the body, is excised and a raw surface remains, one portion of which lies in the cornea, the other in the conjunctiva of the eyeball. This last is covered by uniting the upper and lower margins of the sections by means of one or two interrupted sutures. The wound in the cornea heals by a cicatrix forming over it, in consequence of which a permanent opacity remains. Care in sewing up the conjunctival wound is of the greatest importance, especially in the neighborhood of the limbus, as otherwise the conjunctiva grows anew over the raw surface of the cornea, and thus the pyterygium recurs. However, even after carefully performed operations relapses are not infrequent, and then a second removal is required.

[Fig. 415.—Prince's Divulsor.]

Arlt deserves the credit of having made the operation for pterygium a success by demonstrating the necessity of closing the conjunctival wound. Previously people had been satisfied with simple ablation, and had so frequently had recurrences after this operation that with many it had altogether fallen into disrepute.

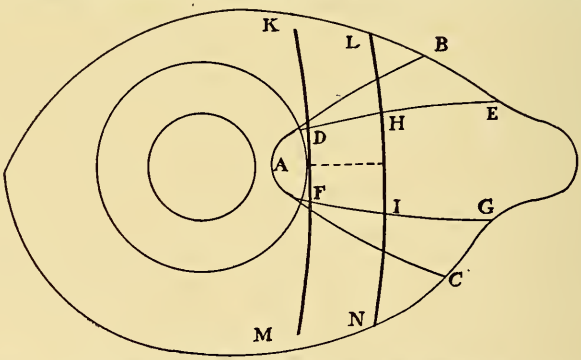
[In *detaching* the pterygium a blunt divulsor (Fig. 415) may be used. This is introduced beneath the pterygium and brought like a draw-knife toward the pterygium head. Or, following the ingenious suggestion of Hunter, the pterygium may be stripped off clean by a thread passed beneath it and carried in toward the cornea with a sawing motion.—D.]



[Fig. 416.—Knapp's Transplantation of Pterygium. (After Norris and Oliver.)

The head of the pterygium (I, *a*) is dissected off, and the incisions *bu* and *bl* carried along the margins of the pterygium through the conjunctiva. These incisions are prolonged into the upper and the lower fornix, producing in each an angular gap (II, *dl* and *du*). The pterygium is then detached, its head is cut off, and the pterygium split by a horizontal incision (I, *c*) into an upper and a lower half. The tip of the upper half is stitched into the defect *du*, the tip of the lower half into *dl*. The incisions *eu* and *el* are made, delimiting two quadrangular conjunctival flaps, which are undermined and then brought together and stitched so as to cover the defect *dm*. The innermost stitch connecting these flaps is also passed through the base of the pterygium.—D.]

In very broad pterygia it is difficult or impossible to unite the edges of the conjunctival wound by a suture. In that case liberating incisions are made through the adjacent conjunctiva in order to make it easy to draw it down; and, instead of cutting off the tip of the pterygium, it may



[Fig. 417.—Beard's Operation for Pterygium.

BAC, pterygium. Two slightly diverging incisions, *DE* and *FG* made through the upper layer only of the conjunctiva (not through the whole thickness of the pterygium), are carried well to the canthus. An instrument to strip up the pterygium is introduced through incision *FI* and brought out in incision *DH;* then is carried toward the cornea so as to detach the head *DAF*. The latter is cut off, and then the body of the pterygium, so far as it is included between the incisions *DH* and *FI*, is carefully dissected from its bed and pushed back toward the canthus, *DF* retracting to *HI* and leaving a raw surface, *DHFI*. The conjunctiva above *DH* and below *FI* is undermined, and the incisions, *HL*, *LK*, *FM*, and *IN*, made so as to delimit the flaps *KDHL* and *MFIN*. These are drawn together so that *DH* and *FI* meet in the dotted line. They are attached to each other by a single suture and by another suture to the retracted pterygium *THIG*.—D.]

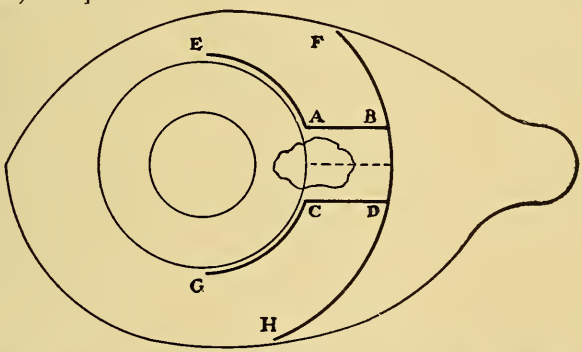
be sewed into the gaps which have been made by the liberating incisions. This is called *transplanting* the pterygium. [*Knapp's* method of transplan-

tation is shown in Fig. 416. *McReynold's* method is somewhat like this, but the conjunctiva is divided along the lower edge only of the pterygium, the conjunctiva below the pterygium is undermined, and the pterygium is drawn down into the pocket thus formed and sewed there by a double-armed suture passed through the tip of the pterygium and brought out through the conjunctiva 6 or 7 mm. below the cornea. The upper edge of the pterygium with the conjunctiva still attached to it is thus drawn out smooth and flat.—D.]

[*Beard's operation* for pterygium is shown in Fig. 417.—D.]

[Pterygia may also be destroyed by *strangulation*. Ligatures are passed round the neck and body of the growths, tied tight, and left in till they cut their way through. The head of the pterygium then atrophies. To secure the same object the neck of the pterygium may be seared with the *cautery*.—D.]

[Quite similar to Beard's operation for pterygium is his *operation for removing superficial growths* (dermoid, etc.) at the sclero-corneal margin. (See Fig. 418)—D.]



[Fig. 418.—Beard's Operation for Superficial Growths at the Limbus.
The incisions, *AB* and *CD*, are made so as to include the whole of the growth. The latter is stripped up and shaved off clean just as in the case of a pterygium (Fig. 417), but, instead of being simply shoved aside, is removed in its entirety. There is thus left a raw surface, *BACD*. The conjunctiva above *AB* and below *CD* is undermined, and by incisions, *AE*, *CG*, *BF* and *DH*, concentric with the cornea, the flaps, *EABF* and *GCDH*, are delimited. These are brought together so that *AB* and *CD* meet along the dotted line. A stitch or two serve to keep them in place.—D.]

V. Operations for Symblepharon

821. Cases of *symblepharon anterius* are easily cured. We separate the adhesions between the lid and the eyeball with great care, so as not to cut into the sclera or the tarsus. When the lid has been set free, we must then make it our business to prevent a readhesion of the freshly made raw surfaces, and to make each of these cicatrize by itself. This is effected by repeatedly drawing the lid away from the eyeball, and also by interposing a pledget dipped in oil or smeared with ointment between the lid and the eye.

In *symblepharon posterius* we also begin by separating the adhesions as far back as the fornix. We then see, when we have drawn the lid

away from the eyeball, two corresponding raw surfaces, one in the eyeball, the other on the lid (Fig. 419). These two opposed surfaces, which meet at the fornix, would soon reunite, beginning at the fornix, if we did not take care to have one of the raw surfaces covered with conjunctiva, so that a place coated with epithelium lies opposite to the raw surface remaining. The wound on the eyeball is the one we select for covering over, because the conjunctiva of the eyeball can readily be displaced, while the conjunctiva of the lid is adherent immovably to the tarsus. We loosen the conjunctiva of the eyeball on both sides of the wound, draw it down over the latter, and unite it by sutures. Especial care must be devoted to uniting the wound in the neighborhood of the fornix. If, after dividing the adhesions, the raw surface upon the eyeball is so large that it cannot possibly be covered by conjunctiva, reunion of the symblepharon will inevitably result. Hence, cases of extensive symblepharon posterius, and obviously also cases of symblepharon totale, are incurable. The same is true of symblepharon induced by the gradual shrinking of the conjunctiva.

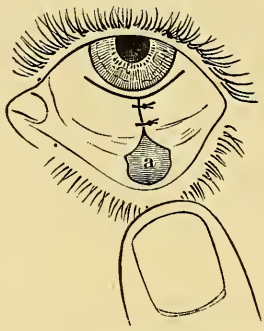
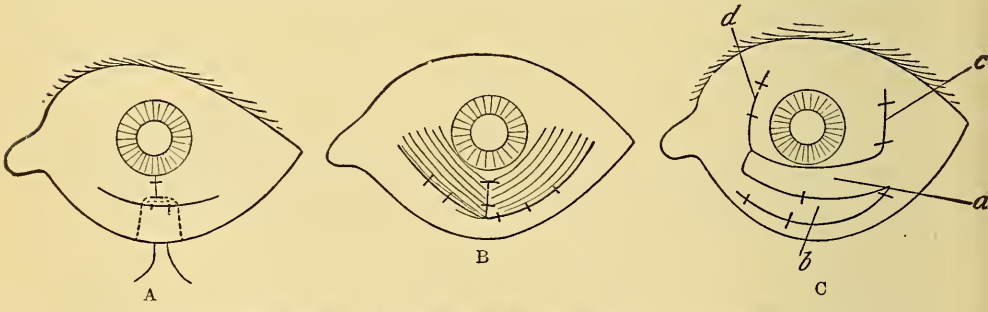


FIG. 419.—OPERATION FOR A SYMBLEPHARON POSTERIUS.

The wound in the palpebral conjunctiva (*a*) has been left open, that in the bulbar conjunctiva closed by two interrupted sutures. In order to be able to draw the conjunctiva up over the raw surface, a liberating incision has been made along the margin of the cornea, inward and outward from the upper end of the wound.



[FIG. 420.—OPERATIONS FOR SYMBLEPHARON. (After Knapp in Norris and Oliver.)

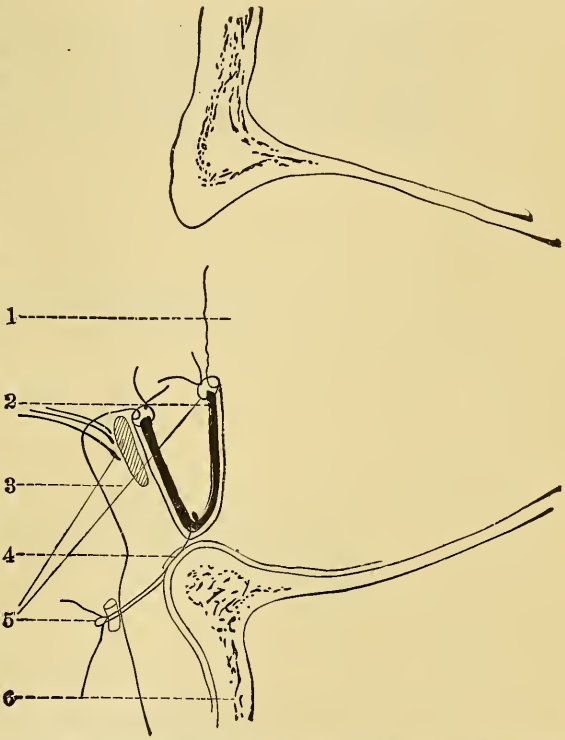
A. Arlt's second method used when the cornea is encroached upon (pseudo-pterygium). This latter is detached from the cornea and eyeball down to the fornix, and the head of it is stitched into the fornix by a double-armed thread, which is passed through the lid so as to form a loop on the cutaneous surface of the latter. This loop is tied over a roll of gauze or bit of rubber tubing. The raw surface of the conjunctiva is covered in as shown in Fig. 419.

B. Knapp's method for covering the defect with two sliding flaps of conjunctiva. To prevent these flaps from over-riding the cornea their lower borders are fastened down with stitches carried through the submucous tissue of the fornix or through the thickness of the lids as described in A.

C. Teale's method. A sliding quadrangular vertical flap is fashioned out of conjunctiva at *c* and another one at *d*. Flap *c*, is brought down, rotated 90°, and stitched in place so as to cover the defects in the bulbar conjunctiva, flap *d* covering that in the tarsal conjunctiva. The raw spots left by the removal of these flaps are covered in by drawing the adjacent conjunctiva over them and stitching it in place.—D.]

We sometimes also operate for symblepharon in the case of a blind and shriveled eye so as to be able to introduce an artificial eye over it.

Different methods [besides the above of Arlt's] have been devised to enable us to operate for cases of symblepharon posterius with extensive adhesion. Himly, in an operation analogous to that for syndactylism, first made a preliminary perforation of the adhesion along the fornix, and carried a lead wire through the canal so formed. The latter, after the wire has been in it a pretty long time, becomes covered with epithelium (like the canal made by piercing the lobule of the ear for ear-rings), so that the symblepharon posterius is changed into a symblepharon anterius, and can be operated upon, like the latter, by simply separating the adhesion. Some have attempted to cover a large loss of substance upon the eyeball, caused by the removal of a symblepharon, by making the conjunctiva that is to be drawn up over it movable by means of liberating incisions, or by fashioning pedicellate flaps out of conjunctiva and attaching them to the wound (Teale, Knapp). Others (Stellwag, Wolfe) have grafted non-pedicellate flaps formed out of bits of mucous membrane from other localities (the conjunctiva of the other eye, the mucous membrane of the lips, or that from the mouth or vagina, or even the mucous membrane of animals) upon the raw spot, and have obtained union. Others again, for covering a loss of substance, take the external skin either in the form of small flaps grafted upon the surface, or as pedicellate flaps which are thrust in between the eyeball and the lid through a fenestra cut through the latter (Kuhnt, Snellen). As an adjunct in these different methods we may directly after the completion of the operation insert a properly fitting shield into the new conjunctival sac between the eyeball and the lids and let it stay until firm cicatrization has ensued.



[Fig. 421. Formation of New Cul-de-sac (after Weeks).—1, orbital tissue; 2, flap in position; 3, lid; 4, periosteum; 5, sutures; 6, bone of orbit.—D.]

Generally speaking, we are obliged to acknowledge that in extensive adhesions all methods have but scanty results to offer, since the symblepharon commonly returns on account of the subsequent shrinking of the conjunctiva.

822. [Formation of New Cul-de-Sac.—When the symblepharon is such as to obliterate the fornix, a new fornix may be made (especially for the reception of an artificial eye) by inserting a Wolfe graft into the groove made by dissecting out the cicatricial tissue, anchoring the graft down by stitches passed through the periosteum

of the orbital margin and the lids (see Fig. 421), and keeping it in place by a gutta-percha plate (Weeks). Maxwell takes a flap from the cheek to form a floor for the socket (Posey). Verhoeff uses Thiersch grafts held in place by a large glass ball, the tendency of the tissues to contract being overcome by a pressure bandage applied over the ball. If the resulting cavity is too deep, he permanently implants a glass ball deep in the orbit. Zentmayer also has used a glass ball to support Thiersch grafts in the orbit.—D.]

823. [Transplantation of Conjunctiva.—This may be done (*a*) for] rapidly advancing *ulcers* of the cornea. The ulcers are cleansed by scraping the floor and walls and then a flap, either with a single pedicle or bridge-shaped, is cut from the conjunctiva of the eyeball close to the margin of the cornea. This flap is then applied to the surface of the ulcer without being attached to it in any special way. [A similar operation is used to close in fistulæ of the cornea (page 266). [(*b*) To cover *wounds*, especially operation wounds, of the conjunctiva, cornea, and sclera. In the case of operations on the cornea or sclera, we form the conjunctival flaps first, and in such a way that the incision in the conjunctiva shall be at some distance from the corneal or scleral incision, and then expose the site of the latter by reflecting the flap (see page 919). When the cornea is cut into or cut away at some distance from the limbus, two or more flaps may be made in the conjunctiva, then drawn up over the cornea and sewed together over the wound (conjunctivo-keratoplasty; cf. page 971). A like principle holds in covering in non-operative wounds of the cornea and sclera. De Wecker in such cases covered the entire cornea by dissecting up the conjunctiva all round the cornea, drawing it down over the latter, and holding it in place with a purse-string suture. This sort of flap is very useful in corneal wounds that are likely to become infected (De Schweinitz). It is kept in place for eight days. For covering defects of conjunctiva, flaps are made of various shapes according to the exigencies of the case. Cf. Beard's operations (Figs. 417, 418) and the operations for symblepharon (Figs. 419, 420).—D.]

824. Division and Excision of the Conjunctiva.—(*a*) *Peritomy* consists in dividing the conjunctival vessels running over the limbus either by simply making an incision round the cornea through the conjunctiva, or by excising a narrow annular strip of the latter [the latter operation is also called *peridectomy* and *syndectomy*]. It is done in order to get rid of a thick pannus [and sometimes also for other conditions (episcleritis, herpes, acne rosacea). In Agnew's operation, which yields good results, the conjunctiva is divided all round close to the limbus, and pushed back for a space of 5 mm.; then the bared episcleral tissue in this zone is scraped clean away, and the larger vessels incised or cauterized (Beard). Panas and others perform peritomy by destroying the vessels round the cornea with the cautery (*igneous peritomy*).—D.]

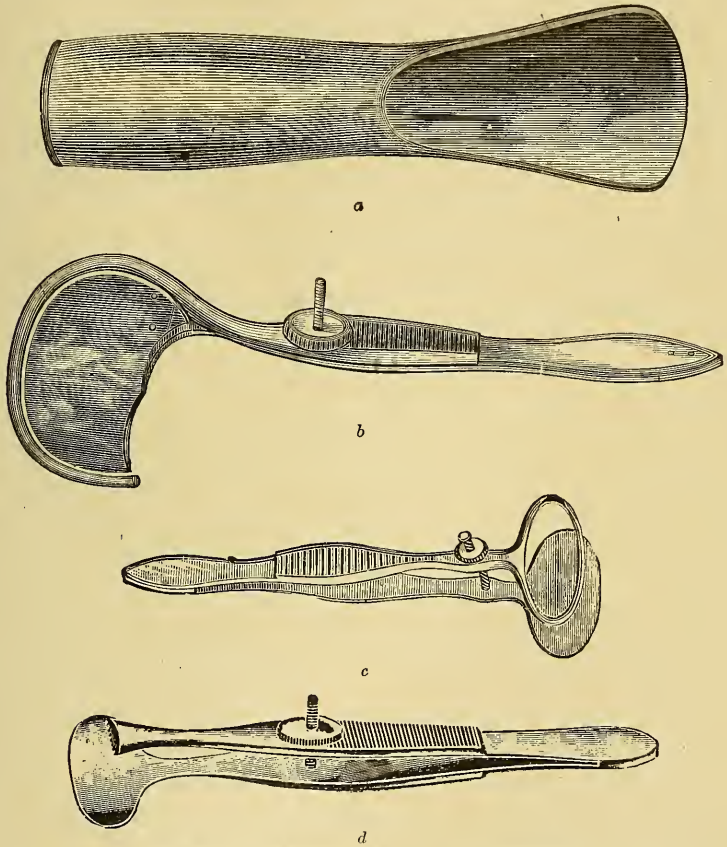
[(*b*) *Excision of the Retrotarsal Fold.*—This operation, advised by von Blascovicz, is suitable for cases of trachoma in which the retrotarsal fold is markedly infiltrated, but the tarsus is not involved. The upper lid is everted and the convex margin of the tarsus is grasped with two pairs of forceps An incision is made in the healthy conjunctiva scleræ, extending from the outer to the inner canthus and close to the line of demarcation between the healthy conjunctiva and the diseased fold. Several sutures are passed through the retracted margin of the scleral conjunctiva, and the latter is undermined. A second incision is made separating the retrotarsal fold from the tarsus, and the fold is then dissected off and removed. The sutures that have been already placed in the scleral conjunctiva are then passed through the upper edge of the tarsus and are tied (Römer). This operation may be combined with excision of the tarsus (see page 950).—D.]

CHAPTER III

OPERATIONS ON THE LIDS

I. Removal of Growths on the Lids

825. Operation for Chalazion.—For this the lid is everted, and the conjunctiva and the wall of the chalazion lying beneath it are opened by an incision with a sharp-pointed scapel. After the fluid portion of the contents has escaped, the granulation masses which still remain are removed



[Fig. 422.—Blepharostats for Lid Operations

a, Jäger's horn plate. *b*, Knapp's lid-clamp. *c*, Desmarre's clamp. The plate in *a*, *b* and *c* is inserted beneath the lid thus affording a resisting surface upon which to cut in all operations upon the lids (operations for trichiasis, entropion, ectropion, ptosis, chalazion, etc.). *b* and *c*, which have a ring that can be screwed down upon the lid, also act as clamps holding the lid firmly in place and preventing hæmorrhage during the operation. Smaller clamps used specially for chalazia are made on the same principle. *d* Ehrhardt's forceps for double eversion of upper lid in resecting the tarsus and removing the accessory lachrymal gland.—D.]

by scraping (with a small, sharp spoon, or with a Daviel's scoop). Even then the tumor does not disappear completely, because its resistant capsule remains, although this shrinks up after a short time. If the contents of the chalazion are not completely removed, it is apt to form again, so that

the operation has to be repeated. [Extirpation (see below) obviates this disadvantage, and is hence preferred to incision by many including the translator. In operating for chalazion it is usually advantageous to apply a lid clamp (see Fig. 422, *b* and *c*) or a smaller chalazion clamp to stop the bleeding which is generally free enough to interfere with the proper performance of the operation.—D.]

If we desire to make the operation of curretting a chalazion absolutely painless it is not sufficient to drop cocaine into the conjunctival sac, but we must also inject a little cocaine into the very substance of the chalazion.

Instead of merely opening the chalazia, we may *extirpate* them by dividing the skin over them and then cutting them out of the tarsus. A fenestra is thus made in the tarsus and in the conjunctiva as well. [Chalazia, especially of the lower lid, can often advantageously be removed through an incision on the tarsal surface.—D.] Such an extirpation of the chalazia is pretty tedious, for which reason we in most cases prefer the simple incision combined with curretting, and are perfectly successful with it, too. Extirpation is indicated only when we are dealing with large chalazia, which project far forward and have a particularly thick capsule.

826. Removal of Lid Tumors.—For vascular tumors we employ *electrolysis*. The positive pole of a constant-current battery under the form of a plate electrode is placed upon the temple, and then the current is passed through the circuit. The negative pole is armed with a needle which is plunged into the tumor. The current is now turned on, and soon bubbles of gas begin to escape along the needle out of the puncture made by it. The needle is then withdrawn, and the same procedure is repeated at another point of the tumor. Owing to the decomposition of the tissue fluids produced by the galvanic current, the blood coagulates in the vessels, which consequently become obliterated. Several sittings are always required for the complete removal of the tumor.

[Other tumors we extirpate according to the usual surgical rules, being careful to remove all the growth and being particularly careful not to cut into cysts. In removing the growth we spare the lid margin if we can.—D.] In removing malignant tumors we must keep at least a centimetre outside of the visible or palpebral border of the neoplasm. If we have to sacrifice so much of the lid that the eyeball remains uncovered, a substitute for the palpebral skin that has been destroyed must be gotten by blepharoplasty (page 959) done immediately after the removal of the tumor. In extensive tumors it is necessary often to remove the eyeball (page 1026), and even the entire contents of the orbit (page 1029).

II. Operations for Trichiasis

827. The number of operative methods proposed for the relief of trichiasis and distichiasis is extremely large. Many of the procedures advocated are, however, distinguished only by trifling details from each other, so that it is sufficient to describe at length only some few of the methods which may be regarded as constituting the main types of operation.

Of any good trichiasis operation it must be demanded that it relieve the faulty position of the cilia and prevent a return to this position (a relapse). Circumstances being the same, preference will be given to that method which attains this result with the least amount of disfigurement. The obvious procedure for surgeons first to hit upon consisted in simply removing that part of the lid which bears the cilia (ablation of the zone of hair follicles). But since the results of this method of operating leave much to be desired, it was so modified that the zone of hair follicles was not removed, but simply displaced in such a way that the cilia took on the direction desired (transplantation of the zone of hair follicles). By these methods the trouble is removed, but without its cause—namely, the distortion of the tarsus—being done away with. Hence, still others conceived the idea of curing trichiasis by giving the distorted tarsus its normal shape again (straightening of the tarsus). Upon some one of these principles depend most of the known operations for trichiasis.

828. (1) *Electrolysis.*—[Instead of removing a portion of the lid, we may simply remove the cilia by electrolysis.—D.]

The two poles of a constant-current battery are so arranged that the positive pole is formed by a plate electrode, the negative pole by a slender needle. The former is applied to the temple, and the latter is introduced into the hair follicles of the cilium, and then the circuit is closed. At once a light foam is seen to exude from the root of the cilium. This is formed by the bubbles of hydrogen gas which are developed at the negative pole and give evidence of the chemical decomposition of the tissue fluids produced by the electric current. By virtue of this decomposition an adequate destruction of the hair follicle results without any eschar being produced. The cilium can now be very readily drawn out, or it falls out afterward of itself, and it never grows again. The operation is pretty painful, and hence it is a good plan to inject some cocaine solution beneath the skin of the lid near its free border. [It is used especially when there are only a few misplaced cilia that require removal.—D.]

829. (2) *Ablation of the Zone of Hair Follicles by Flarer's Method.*—During the operation some firm support upon which the cutting can be done must be placed beneath the lid. For this purpose a lamina of horn is employed, which is pushed beneath the lid, the lamina being either in the simple form of Jäger's horn plate (Fig. 422, *a*) or in the complicated form of Knapp's blepharostat [lid clamp] (Fig. 422, *b*), in which the lid is kept pressed against a horn plate by means of a metal ring. The lid must be similarly fixed in the other methods of operating for trichiasis. After inserting the horn plate an incision is made with the lance knife (or with a scalpel) in the intermarginal space, and in fact in that gray line which separates the orifices of the Meibomian glands from the roots of the cilia (*i*, Fig. 284). When we make the incision here we get into the loose connective tissue

which lies between the tarsus and the muscular fibers of the orbicularis, and which is readily divided. We thus split the lid into two laminæ, the anterior of which contains the skin with the cilia, and the posterior the tarsus with the conjunctiva. This process of cleavage must run along the whole length of the edge of the lid and be carried inward to a point over the roots of the cilia—i. e., to a distance of about 3 mm. from the free edge of the lid. When the zone of hair follicles is thus detached from the tissues beneath it, we now need only separate it from its connection with the skin of the lid. This is done by an incision carried through the skin parallel with the edge of the lid and situated at the limits of the zone of hair follicles. This latter is then connected with the skin of the lid at its two extremities. This connection being now divided with the scissors, the zone of hair follicles (the portion *a*, bounded by the dotted line in Fig. 423) is detached. A raw surface now remains along the border of the lids, the floor of which is formed by the anterior surface of the denuded tarsus. This wound heals by granulation within a few days.

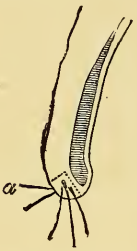


FIG. 423. — ABLATION OF THE BED OF HAIR FOLLICLES BY FLARER'S METHOD. Magnified 2 × 1.

This is one of the oldest methods of operating on trichiasis. It has the advantage of simplicity, and, moreover, when nothing has been left, makes all relapses impossible; but it produces a permanent disfigurement due to the removal of the cilia, and deprives the eye of the protection which the cilia afford. This is of particular importance in the upper lid, where the cilia are more numerous and are larger. Moreover, the hard cicatrix which is formed at the site of the ablated zone of hair follicles is often a cause of renewed irritation of the eye. Hence, ablation of the zone of hair follicles is at present but rarely performed, being almost entirely replaced by the transplantation methods (§§ 830 et seq.). It is employed upon the lower lid, where the cilia in any case are small and scanty, and particularly in cases of partial trichiasis in which ablation need be done only over a short extent of surface.

830. (3) *Displacement (Transplantation) of the Zone of Hair Follicles by Jaesche-Arlt's Method.*—In the upper lid the operation is performed by beginning, as in the case of ablation of the hair follicles, with an incision in the intermarginal space, which splits the lid into two laminæ as far as the upper limit of the bed of hair follicles (*m*, Fig. 424 A). In this way the bed of hair follicles is detached from the underlying tissue and is made movable. In order now to draw this zone up and attach it at a higher point, the skin of the lid is shortened in the vertical direction by excising a fold of it. The fold to be excised is bounded by two incisions. One runs 3 or 4 mm. above the free border of the lid and parallel with it; the other is carried above the first along a curved line, so that at its center it is farthest (6 to 8 mm.) from the first incision, but at its extremities coincides with it (Fig. 424 C). In this way an elliptical piece of skin is circumscribed, which then is dissected from the underlying tissues with the scissors, care being taken

to preserve the subjacent muscular fibers. The two lips of the wound being then united by a number of sutures, which are applied in a vertical direction (*s*, Fig. 424, B), the lower lip of the wound, together with the zone of hair follicles, is drawn well up, and the cilia are in this way directed straight. At the same time the incision in the intermarginal space gapes open and displays at its bottom the raw anterior surface of the tarsus. In order that the bed of hair follicles may not be drawn down again by the cicatrization of this wound, the piece that has been excised from the skin of the lid is inserted into the wound so as to be implanted there. This piece must be trimmed down so as to fit well into the wound (*h*, Fig. 424, B). If care is

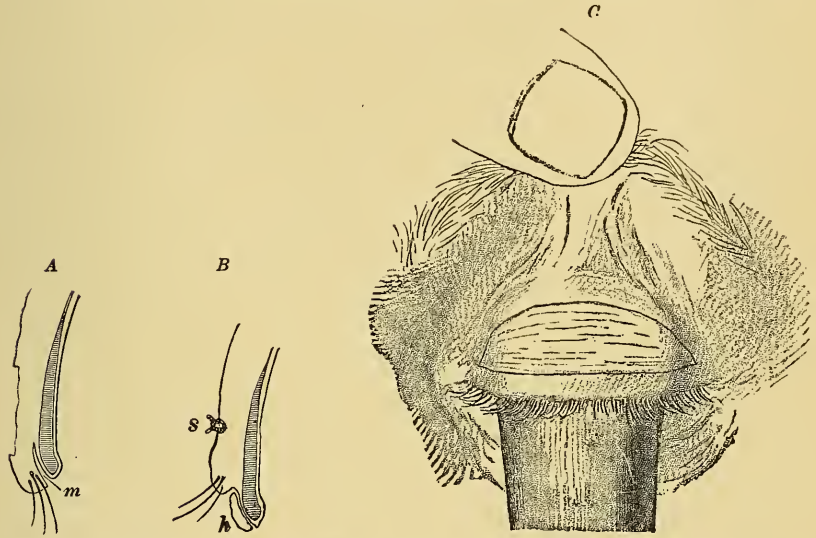


FIG. 424.—TRANSPLANTATION OF THE BED OF HAIR FOLLICLES BY JAESCHE-ARLT'S METHOD.

A, cutaneous incision and incision in the intermarginal line, *m*. Magnified 2 × 1. B, operation after tying the cutaneous sutures, *s*, and implanting the piece of skin, *h*. Magnified 2 × 1. C, front view after the excision of the skin has been made and with the horn plate inserted. Natural size.

taken by proper bandaging of the eye to keep the transplanted strip of skin pressed against the raw surface forming the bed of the wound, it almost always becomes well attached to the latter in healing.

In performing this operation the mistake is frequently made of excising too broad a piece of skin from the cutaneous surface of the lid, which thus becomes so much shortened that ectropion or lagophthalmus develops. We should therefore, before the operation, determine the size of the piece to be exsected, by pinching up a fold of skin with the finger and seeing how large this must be in order to effect its object without shortening too greatly the skin of the lids.

In the lower lid the operation is performed in the same way, except that here, in order not to get ectropion, the piece of skin to be exsected must be made still narrower.

The most important step in the way of improving the operations for trichiasis was made by Arlt, who devised the plan of detaching the bed of hair follicles from the tarsus. In this way one was enabled to do an extensive transplantation without having to fear a necrosis of the bed of hair follicles, as in the original method of Jaesche, who released the bed of hair follicles, along with the subjacent tarsus, from end to end, and separated both completely from the underlying tissues. Even Arlt's method, however, is not free from disadvantages. One of these consists in the difficulty of gauging accurately the size of the piece to be exsected; another, in the possibility of a relapse. As regards the first point, no fixed breadth can be assigned for the strip to be removed, because this breadth varies according to the character (i. e., the elasticity or laxity) of the skin of the lids. If too little is exsected, the zone of hair follicles is not drawn up sufficiently and the trichiasis returns; if too much skin has been removed, we get ectropion or lagophthalmus, which could only be got rid of by subsequent operations. For this reason methods have been devised to effect the elevation of the zone of hair follicles without excising any skin. Hotz's operation (§ 831) belongs in this category.

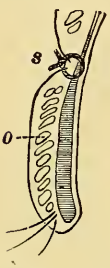


Fig. 425. — Hotz's Operation.

831. 4. *Drawing up the Bed of Hair Follicles by Hotz's Method.*[1]—When this operation is made upon the upper lid, an incision is first carried through the skin of the latter, passing along the upper border of the tarsus from one end of it to the other. Then the lips of the wound are separated, and the bundles of fibers of the orbicularis (Fig. 425, *o*) which are visible at its bottom are exsected. The wound is next closed and at the same time its lower lip is stitched to the upper border of the tarsus. For this purpose the needle is passed first through the upper lip of the cutaneous wound, then through the upper border of the tarsus, and lastly from within outward through the lower lip of the cutaneous wound (*s*, Fig. 425). As many of these sutures are applied as necessary. The idea underlying this operation is to raise the bed of hair follicles, not by shortening the skin of the lids, as in Jaesche-Arlt's method, but by attaching the skin to a fixed point—namely, the upper border of the tarsus. The exsection of the fibers of the orbicularis is done with the intention of diminishing the power of this muscle which tends to force the lid backward. This operation dispenses with the step of detaching the bed of hair follicles by an incision in the intermarginal space; but it may, if necessary, be combined with such an incision.

In the lower lid the method of operating is the same, but the cutaneous incision, in accordance with the smaller height of the tarsus, runs closer to the free border of the lid.

A somewhat different method is that of *Oettingen*. He carries the incision in the intermarginal space to a point above the upper border of the tarsus, so that all the skin which covers the tarsus can be displaced upward. This portion of skin is then, by means of sutures, attached near its free border which carries the cilia, to the upper border of the tarsus (Fig. 426). Below the free border of the lid, which has been

[1] [As Hotz says, first proposed by Anagnostakis.—D.]

thus elevated, there remains quite a large wound, formed by the anterior surface of the tarsus. Upon the same principle depend the methods of Kostomyris, De Wecker, and Warlomont.

832. 5. *Straightening of the Tarsus by Snellen's Method.*—This object is sought to be obtained by the excision of a prismatic piece from the tarsus. The skin is incised about 2 mm. above the border of the lid, and in a direction parallel to the latter along the entire length of the lid (*i*, Fig. 427 A). Then the lowermost bundles of fibers of the orbicularis which lie exposed in the wound are excised, so that the tarsus is presented to view. A prismatic piece is now exsected from this latter along its entire length, and in such a way that the base of the wedge corresponds to the anterior, the apex of the wedge to the posterior surface of the tarsus (*e*, Fig. 427 A). The next thing to do is to bring the two cut surfaces of the tarsus into contact. This is accomplished by sutures applied under the form of loops by means of double-armed threads. One needle is first passed through the upper border of the tarsus (*a*, Fig. 427 B), and is then carried down in front of the wound in the cartilage and between the tarsus and the skin as far as the free border of the lid, above which it is brought out again. A similar manœuvre is performed with the other needle. The loop then lies upon the upper extremity of the tarsus, while the two ends of the thread come to view above the border of the lid. These ends are then tied upon a bead (*p*), and are afterward carried up to the forehead, where they are attached with plaster above the eyebrow. In this way the lid is kept drawn up, and coaptation of the cutaneous wound is rendered superfluous, as this then closes of itself.

FIG. 426.—OETTINGEN'S OPERATION. Magnified 2 × 1.

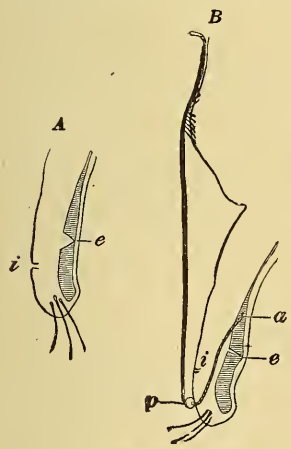


FIG. 427.—SNELLEN'S OPERATION. Magnified 2 × 1.
A, after completing the cutaneous incision, *i*, and the exsection of the tarsus, *e*. B, after applying the suture.

Snellen's operation was modified by *Panas* in the following manner: He divides the skin of the lid 2 or 3 mm. above the free border of the lid and parallel to the latter (*i*, Fig. 428 A), the incision running the whole length of the lid. Then, beginning at this incision, he dissects up the skin as far down as the free border of the lid and as far up as the upper border of the tarsus. Next, cutting down upon the horn plate, he makes an incision which runs in the same way as the incision in the skin, and divides the tarsus through its entire thickness, including the conjunctiva tarsi. By this incision the lower half of the tarsus, together with the free border of the lid, is made freely movable, and by means of sutures can readily be rotated forward far enough for the cilia to be properly directed. The sutures are formed into loops and passed above through the

edge of the tarsus and the tarso-orbital fascia, and the free ends of the loop are brought out behind the skin of the lid along the intermarginal line, and here are tied over a glass bead (Fig. 428 B).

833. [Excision of Tarsus (Tarsectomy).—This is done not only to relieve trichiasis, but also to effect a radical cure of its underlying cause, trachoma. Two operations are done.

(*a*) *Partial Tarsectomy with Excision of the Retrotarsal Folds.*—As performed by von Blaskovicz, this operation is advised in trachoma with marked infiltration of both the tarsus and retrotarsal folds. The upper lid is everted and an incision parallel to the lid border is made from outer to inner canthus through the healthy scleral conjunctiva demarcating it from the diseased fold. Three sutures are introduced through the lower lip of the conjunctival wound in such a way that each forms a loop 2 mm. broad, with its bight on the epithelial surface of the conjunctiva. Traction is made with these, and the scleral conjunctiva is dissected far down (nearly or quite to the cornea—Orcutt). An incision is then made through tarsal conjunctiva and tarsus, parallel with the free border of the lid and 2.5 or 3 mm. from it, so as to delimit all the diseased portions of tarsus and conjunctiva. These diseased portions are dissected out, care being taken not to injure the orbicularis or Müller's muscle. The piece thus removed measures about 2.5 cm. long and 1 cm. broad. The conjunctiva is then drawn up by the looped sutures, which are passed through the lid from within outward, entering the stump of the tarsus 1 mm. in front of its cut edge, and emerging on the skin surface where they are loosely tied (Römer).—D.]

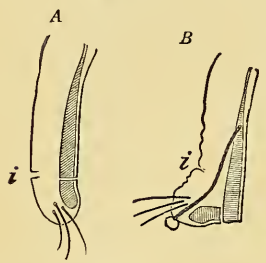


Fig. 428.—Panas's Operation.

A, after completing the incisions in the skin and tarsus. B, after applying the suture.

[For Gillet de Grandmont's tarsectomy see page 963.]

[(*b*) *Total Tarsectomy.*—This operation, devised by Heisrath, has been modified by Kuhnt. It is used in the cicatricial stage of trachoma, when the tarsus is degenerated and incurved and the conjunctiva is wholly or in large part cicatricial. The lid is everted, and an incision made through conjunctiva and tarsus close to the free border of the lid and parallel with it. The cut segment of tarsus is then dissected from its attachments and particularly from the conjunctiva, which is no longer tightly adherent to it (see page 169). The dissection is carried as far as the upper border of the tarsus. The insertion of the levator is then divided, and the tarsus, being now free from attachments, is removed. The cut edge of conjunctiva is sutured to the rim of tarsus left at the free border of the lid (Römer).—D.]

834. Other Methods. Advantages of Each.—The *relapses* which are of frequent occurrence after Arlt's operation, as well as after many other methods, have the fol-

lowing causes: 1. If the trachomatous process has not completely run its course, the shrinking of the conjunctiva and of the tarsus continues to progress after the operation, and once more throws the cilia into a faulty position. 2. The shortening of the skin produced by excising a strip of it often fails to be permanent, because the skin, especially in old persons, gradually stretches out again. 3. The wound in the intermarginal space, which remains uncovered, heals by granulation and the formation of cicatrices. By the gradual contraction of the cicatricial tissue the free border of the lid may be drawn down again and the position of the cilia be made worse. The first point has its basis in the nature of trachoma, and can not be charged to the account of the method of operating; but the second and third points constitute defects in the operation, which it has been sought to remedy. In order to make the contraction of the skin permanent, the latter has been attached to a fixed point—namely, the convex border of the tarsus (Hotz). The main cause of relapses—i. e., the depression of the bed of the hair follicles due to the cicatrization of the wound—it has been attempted to get rid of by covering the wound with skin. In Arlt's method this object may be attained, as has been described above, by transplanting the excised portion of skin upon the wound (Waldhauer). But as this piece of skin is covered with fine lanugo hairs, which may cause renewed irritation of the eye, *Van Millingen* prefers to cover the wound with mucous membrane which he takes from the lips of the patient, or from the conjunctiva of a rabbit. Since flaps of skin or mucous membrane, when destitute of a pedicle, are exposed to the danger of mortification, and in any case shrink very greatly, others have chosen pediculate skin flaps for covering the wound. In *Spencer Watson's method* this flap is formed in the following way: An incision is made in the intermarginal space, and a second one is made parallel to the border of the lids and above the row of cilia, as is done for their ablation (Fig. 429, in the outer half of the upper lid). Then, instead of detaching the strip of skin, thus marked out, at both ends, as in the operation of ablation, it is separated at one extremity only. The bed of hair follicles is thus transformed into a long, slender flap which is free at one extremity, but at the other is connected with the skin of the lids (*a*, Fig. 429). Then a second flap of skin similar in shape is fashioned by making a second incision about 3 mm. above the first and parallel to it, and thus marking out a narrow strip of skin, which is likewise left in connection with the skin of the lid by one extremity only (Fig. 429, *b*). The base of this flap must lie at the temporal end of the lid, provided the base of the lower flap, which bears the cilia, is situated at the nasal end, and vice versa. Then the two flaps are interchanged, so that the one which carries the cilia gets to lie above, and the one that was above gets to lie below, along the free border of the lid (Fig. 429, a_1 and b_1). The flaps are kept in their place by sutures. This operation is best adapted for those cases in which the trichiasis is present simply at one or the other extremity of the row of cilia, and in which, therefore, a short flap is sufficient (Fig. 429). If Spencer Watson's operation is performed along the whole length of the lid, it has the disadvantage that the flaps have a very great length in proportion to their narrow base, and hence are apt to die.

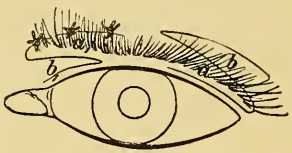


Fig. 429.—Spencer Watson's Operation for Trichiasis.

This is represented as being performed in such a way that the flaps do not extend over the entire length of the lid, but only over one-half of it. The outer half of the upper lid shows the flaps in their natural position; the inner half shows the position of the flaps after they have been interchanged.

Gayet, Jacobson, Dianoux, and Landolt likewise employ flaps with a pedicle.

Each of the methods named has certain *advantages* and disadvantages. The experienced operator will not employ any one of them exclusively, but will select the method to suit the case in hand, and when necessary will even combine two different

methods. Thus Hotz's method may be combined with the excision of a strip of skin, with the detachment of the bed of hair follicles by an intermarginal incision, or, if the distortion of the tarsus is a very conspicuous feature, by the excision of a wedge from the latter. In cases in which the trichiasis is greatest in the center Arlt's method is very suitable, since in this method the exsected piece of skin is broadest in the center, and hence it is in this place that the greatest displacement is produced in the bed of hair follicles. The methods of Hotz, Snellen, and Panas also exert their greatest effect in the center of the lid. For trichiasis at one end of the row of cilia implantation of a skin flap with a pedicle—e. g., by Spencer Watson's method—is the most suitable procedure.

III. Canthoplasty (Canthotomy)

835. The object of canthoplasty (Von Ammon) is to dilate the palpebral fissure by dividing the outer commissure. The two lids are separated widely with the fingers and drawn toward the nose, so that the outer commissure is put upon the stretch, and the blunt-pointed blade of a straight pair of scissors is introduced behind it as far as possible; then, with one snip the skin lying between the blades of the scissors is divided in a horizontal direction; then, upon drawing the lids apart, a rhomboidal wound is exposed to view (Fig. 430). The two outer sides of this wound lie in the skin, the

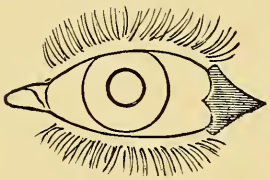


Fig. 430.

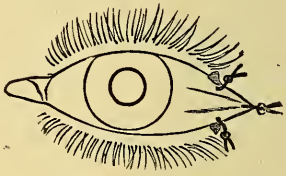


Fig. 431.

two inner sides in the conjunctiva. In order to make the sides of the commissure draw far enough apart, [we divide the canthal ligament. To do this we seize the conjunctival border of the wound with the forceps and drawing on it until the resistance of the fibers (canthal ligament) is distinctly felt, then pass the closed blades of the scissors into the wound, feel for the ligament and, when it is found, open the scissors blades so as just to include and divide it (Hotz).—D]. The inner sides of the wound are next stitched to the outer by grasping the conjunctiva at the spot where the two borders of the wound come together, and attaching this point to the external angle of the wound by a suture. Then an additional suture, uniting skin and conjunctiva, is passed through the upper and the lower portions of the wound respectively (Fig. 431).

If the wound in the external angle of the eye were not covered with the conjunctiva thus stitched to it, it would reunite within a short time. Hence, if all that we need is a temporary dilatation of the palpebral fissure, we satisfy ourselves with splitting the external commissure without applying any subsequent suture—temporary canthoplasty.

In trachoma it is often difficult, owing to the shrinking of the conjunctiva, to draw the latter sufficiently into the skin wound and sew it there. In this case we follow Kuhnt's method and take instead of conjunctiva a narrow *cutaneous flap* which is fashioned from the skin of the lower lid. The flap is cut parallel to the free border of the lid and close to the latter in such a way that its base lies to the outer side of the commissure of the lids so that the flap can be placed in the split commissure without being turned.

The *indications* for canthoplasty are: 1. *Blepharophimosis* and *ankyloblepharon*. In this case a permanent effect is desired from the operation, and this is therefore made with a conjunctival suture.

2. *Blepharospasm*, especially if it gives rise to spastic entropion. Here temporary canthoplasty is sufficient. The success of the operation in these cases is attributable not only to the lengthening of the palpebral fissure, but also and mainly to the division of the fibers of the orbicularis, in consequence of which the latter suffer a considerable impairment of strength. If, as is so frequently the case, the spastic entropion is combined with blepharophimosis, the canthoplasty must be combined with suture.

3. *Gonorrhœal conjunctivitis*, when the lids are extremely swollen and exert a considerable pressure upon the eye. In this case temporary canthoplasty is sufficient. The same thing is true when the enlargement of the palpebral fissure serves as—

4. *A preliminary step* to enable us to remove through the palpebral fissure an eyeball which is very much increased in size, or an orbital tumor.

IV. Tarsorrhaphy

836. Tarsorrhaphy consists in shortening the palpebral fissure by uniting the edges of the lids; it is accordingly the direct opposite of canthoplasty. The union of the edges of the palpebral fissure may be effected either at the outer or at the inner angle of the eye (tarsorrhaphia lateralis and medialis).

1. *Lateral tarsorrhaphy.*—In Von Walther's method this is performed by paring the upper and lower borders of the lids by ablation of the bed of hair follicles over the space adjoining the external angle of the eye, and then stitching the borders of the lids to each other along the denuded area. As in this way it is only a very narrow raw surface—i. e., the raw edges of the lids—over which union is effected, the wound is apt to tear apart under any considerable amount of strain. Hence in these cases I perform the operation in a different way. First, the extent to which it is desired to join the lids together is marked out; then to the same extent the lower lid is split into its two laminæ by an intermarginal incision. From the inner extremity of the section a short incision is carried downward through the skin, thus converting the anterior lamina of the bisected portion of the lid into a flap (*a*, Fig. 432). The upper and inner borders of this flap are free, while the lower and outer borders are connected with the skin of the lid. The follicles of the cilia, which lie exposed along the posterior border of the

upper end of the flap, are removed by a scissors applied flatwise—this being done so that the cilia may afterward fall out. Then the upper lid is denuded by first making the intermarginal section in the same way as upon the lower lid and then ablating the bed of hair follicles thus detached, as in Flarer's operation. There is thus produced a raw surface (*b*, Fig. 432), to which it is intended that the skin flap of the lower lid shall adhere by its raw surface. In order that adhesion of the raw surfaces themselves, and not simply of their edges, shall take place, the suture is applied as follows: Both ends of a thread armed with a needle at each end are carried through the upper lid near its free border, the needles being passed from behind forward. In this way the loop of the thread gets to lie upon the conjunctival side of the lid, while the free ends come out upon the raw anterior surface. These ends are then passed through the base of the skin flap below and are tied upon its anterior aspect over a glass bead. By this suture the base of the flap is kept pressed against the raw surface of the tarsus of the upper lid; then, as an additional precaution, the edges of the skin flap are accurately united to the edge of the wound in the upper lid by means of a few fine sutures. The adhesion of the lids obtained by this method is firm enough to withstand even a powerful strain.

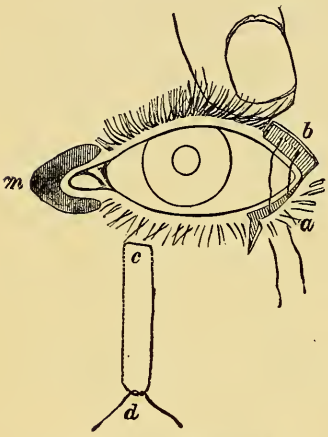


Fig. 432.—Tarsorrhaphy.
A lateral tarsorrhaphy is represented at the external angle of the eye; a median tarsorrhaphy at the internal angle. On the lower lid is shown the position of the loop of thread in Gaillard's suture.

2. *Median tarsorrhaphy*, as devised by Arlt, is performed by excising with the forceps and scissors a narrow strip of skin from both the lower and the upper lid close to their inner angles. The long and slender wounds thus produced should meet in an acute angle at a point on the inside of the inner commissure (*m*, Fig. 432). They are then united to each other through their entire extent by means of interrupted sutures. If a firmer adhesion is desired, this operation, too, like external tarsorrhaphy, may be made with the formation of a small skin flap.

Tarsorrhaphy is indicated—

1. In *ectropion*.—In this the lower lid is raised by attaching it to the upper. Tarsorrhaphy is most effective in senile and paralytic ectropion, and also in slight cases of cicatricial ectropion. Tarsorrhaphy is very often performed, in conjunction with blepharoplasty, in order to insure the lids remaining in the proper position.

2. In *lagophthalmus*, because the closure of the palpebral fissure is facilitated by shortening the latter. The form of lagophthalmus that most

often gives occasion for the performance of tarsorrhaphy is that which develops in Basedow's disease in consequence of the exophthalmus, inasmuch as here we have no other means of relieving it.

As a rule, lateral tarsorrhaphy is the operation which is done. Median tarsorrhaphy is scarcely ever employed except in paralytic ectropion, and is done then only because in this condition the lower lid generally droops more in its inner than in its outer half.

When the lower lid has been in a condition of ectropion for a pretty long time, it is ordinarily found to have become elongated by the stretching it has undergone. In order to make the lid short again, a procedure by which it is at the same time put upon the stretch and pressed against the eyeball, the lower lid, in the performance of tarsorrhaphy, is pared to a greater extent than is the upper. When the elongation is particularly marked, the lid is shortened by excising a triangular piece at its outer extremity. The apex of the triangle is directed downward, and its base corresponds to the free border of the lid. The two sides of the triangular notch are united by means of sutures.

The operation of uniting the lids by means of tarsorrhaphy is sometimes done under great tension—e. g., when the attempt is made to bring closer together lids that have become shortened, or when the operation is done in a case of exophthalmus. In the latter event it is the enlarged eyeball that tends to push the lids apart. To diminish the tension, that portion of the palpebral fissure which is to remain open may also be closed by means of sutures, this being done without paring the edges of the lids, so that the union shall be only a provisional one. The sutures are left in until they cut their way through, or until the healing of the wound made by the tarsorrhaphy has become sufficiently firm.

V. Operations for Entropion

837. *Spastic entropion* develops only when the skin of the lids is abundant and relaxed (see page 679). If then the skin is put upon the stretch by pinching up a horizontal fold of it in the fingers, the entropion disappears. Upon this observation depend those methods of operating for entropion which produce contraction of the skin of the lid in a vertical direction. The methods of operating that are most in use are:

1. *Gaillard's Suture.*—This operation as modified by Arlt is performed as follows: One needle of a double-armed thread is entered at the junction of the middle and inner thirds of the lower lid. The point of entry lies close to the border of the lid (*c*, Fig. 433 B), the point of exit at a distance below it upon the cheek equal to about the breadth of the thumb (*d*). The second needle is passed in a similar fashion and near the first, so that the bight of the thread lies upon the skin near the border of the lid (Fig. 433 A), and the two threads run downward beneath the skin of the lids in a parallel direction. A similar loop of thread is applied at the junction of the middle and outer thirds of the lid. If the two ends of each thread are tied over a small roll of adhesive plaster or of iodoform gauze and drawn tight, a horizontal fold of skin is pinched up upon the lower lid (*a*, Fig. 433, C), and thus

the entropion is made to disappear. In order to produce a sufficient effect, the threads are drawn tight enough to produce a slight degree of ectropion, which subsequently disappears. The threads should be left in until cicatricial bands, which shall produce the same effect that the threads did, form along the channels made by the threads. The entropion is apt to set in again subsequently in spite of the operation. The latter, accordingly, is adapted to those cases only in which we are dealing with an entropion of presumably short duration, as, for example, that form which develops beneath a bandage.

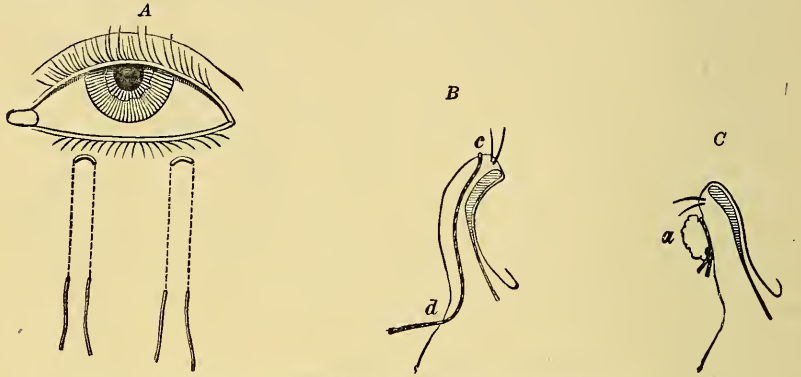


FIG. 433.—GAILLARD-ARLT'S OPERATION FOR ENTROPION.

A, suture seen from in front. B, seen in vertical section; situation of the loop. C, the same after the loop has been drawn tight. B and C magnified 2 × 1.

Instead of Gaillard's suture we may also employ one of Snellen's (not to be confounded with Snellen's suture for ectropion). This was modified by Stellwag in the following way: "One or two stout threads armed with curved needles at both ends are passed through the entire thickness of the lid, starting from the deepest portion of the retrotarsal fold of the conjunctiva (*a*, Fig. 434), so that they form at the fundus of the conjunctival sac one or two loops 4 to 5 mm. in length and placed parallel to the free border of the lid. Each needle is next carried back through its point of exit in the external covering of the lid (*b*), then is passed vertically between the latter and the fibro-cartilage to a point just at the external lip of the lid, there (*c*) is brought out again, and the two ends of each thread are drawn as tight as necessary over a roll of adhesive plaster and tied."

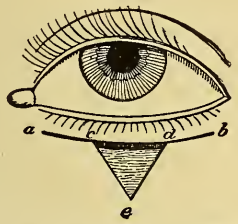
FIG. 434.

2. *Excision of a Horizontal Strip of Skin.*—A horizontal fold of skin is pinched up between two fingers, its size being so gauged that the entropion shall be relieved by it without, however, an ectropion or a lagophthalmus being produced; then this fold is snipped off with one stroke of the scissors, and the two edges of the wound are next united by means of some sutures. If the operation is to be effectual, the upper edge of the wound must lie pretty close to the free border of the lid. The result of this operation is ordinarily permanent, although here, too, recurrences of the ectropion some-

times take place through subsequent stretching of the skin. Hence the much more complicated Hotz's operation (see page 948), which fastens the skin of the lids to a fixed point, the attached border of the tarsus, is also employed in entropion.

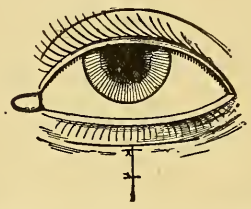
3. *Excision of a Vertical Strip of Skin.*—If we push backward upon the entropionized lid in the region of the orbital margin, the lid goes back into place. We make use of this fact when we try to keep the lid in the proper position by means of an appropriately placed bandage (page 680); but we can also secure a permanent effect of this kind by shortening the skin of the lid at the margin of the orbit in a horizontal direction, so that it becomes tense and presses the lid backward. We first make an incision *ab* (Fig. 435) parallel to the lid and then from the extremities of the middle third of this incision make the converging incisions *ce* and *de* downward as far as the margin of the orbit. The piece so delimited is cut out, and then the two lateral borders of the incision, after the connections have been loosened up

Fig. 435



Operation for entropion by Von Graefe's method. Way of making the incision.

Fig. 436



The same after the edges of the wound have been united.

sufficiently, are united in a vertical direction (Fig. 436; Von Graefe). The scars resulting from this operation are rather more visible than those resulting from the excision of a horizontal strip of skin, because they run perpendicular to the direction of the cutaneous folds; but, on the other hand, this operation affords more security against relapses.

4. *Canthoplasty*, since it relieves blepharospasm, may also be employed for the abrogation of spastic entropion. In those cases in which blepharophimosis is present with the entropion, the canthoplasty must be made with a conjunctival suture; in other cases a temporary canthoplasty (page 952) often suffices.

In *cicatricial entropion*, due to shrinking of the conjunctiva and the tarsus, those methods of operating are indicated which are employed for trichiasis, since cicatricial entropion may, as far as its origin is concerned, be regarded as nothing but a trichiasis that has become far advanced.

VI. Operations for Ectropion

838. Spastic Ectropion.—In spastic ectropion, which does not yield to reposition and the application of a bandage, *Snellen's suture* gives the best service. Like Gaillard's suture in entropion (page 955), it consists of two

loops, one of which is situated at the junction of the external and middle thirds, the other at the junction of the middle and inner thirds, and which are passed downward (or in the upper lid upward) beneath the skin of the lid. The point of entry, however, is different. In Snellen's suture this lies at the summit of the ectropionized conjunctiva—that is, under ordinary circumstances close to the posterior margin of the tarsus (*a*, Fig. 437 A). From this point the needle is passed down beneath the skin of the lid to a point about at the summit of the lower margin of the orbit, and there is brought out again (*b*). The second needle with the other end of the thread is carried down close to the first and parallel with it. The two extremities of the thread which thus come to view upon the cheek are tied over a roll of adhesive plaster or iodoform gauze and drawn tight until a slight degree of entropion (Fig. 437 B) is produced. The same thing is done with the second loop. The mode of action of the operation depends upon the fact that the protruding portion of the conjunctiva, which is caught in the loop, is drawn downward and forward in the direction of the skin. This suture can also be employed in senile ectropion, although in this case it usually has no permanent effect.

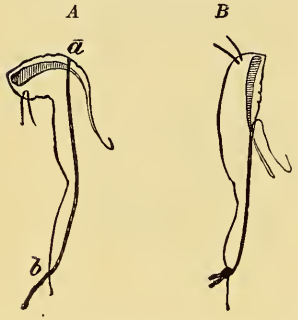


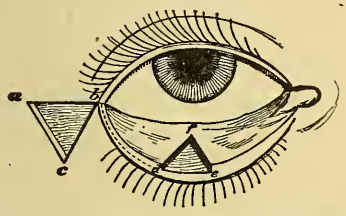
FIG. 437.—SNELLEN'S SUTURE FOR ECTROPION.
A, before tightening the loop. B, after tightening the loop.

839. Paralytic and Senile Ectropion.—*Paralytic ectropion* indicates the performance of *tarsorrhaphy*. The latter is also frequently performed in *senile ectropion*, although in the latter *Kuhnt's operation* gives much better results. This is nothing but a modification of the old method of Antyllus. It depends upon the fact that the lid when ectropionized is elongated, and that, when it has been made stiffer by being shortened, it becomes applied to the eyeball. The shortening is accomplished by cutting out a triangular piece, the base of which must lie at the border of the lid, since here the elongation is most considerable. In order that a coloboma of the lid may not be produced, the excision should not affect the entire thickness of the lid, but only its inner layers —namely, the conjunctiva and tarsus, (see Fig. 438).

The operation as performed by Kuhnt is done by starting from the intermarginal line and dividing the lid at its middle portion into its two laminæ, doing this over as great an extent as that by which we wish to shorten the lid margin (Fig. 438 from *d* to *e*). From the ends of the intermarginal incision we carry two converging incisions backward through conjunctiva and tarsus (to *f*) and excise the triangular piece so delimited, and then unite the edges of the wound in the tarsus with a suture (Fig. 439, *fe*). Since no skin has been cut out, an excess of skin is left so as to form a beak-like projection, after the wound in the cartilage is united. Later on, this

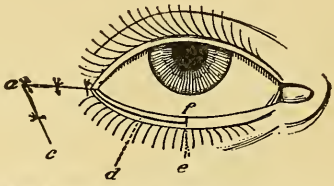
projection smooths down almost completely. If we wish to avoid it, we may follow *Müller's* method and by means of the intermarginal section *edb* (Fig. 438) split the lid in its entire outer half into its two laminæ, and then after exsecting the cartilage and sewing it up again, distribute the excess of skin over the entire half of the lid by applying the stitches obliquely (sewing "on the bias"). Or following Dimmer's proposal we may combine Kuhnt's operation with that of Dieffenbach. The latter consists in excising a triangular piece of skin adjoining the outer commissure (Fig. 438, *abc*) and then by uniting the edges of the wound, producing traction on the lower lid,—an effect which renders this, too, an operation which is effective against ectropion. If we wish to combine Dieffenbach's operation with that of Kuhnt we first split the lid by an incision in the intermarginal line *edb* (Fig. 438) and then cut out the two triangles *edf* and *abc*. Next we displace the cutaneous lamina of the lid to the temporal side, sliding it on the lamina containing the mucous membrane until *ef* gets to lie against *df* and *bc* against *ac*. Then these cut edges are united by stitches (Fig. 439, *ef* and *ac*).

Fig. 438.



Operation by the method of Kuhnt and Dieffenbach. Way of making the incision.

Fig. 439.



The same after the edges of the wound have been united.

[This last operation, which gives excellent results, is also called the Kuhnt-Meller and Kuhnt-Szymanowski operation.—D.]

[Slight cases of senile ectropion may be relieved by the moderate cicatricial contraction produced by a row of *galvano-cautery* punctures made in the tarsus 4 mm. from the lid border and parallel with it, the punctures being 4 mm. apart (Ziegler).—D.]

840. Cicatricial Ectropion. Blepharoplasty.—With respect to cicatricial ectropion, the least serious cases are those in which not much skin has been lost and the contraction of the lid is caused simply by a few cicatricial bands, especially if those draw the lid in against the bone and attach it to the latter—a condition which so frequently remains after caries of the margin of the orbit. In such cases the cicatricial bands may either be *divided* subcutaneously or be cut out altogether, according to their situation, and the cutaneous wound be closed again by sutures. The effect of such an operation is made more thorough and also more lasting by a tarsorrhaphy performed at the same time. If, however, much of the skin of the lid has been lost, we will have no success with this simple operation, but must proceed to the performance of *blepharoplasty*, the object of which is to replace

skin that has been lost. [Blepharoplasty is used not only in cases of cicatricial ectropion, but also to cover large defects produced by operations, e. g., those for removal of tumors of the lids.] It may be done by the implantation of pediculate or non-pediculate flaps.

(*a*) *Pediculate Flaps.*—If we are going to implant a pediculate flap into the lid, the latter must be properly prepared. The cicatricial portion of the lid is first divided by an incision parallel to the free border of the lid, and then the more deeply situated cicatricial bands are also cut through until the lid is freely movable and can be brought into its normal position without any strain being put upon it. Those portions of the cicatricial skin which appear to have but little vitality are excised, in doing which, however, the free border of the lid, wherever it has been preserved, is spared as far as possible; then the lid is brought into the proper position and retained there by being united to the other lid. The union of the two lids should be made a permanent one for about the outer third of the palpebral fissure—i. e., should be combined with paring of the borders of the lids as in the method of tarsorrhaphy—but the inner two-thirds of the palpebral fissure are united simply by means of a temporary suture. After the lid has been thus put in place, the incision which was made for the purpose of releasing it gapes widely so as to form quite a large raw surface, which must be covered with skin. We now proceed to the formation of the flap. For this purpose a great number of methods have been proposed, according to the size and shape of the loss of substance. The methods most employed are those of Fricke and Dieffenbach.

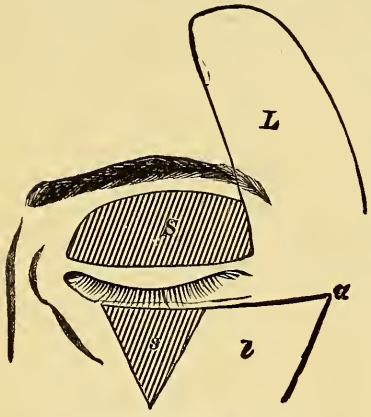


FIG. 440.—BLEPHAROPLASTY. The method of Fricke is presented on the upper lid; that of Dieffenbach on the lower.

Fricke's method is especially adapted for elongated losses of substance, whether upon the upper or the lower lid. To cover these losses of substance a tongue-shaped flap (*L*, Fig. 440) is fashioned, the base of which adjoins one end of the loss of substance (*S*). The flap is most commonly taken from the skin of the temple and the cheek, and in size and shape must be made to fit the loss of substance. In doing this it must be borne in mind that the flap shrinks not only immediately after it has been detached, but also subsequently. It must therefore be made about one-third larger in all its dimensions than the loss of substance. In order that its nourishment may be satisfactory, its base must be made broad enough, and, moreover, should not be twisted too much in the act of transplanting the flap into the wound. For the same reason, the skin should not be dissected off clean, but the sub-

cutaneous fat, together with the vessels running in it, should be taken along with the flap. The flap thus dissected off is placed upon the loss of substance, and is attached by sutures to its edges, which have previously been rendered movable by undermining. The raw surface which remains at the spot where the flap has been dissected off can usually be diminished considerably in size by means of sutures; the remaining surface is allowed to heal by granulation or it is covered with pieces of epidermis by Thiersch's method. At the base of the flap a swelling is formed, due to twisting and larger in proportion to the amount of the twisting. This swelling flattens afterward so as to become less perceptible; should it cause disfigurement, it may be excised later.

Dieffenbach's method becomes applicable whenever the loss of substance either has the form of a triangle (the base of which looks toward the border of the lid), or can be readily brought into the triangular form (*s*, Fig. 440). It is better adapted to the lower than to the upper lid. The flap (*l*) is ordinarily taken from the temporal side of the loss of substance—i. e., from the cheek. An incision is made toward the temporal side along the prolongation of the base of the triangle. It should be somewhat longer than the latter, in consideration of the shrinking which the flap will undergo. From the outer extremity of this section (*a*) a second incision is made downward parallel to the outer side of the triangle. In this way a quadrangular flap is circumscribed, the base of which is situated below. This flap is now detached by dissection, and is turned slightly toward the nasal side so as finally to lie upon the loss of substance, to which it is then attached by sutures. The loss of substance which remains at the spot from which the flap was taken is made as small as possible by means of sutures, and the remainder of it is allowed to heal by granulation.

[The *Wharton-Jones operation* is applicable to scars near the lower lid margin. A V-shaped incision with its apex downward is made so as to include the scar. The triangular piece of skin, thus delimited, which includes the scar tissue, is dissected up to near the lid margin, and the eyelid, being thus released is shoved up into its proper position, carrying the triangle of skin with it. Below this there is now a raw spot shaped like an arrowhead pointing downward. To cover this in, the skin on either side of the V is undermined, and then the lower portions of the V are stitched to each other, and the upper portions are stitched to the displaced triangle of skin. The arrow-shaped wound is thus converted into a Y-shaped scar (Wheeler).—D.]

841. (*b*) *Skin Grafting.*—The raw surface which is exposed after the ectropionized lid has been put into place may also be covered by portions of skin destitute of pedicles—an operation which is called skin-grafting. This was first introduced into surgery as a well-established procedure by Reverdin, although isolated experiments had been made with it before.

Two different methods are distinguished, according to the thickness of the piece of skin that is grafted. In one method very thin pieces are taken which contain only the most superficial layers of the skin—namely, the epidermis, the rete Malpighii, and the apices of the papillæ (*epidermic graft*). In the other method portions of skin are used which include the entire thickness of the cutis, and which are transferred to the loss of substance either cut up into small pieces, or under the form of flaps of some size (*dermic graft*). The skin may also be taken from other persons or from amputated limbs with sound skin. [Dermic grafts are also called *Le Fort* and *Wolfe* grafts from Le Fort who first employed them and Wolfe who popularized their use in blepharoplasty.—D.] A position midway between the epidermic and the dermic method of making grafts is occupied by *Thiersch's method*, in which comparatively large-sized pieces of skin are taken, but which besides epithelium contain only the most superficial layers of the cutis.

With the pieces of skin, which are trimmed to fit the freshly made raw surface, the latter is carefully covered over in such a way that they are everywhere in intimate contact with the subjacent parts, against which they are then kept applied by means of a light pressure bandage.

The method of grafting has the advantage over the formation of skin flaps with a pedicle, that the face is not disfigured by any additional cicatrices. On the other hand, it affords less certainty of success, since the pieces of skin often become gangrenous. But even when they do become attached they afterward shrink very much, so that the success of the operation is diminished, or is even entirely nullified. This is especially true of bits of epithelium transplanted by Reverdin's method, and therefore this is not good for blepharoplasty; larger Thiersch grafts, and especially larger flaps taken from the whole thickness of the cutis shrink less, and are hence available for this purpose. In these methods too we must always take account of the *subsequent contraction* in that we start by making a flap much larger than the loss of substance. Hence also the ground for the transplantation must be prepared in such a way as to make the raw surface that is to be covered as large as possible. After a thorough-going dissection the shortened lid is drawn far over the other lid and attached in this position, so that the two cut edges are separated as far as possible from each other and the loss of substance is made correspondingly large. The flap for covering in this raw surface is taken from a part of the body which has a skin that is as thin and as free from hair as possible—usually from the inner surface of the upper arm. Here a piece of skin is marked out which is somewhat larger than the loss of substance, and this piece is dissected off in such a way that we get only the cutis without any subcutaneous fat. This is carefully fitted to the raw surface and without being attached by stitches is kept applied simply by a light pressure bandage.

Grafting is especially adapted for those cases in which we are dealing with only small losses of substance and in which the operation is done mainly for cosmetic purposes. It is further indicated when the skin surrounding the lids for some reason—for instance, because of its cicatricial character—cannot be employed for the fashioning of pediculate flaps.

Blepharoplasty is not difficult if the free border and the conjunctiva of the contracted lid still remain, as ordinarily happens when the lid has been destroyed by

ulceration or gangrene. The case is otherwise when the *lid is completely absent*, as, for instance, when it has had to be removed in its entire width on account of epithelioma. In such a case, of course, we can use only a flap with a pedicle, as there would be no substratum for the non-pediculate flaps. But in this case a smooth lid border and an epithelial lining on the posterior surface of the flap are wanting even when the flap is pediculate. The posterior surface becomes constantly more and more contracted by cicatrization, and the flap is thus drawn up into a shapeless roll whose cicatricial free border scratches the cornea. Of the methods which have been devised for remedying this defect the best is that of *Büdinger* who makes use of the cartilage of the ear. After the pediculate flap has been fashioned there is cut from the proximal portion of the auricle a flap the size and form of which correspond to the free posterior surface of the lid-flap that has to be covered. This flap consists of the skin on the posterior surface of the auricle and of the portion of cartilage belonging to it. It is placed on the raw posterior surface of the new lid and is attached there by stitches. The cartilage of the ear replaces the tarsus, and its cutaneous lining replaces the conjunctiva. Since the skin on the anterior surface of the auricle has no defect in it, the auricle is not button-holed; the wound on the ear heals without being sewed up, leaving but a small scar which on account of its situation on the posterior surface of the ear is not conspicuous.

VII. Operations for Ptosis

842. An operation is indicated both in congenital ptosis and in old cases of acquired ptosis which cannot be cured in any other way. A whole series of methods of operating for ptosis has been proposed, a circumstance which proves that no one of them gives perfectly satisfactory results. This can be readily understood, since, in fact, the physiological action of a muscle cannot be perfectly replaced by any surgical operation whatever.

The oldest method consisted in shortening the lid by excising a *fold of skin*, a procedure which Von Graefe combined with excision of the subjacent bundles of fibers of the orbicularis (the antagonist of the levator palpebræ). This method has been abandoned, because a sufficiently great shortening of the lid always results in a considerable degree of lagophthalmus; and if only a little skin is excised the operation is unsuccessful. It is true that the new methods also are not free from the objection that they cause a certain degree of lagophthalmus, but, in comparison with the effect produced, this is slight and not injurious. These new methods start from the idea that the action of the levator can be replaced only by a muscular action. If there is a levator which, though weakened, can still contract, we attempt to increase its effect upon the lid; if the levator is entirely absent, we endeavor to invoke the aid of other muscles, like the frontalis or the superior rectus, for lifting the lid. [Good results, however, especially in congenital ptosis, may be obtained by *tarsectomy*. This is done in the same way as in Kuhnt's total tarsectomy for trachoma (page 950) except that the conjunctiva being in this case intimately adherent to the tarsus is not dissected from the latter, but is removed with it. If the ptosis is incomplete, a slice of the tarsus may be left at the upper end (Wheeler).—D.]

[In Gillet de Grandmont's operation the tarsus is exposed by an incision made through the skin of the lids, parallel to the free border of the latter and 3 or 4 mm. from it. Then two incisions are carried through tarsus and conjunctiva, one parallel with the free border of the lid and 2 or 3 mm. from it, the other arching up from the two ends of the first incision, so as to include a semilunar segment of tarsus and conjunctiva some 3 mm. high in its middle. This segment is removed and the gap in tarsus and skin closed by sutures.—D.]

843. 1. An increase in the action of the levator is effected by shortening the latter. This idea lies at the foundation of the operation for *advancement of the levator* proposed by Eversbusch. A cutaneous incision is made about midway between the border of the lid and the eyebrow, and passing in a horizontal direction from one end of the lid to the other. Starting from this incision, the skin and the fibers of the orbicularis are loosened from their attachments, so that they can be pushed to one side, and the upper border of the tarsus together with the levator can be exposed to view. A loop of thread is then passed through the tendon as high up as possible, the two ends of the loop being carried down between the tarsus and the skin of the lids and brought out in the intermarginal space. If these ends are tied there and drawn taut, the tendon of the levator is drawn down by means of the loop thus passed through it, so that the tendon is folded and thus shortened. Three such loops should be applied—one in the middle and one on either side. This method gives good results, which, however, are not always permanent, as the tendon which has been drawn down by the loops of thread subsequently retracts. Better and more permanent results are obtained if we perform the operation of shortening the tendon by *excising* a portion of it. The skin of the lid is incised along the convex border of the tarsus, and from this point dissected up as far as the eyebrow. The fascia tarso-orbitalis and, after this has been divided, the tendon of the levator now lie exposed to view. Of the tendon a portion is exsected whose length must be regulated by the degree of ptosis. Then the anterior extremity of the shortened muscle is sewed again to the convex border of the tarsus.

2. If the levator is completely inactive, shortening of it would be without effect. We then endeavor to replace its action by that of the *frontalis;* starting from the fact that persons with ptosis strive to lift the lid by wrinkling the forehead. This is regularly done in bilateral ptosis, since otherwise the patient would not be able to see. But in unilateral ptosis where there is no such compulsion, wrinkling of the forehead commonly is absent and in that case, then the following methods are inapplicable. By the wrinkling of the forehead the eyebrows are lifted and thus indirectly the lid is lifted too. But the skin of the lid must be entirely smoothed out before the edge of the lid can be elevated at all, and hence the greatest part of the effect that the contraction of the frontalis is able to exert is lost. Hence, the idea arose of making the elevation of the lid greater by connecting it directly with the fibers of the frontalis. This may be effected by means of a *subcutaneous suture* (Dransart, Pagenstecher).

One needle of a double-armed thread is entered above the free border of the lid and is carried up beneath the skin of the lid to the arch of the eyebrows, above which it is brought out again. The second needle is passed alongside the first. There is thus a loop of thread lying within the lid, the middle of the loop being situated above the free border of the lid and its two ends emerging above the eyebrows. These ends are tied over a roll of adhesive plaster or iodoform gauze and drawn tight. Ordinarily three such loops are applied alongside of one another and they are left in situ until firm cicatricial bands are formed along the suture tracks—bands which extend from the lid to the surface of the frontalis muscle and connect these two structures together (give the muscle what may be called a tendon for the upper lid).

Hess's method depends upon the same principle. In order that the scar which is left shall not be visible the incision is made in the eyebrow which has previously been shaved; it occupies the middle of the latter and traverses its entire length. Starting from this incision, the skin of the lid is detached with the knife downward as far as the free border of the lid. Then, as with the sutures described above, three loops of thread are passed from the lid up to the forehead. The entry is made in the outer skin about in the middle of the topmost part of the lid in order that when the threads are drawn up a fold of skin may be formed which will imitate the covering fold. The point of exit is about 2 cm. above the eyebrow. The loops of thread are drawn up until the lid is elevated a good deal, since the effect of the operation afterward diminishes somewhat. Since in the period immediately succeeding the operation, the lid cannot cover the eye, the latter during this time must be protected from desiccation by a dressing with a moist chamber (page 687). Hess's method is much more successful than simple sutures because the skin of the lid which has been made raw on its under side is displaced superficially on its bed which is also raw; and by the adhesion of these raw surfaces it is kept permanently in its new place.

The operation of Panas tries to secure the connection between the lid and the frontalis muscle by the formation of a pedicle from the skin of the former, which pedicle is attached to the skin of the forehead and to the surface of the muscle. Fig. 441 shows how the pedicle is cut out of the skin of the lid. The pedicle, *s*, after being defined by incision, is dissected from its bed until it is freely movable; then a horizontal incision, *a*, is made through the skin directly above the eyebrow. Starting both from this incision and from the wound already made below, the skin of the eyebrow is undermined so that a bridge of tissue is formed, beneath which the pedicle, *s*, is slipped so that its upper margin is in contact with the upper lip of the incision, *a*. Its attachment to the latter is effected by means of a loop of thread, the center of which lies on the cutaneous side of the pedicle, while its extremities, *b b*, are passed through the upper lip of the wound. By drawing the loop tight the pedicle is lifted up and is attached to the upper

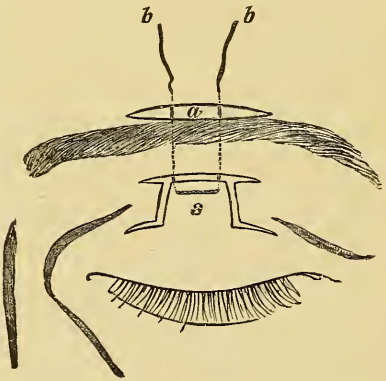


Fig. 441.—Panas's Operation for Ptosis.

border of the wound. If necessary, a second loop may be applied, and also some interrupted sutures, to secure exact adaptation of the edges. [In the *Tansley-Hunt* modification of this operation the incision is so made that the base of the pedicle is close to the free border of the lid. On either side of the lower half of the pedicle, an elongated triangle of skin is exsected, each triangle having its base directed toward the pedicle. In order to secure better union, the epithelial surface of the pedicle is scraped before the latter is drawn up into the hole under the eyebrow (Wheeler).—D.]

The operation produces a satisfactory effect but has the disadvantage of leaving scars which run perpendicular to the direction of the fibers of the orbicularis and hence are pretty conspicuous.

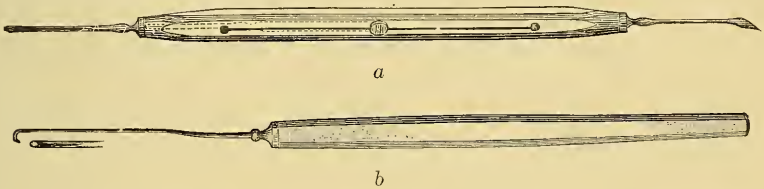
845. 3. The *superior rectus* is also available for replacing the levator [provided the former itself is not defective nor paralyzed, as it may be in congenital ptosis.—D.] Following Motais's method, we first expose this muscle by [making a transverse incision 2 cm. long through the conjunctiva over the insertion of the tendon, and dissect up the conjunctiva back to the fornix. We retract the upper flap by two sutures passed through its middle and 5 mm. apart. Between these sutures we divide the conjunctiva by a vertical incision, which is carried back to the fornix. We now evert the upper lid, grasp the upper border of the tarsus with fixation forceps, and carry the vertical incision in the conjunctiva on to the upper border of the tarsus. Next we retract the two sides of the split conjunctival flap by means of sutures passed through each close to the tarsus, and free the superior rectus from its attachments on either side. Then, starting from the end of the vertical incision in the conjunctiva, we make a transverse incision through the levator along the upper border of the tarsus, and working through this incision, detach the tissues from the anterior surface of the tarsus, so as to form a pocket extending down to the lower border of the lid. Lifting the superior rectus with a squint hook, we pass a double-armed thread through the tendon 3 mm. from its insertion, so as to form a loop on the scleral surface of the latter. On either side of this loop we split the muscle longitudinally by incisions, 3 to 4 mm. apart, running forward to the insertion and backward 10 to 12 mm. The tongue thus fashioned we detach at its insertion, leaving the nasal and temporal portions of the tendon connected with the sclera, and then by means of the two threads projecting on the upper surface of the tongue, carry it forward into the pocket on the anterior surface of the tarsus. The threads are brought out just above the free border of the lid, and tied over a roll of gauze tight enough to produce a marked primary over-effect. The conjunctival wound, especially in the cul-de-sac, must be carefully closed (Bruns, Shine). Good operators regard this as one of the best procedures for ptosis.—D.]

CHAPTER IV

OPERATIONS ON THE CORNEA AND REGION OF ANTERIOR CHAMBER

I. Removal of Foreign Bodies

846. Foreign bodies penetrating into the cornea should be removed as soon as possible. If they are superficially situated, it is an easy matter to pry them up with a suitable instrument. For this purpose we use a special needle, which is made broad at its upper end (Fig. 442). In default of such a foreign-body needle we also may make use of a sharp sewing needle which has been previously sterilized by heating in a flame. [When the foreign body is not deeply imbedded, it is better to use a blunt spud to pry it up with.—D.] It is advantageous first to render the cornea insensitive by the repeated instillation of a 5-per-cent solution of cocaine [holocaine, or alypine]. In the case of fragments of iron, besides the foreign body the ring of brown-colored corneal tissue adjoining it should be scraped off [with a tiny curette or gouge].



[Fig. 442. *a*, foreign-body needle and spud. *b*, Knapp's flexible and blunt foreign-body hook.—D.]

If the foreign body has penetrated into the deeper layers of the cornea, it is often necessary to incise the lamellæ of the cornea that lie above this foreign body so as to be able to draw it out with the forceps. If the point of the foreign body projects into the anterior chamber there is the danger that, in attempting to grasp the foreign body, the latter may be pushed in still farther, and may injure with its point the capsule of the lens. In such a case, therefore, the indication sometimes is to make a preliminary opening in the cornea near its margin and to introduce an instrument from this point into the anterior chamber. By this means we press the foreign body from behind forward, so that we can grasp it by its anterior extremity and extract it.

II. Keratotomy and Keratectomy

847. Keratotomy.—Paracentesis, or puncture of the cornea [keratotomy], may be performed either with the lance knife (Fig. 444, *a*) or with Von Graefe's linear knife (Fig. 452, *a*).

(*a*) In making *paracentesis with the lance knife* the latter is plunged in

close to the outer and lower margin of the cornea. Then the lance knife is pushed a little farther forward, so that the wound gets to be 2 or 3 mm. long, when the knife is withdrawn very slowly from the wound. Then, to make the aqueous flow off, we need only depress the peripheral edge of the wound (*c*, Fig. 408) gently with a Daviel's scoop (Fig. 452, *b*). The escape of the aqueous should be gradual, and preferably intermittent.

Paracentesis with the lance knife is made—1. In progressive ulcers of the cornea whose advance either along the surface or into the depth of the tissues cannot be arrested by medicinal treatment. In the case of corneal ulcers which threaten rupture we anticipate the occurrence of the latter by performing paracentesis. We thus avoid having the perforation take place too suddenly, and also avoid the occurrence of a prolapse of the iris. When the floor of the ulcer is greatly thinned and is bulging, we select this as the site of our puncture. 2. In ectasiæ of the cornea of various kinds, and also in markedly bulging prolapses of the iris, or in the staphylomata that develop from the latter. In these cases paracentesis must be followed by the use of a pressure bandage [see pages 970, 971]. 3. In obstinate inflammations of the cornea or uvea, and also in opacities of the vitreous, in order to exert a favorable effect upon the nutrition of the eyeball by altering the conditions of tissue metamorphosis. [4. In suppuration of operative wounds. Here repeated paracentesis (H. Knapp), combined with irrigation and the introduction of antiseptic solutions (De Schweinitz) may sometimes help.—D.] 5. In elevation of tension when this is likely to be transient, as, for example, in irido-cyclitis or in swelling of the lens. 6. In hypopyon, for removing it when it extends high up.

In all these cases it is not infrequently necessary to repeat the paracentesis one or more times. If only a few days have elapsed since the first paracentesis, a new incision is not required when the latter is repeated, but the wound, not being solidly united, may be pried open with a Daviel's spoon (Fig. 452, *b*).

Paracentesis with the *Graefe linear knife* is made according to the method proposed by *Saemisch* [really by Heuerman in 1765 and Guthrie in 1843] in ulcus serpens (see page 275). The Graefe knife, whose cutting edge is directed straight forward, is entered to the outside of the temporal border of the ulcer, in the healthy portion of the cornea; then it is pushed in the anterior chamber toward the nasal side until its point is brought out again through the cornea to the inside of the nasal margin of the ulcer. We then may be said to have the ulcer lying upon the edge of the knife, which latter, therefore, has simply to be pushed farther to the front in order to split the ulcer from behind forward. The section should have both its terminal points lying in sound tissue, and, if possible, should be so made that the most intensely yellow, progressive portion of the ulcer is bisected by it. After the section has been completed the hypopyon is removed. The sec-

tion must be reopened daily (with a Weber's knife (Fig. 412, *f*) or with a Daviel's scoop), until the ulcer begins to grow clean.

In performing paracentesis for ulcus serpens we must take care not to injure the lens and not to let the escape of aqueous take place too suddenly. The hypopyon is either evacuated spontaneously, especially if the patient makes pressure with his lids, or it can be grasped by means of forceps introduced into the wound, and drawn out. For, in ulcus serpens, it is not thin and liquid, but of a tenacious, viscid consistence. In consequence of the diminution of pressure after the escape of the contents of the anterior chamber hæmorrhages often take place from the iris, which, already hyperæmic before the operation, now becomes still more distended with blood. This distention of the iris is probably the cause of the violent pain which regularly follows upon the discharge of the contents of the anterior chamber, although the incision itself is but little felt. After incising the ulcus serpens we always get an attachment of the iris to the cornea during healing, which, however, would not have failed to occur, even apart from the operation, in those cases in which incision is indicated at all.

848. Keratotomy and Keratectomy in Staphyloma.—(*a*) *Total Staphyloma.*—The simplest procedure in total staphyloma is *incision.* This is done with the expectation that as a consequence of it the staphyloma will collapse, and, because of the retraction of the cicatricial tissue of which it consists, will remain permanently flat. Evidently this procedure is crowned with success only when the staphyloma is thin-walled enough to collapse after the incision has been made; it is, therefore, indicated only in those recent staphylomata which are still akin to prolapse of the iris. The incision, which is done with a cataract knife, is made in various ways: either in a straight line and transversely across the middle of the staphyloma (Küchler), or in a curved direction and concentrically with the lower corneal margin, so that a flap is formed of the wall of the staphyloma. The latter way of making the section has the advantage of causing a marked gaping of the wound, since the flap contracts owing to the drawing up of the cicatricial tissue. Consequently, the lips of the wound are prevented from rapidly reuniting, in which case the ectasis would soon be reproduced and the incision would have to be repeated. If the wound in the flap should not gape sufficiently, the flap must be retrenched by the removal of a part of it. After the completion of the section, the lens, in case it is still in the eye, must be removed by lacerating the anterior capsule.

Simple ablation of the staphyloma by Beer's method is performed by first separating the lower half of the staphyloma from its base by a curved incision made with the cataract knife. The flap thus formed is grasped with the forceps, and then the upper half of the staphyloma is cut off from its base by means of the scissors. The lens, which now presents, is removed by opening the capsule. The gap which has been substituted for the staphyloma by the operation may now be left to be closed by the unaided action of cicatrization. It is better, however, to close the gap by sutures passed through the upper and lower margins of the staphyloma, which

have been left for the purpose. These sutures on being drawn tight bring the lips of the wound together horizontally.

A still more secure union is effected if we follow up the *ablation with conjunctival suture* (De Wecker), and it is in this way that the operation is usually performed at present. We begin the operation by dividing the conjunctiva all round the limbus and separating it for some little distance from the subjacent sclera, so that it can be drawn forward to the proper extent. Then we pass the threads through the free edge of the conjunctiva. We do this by running through the upper and lower edges a number of vertical threads, which subsequently are tied so as to form interrupted sutures. Before the sutures are drawn tight, we ablate the staphyloma as in Beer's method, and expel the lens from the eye. Then we close the conjunctival wound by knotting the sutures. We may also apply the suture by passing a single thread in and out all round the margin of the detached conjunctiva, in such a way that the two ends of the thread come out close by each other, and, when they are knotted together, constrict the conjunctiva after the manner of a tobacco bag (tobacco-bag suture). After passing stitches through the conjunctiva, we turn the latter back and the stitches with it, and then carefully scrape off the limbus conjunctivæ as well as the epithelium at the margin of the staphyloma, since if we did not do so the conjunctiva, being stitched over an epithelial-clad surface would not adhere to it. Then we abscise the staphyloma by Beer's method, in such a way that a narrow marginal portion is left above and below. Through these marginal portions threads which are designed to close the gap in the eyeball are passed and drawn out into loops. Then before the stitches are approximated we expel the lens by opening the lens capsule. We apply the stitches beforehand, because after the lens is removed there is a risk that the vitreous will escape, and hence, we ought not to be losing time in applying sutures, but, on the contrary, should close the wound promptly by tying threads that have been already passed. Last of all, the stitches that have been passed through the conjunctiva are drawn together and tied [see also Fig. 443].

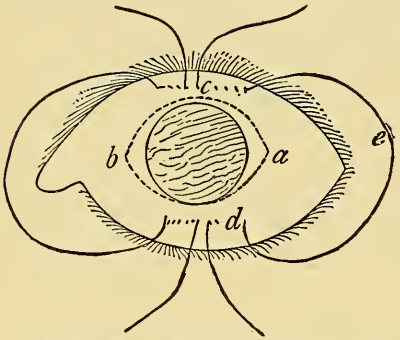


FIG. 443.—ABSCISSION OF STAPHYLOMA. KNAPP'S METHOD. (After Norris and Oliver.)

Before abscising the staphyloma a stitch is entered 4 mm. above the vertical meridian of the cornea (at *c*), passed temporally through the conjunctiva and episcleral tissue, brought out so as to form the loop *e*, re-entered below at *d*, and brought out again beneath the vertical meridian of the cornea. A similar stitch is passed on the nasal side. The staphyloma is then abscised by Beer's method and the two ends *c* and *d* of the first stitch drawn taut until the loop *e* is converted into a straight line. The same thing is done with the inner stitch. The gap made by the abscission is thus converted into a vertical slit. The sutures are then tied.—D.]

Ablation with the application of a suture is suitable for all cases of old staphylomata with thick walls, for which incision alone would not be sufficient.

Those cases of staphyloma of the cornea in which a considerable ectasis of the sclera has developed as the result of an increase of tension are in general not adapted for ablation. We should then run the risk of getting a violent hæmorrhage in consequence of the sudden diminution of the previously increased tension. For such cases, in which the eyeball is increased sometimes to quite an enormous size, the only thing feasible is *enucleation*, which relieves the troublesome symptoms and at the same time also the disfigurement, inasmuch as an artificial eye can then be worn in place of the hideous, enlarged eyeball.

849. (*b*) *Partial Staphyloma.*—In this, treatment seeks a threefold object: to improve the sight, to cause flattening of the ectasis, and to prevent the development of an increase of tension, or to do away with it in case it has already set in.

Simple incision, which must be followed up by the application of a pressure bandage for a pretty long time, accomplishes its end only in recent staphylomata, the walls of which are still soft. In older and thicker scars, *excision*, with or without the operation of uniting the edges of the wound by sutures [or of covering them with a conjunctival flap (conjunctivo-keratoplasty)], is to be preferred. But the most approved remedy that we possess against ectatic scars is *iridectomy* followed by the application of a pressure bandage (see page 980). [Iridectomy should be done before the process is far advanced, otherwise the result is apt to be unsatisfactory (A. Knapp). The same effect may be produced by Elliot's sclero-corneal *trephining*, see page 984).—D.] In thick-walled and unyielding staphylomata, it is advisable to combine excision [and conjunctival transplantation] with iridectomy. We begin by doing the former, and put off the iridectomy to some weeks later, when a flat cicatrix forms—doing this to prevent renewed bulging of the recent cicatrix. A very small staphyloma we may remove completely by excising with a corneal trephine and implanting a piece of normal cornea in the gap (*transplantation of cornea*, see page 972).

If we have to do with a partial staphyloma in which, owing to the increase in tension, the sight has been already either in great part or altogether annihilated, no gain in this regard can be expected; yet in most cases it will be best to perform iridectomy in order to prevent the further consequences of the increased tension, such as partial ectasis of the sclera, enlargement of the entire eyeball, etc. Of course, iridectomy can be performed in such cases only so long as the anterior chamber is still existent. When by increased tension the iris has been squeezed against the posterior surface of the cornea and cemented to it, iridectomy has become technically impossible [cf. page 325].

850. Operations for Keratoconus.—Operation is performed in keratoconus in order to substitute a resistant cicatrix for the attenuated apex of the cone. For this purpose the latter is destroyed by *excision* or by cauteri-

zation. The former is done with the corneal trephine, the latter with the *galvano-cautery* and if necessary the operation must be repeated several times until sufficient flattening of the cone is produced by the contraction of the scar. [The best results seem to be obtained if the cauterization is preceded by a sclero-corneal trephining by Elliot's method. By this means the tension which, though not too high for a normal eye, is too high for the attenuated and yielding cornea of a keratoconus, is reduced, and bulging of the scar is prevented. Some have also recommended *crucial incision* of the conical cornea.—D.] Since the corneal cicatrix thus obtained lies directly in front of the pupil, a displacement of the pupil to one side by means of an iridectomy is afterward usually required; also tattooing of the central scar to diminish the dazzling.

The operation for keratoconus does bring a flattening of the cornea, but the curvature of the latter is never quite regular, and, moreover, there is produced a central scar of the cornea with its optical disadvantages. We must therefore be contented if a moderate improvement of sight is secured by the operation. The operative treatment does not always give a lasting result, the cornea in many cases bulging again subsequently, although not as much as it did before. For these reasons we usually decide upon operation only in the more advanced stages of the disease. [All operations involve some risk, as glaucoma, irido-cyclitis, cataract, or possibly sympathetic inflammation may result from them.—D.]

851. Keratoplasty.—*Transplantation of the cornea* (keratoplasty) may be used to partly replace diseased by healthy cornea. Hippel's trephine, the small crown of which is set in rotation by clock work, serves for performing this operation. The operation was first devised with the view of making a transparent spot in the case of complete opacity of the cornea which left no portion open for iridectomy. A round piece is excised, with the trephine from the opaque cornea usually in its pupillary area. In the gap is placed a piece which is taken from a healthy cornea with the same trephine crown and which, therefore, exactly fits in the hole. It is most advantageous to take the piece designed for transplantation from a human cornea. Opportunity for getting this is afforded when an eye with a sound cornea has to be enucleated. The human cornea has the advantage over that of animals in that it has the same thickness as the diseased cornea and hence fits into it, and also because it becomes readily united. Union in fact almost always takes place, but in very few of the cases as yet observed has the piece thus incorporated remained transparent. In other cases it gets opaque in the weeks immediately following, and so much so generally that the purpose of the operation—the restoration of sight—is not attained. Moreover, I have had no better results with trephining by Hippel's method in which the most posterior layers of the cornea were allowed to remain at the site of the trephining. On the other hand, transplantation satisfactorily accomplishes its object when the case is one of thin, protruding, or fistulous scars which it is desirable to remove entirely in order to prevent subsequent harmful consequences. The operation in this case is done in the way described above, except that after cutting out the diseased portion we must start from the opening and free the iris as far as possible from its attachments to the cornea, in order to effect a thorough removal of the anterior synechia. The operation is possible only when the scar that is to be excised has not too great an extent, and it requires care and skill to avoid injuring the capsule of the lens. The transplanted portion becomes opaque in these cases also, but the object of the operation, which is to replace the thinned scar by firm tissue and to remove the anterior synechia, is not on this account frustrated.

III. Tattooing of Cornea

852. This is done to diminish the disfigurement produced by white scars on the cornea. It depends upon the observation that many bodies, as, for example, grains of powder, may become imbedded in the cornea and remain there permanently. Tattooing consists in giving the white scar a black tint by means of india ink, which is introduced into the cicatricial tissue by being repeatedly pricked in with a needle.

The tattooing needles used for this purpose consist either of a bundle of ordinary sharp-pointed needles (Taylor), or of a single broad needle which is channeled for the reception of the ink (grooved needle of De Wecker). [The ink is poured on the cornea and pricked in by repeated thrusts of the needle. Then the ink is washed off, the effect observed, and the process repeated until the effect is satisfactory.—D.]

Tattooing should be applied only in the older scars, which are solid and flat. For in thin or ectatic scars, the latter may be weakened by the inflammatory reaction, which always follows tattooing, and thus an increase of the ectasis or even an elevation of tension may be set up. Nor is tattooing a proper procedure for eyes that have passed through a severe attack of irido-cyclitis, since this disease might be lighted up again by the operation.

In dense white scars *Fröhlich's method* is advisable. By cutting a shallow furrow with Hippel's corneal trephine we demarcate a circular area of the size of the pupil in the scar. Within this area either the epithelium alone or this and the most superficial lamellæ of the cornea with it are removed, the parts thus exposed are covered with minute scarifications, and then the india ink is rubbed into them. In the course of years the black color fades somewhat and requires to be renewed by a repetition of the operation.

In cases in which only a part of the pupillary area of the cornea is opaque, and the rest is transparent, tattooing the opaque portion may actually improve the sight, since the cicatrix, being made less transparent, does not produce as much diffusion of light as before.

IV. Iridectomy

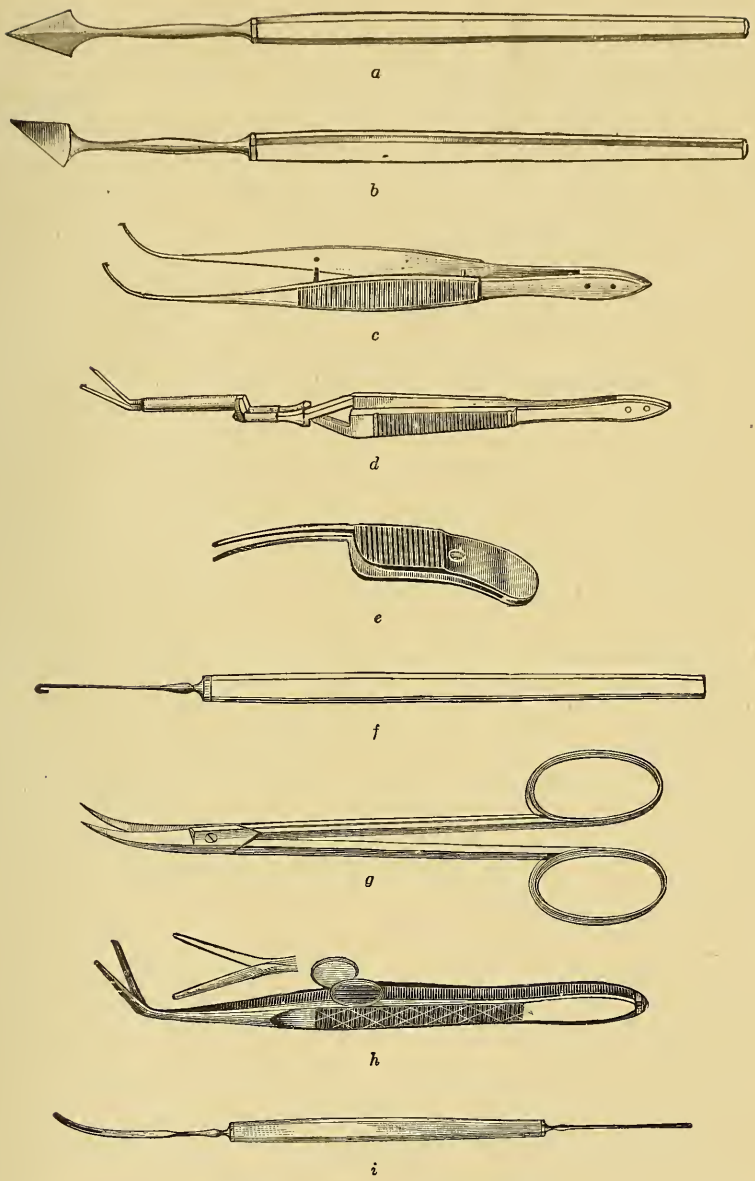
853. Technique.—Iridectomy by Beer's method is performed as follows: Entry is made with the lance knife in the vicinity of the margin of the cornea, and sometimes a little to the outside, sometimes a little to the inside of the latter, according as the point at which it is desired to excise the iris is more or less close to its ciliary margin. The lance knife is then pushed forward until the wound is of the desired length (4 to 8 mm. according to the breadth of the portion of the iris that it is proposed to cut out); in doing this the lance knife must be so held that the section is concentric with the margin of the cornea. The withdrawal of the lance is performed slowly, and with the instrument pressed against the posterior surface of the cornea, so as not to injure the iris or lens, which push forward as the aqueous flows off. After completing the section, the iris forceps (Fig. 444, *c*, *d*, *e*) with its branches closed is introduced into the anterior chamber and pushed on up to the border of the pupil. At this point the branches are allowed to separate and a fold of the iris is grasped, gentle pressure being at the same time made upon the latter. The iris is now drawn from the wound, and at the moment when it is most upon the stretch it is cut off close to the wound

with the curved scissors or with the scissors forceps (pinces ciseaux of De Wecker) (Fig. 444, *h*). This ends the operation, and it only remains by introducing a spatula (Fig. 444, *i*) into the wound to put back into the anterior chamber any iris that may have been wedged into the wound, so that at the completion of the operation the pupil and the coloboma have their proper shape.

The section in the tunics of the eye for an iridectomy may be made with the *lance* or with *Graefe's narrow knife*. With the latter the tunics of the eye are divided from behind forward as is done in the cataract operation. The section made with the lance is smoother and passes much more obliquely through the tunics of the eye than does the section made with a narrow knife. It therefore closes better after the knife is withdrawn. Hence the lance is to be preferred in every case in which no special obstacles oppose its application. This latter is the case: 1. When the anterior chamber is shallow. Here we can not push the lance far without running the risk of pricking the iris and lens. In optical iridectomies, in which a short section is ordinarily sufficient, this is a matter of less importance, but in glaucoma, in which the anterior chamber is often so shallow, we would be able to make the section with the lance neither peripheral enough nor long enough. 2. When the anterior chamber is absent the section with a lance is absolutely impossible, while we can always push a Graefe knife for a sufficiently great distance between the cornea and iris. 3. When the patients are unruly or when the operator's experience is small, the danger of wounding the lens with a Graefe knife is always less than with a lance and many operators employ Graefe's knife to the absolute exclusion of anything else for iridectomy. Unfortunately, the use of the Graefe knife meets with a limitation in that we can cut with it only at the upper and lower borders of the cornea (because elsewhere the orbital margin gets in the way). Sections at the nasal or temporal borders of the cornea can be made only with the lance. If we can not push the lance far enough along, because the anterior chamber is shallow, we enlarge the section by cutting laterally with the lance as we withdraw it.

For grasping the iris a minute *blunt hook* [see Fig. 444, *f*] may be used instead of an iris forceps. With this the margin of the iris is caught up and drawn out. The hook is suitable for cases when (1) we wish to excise only a small bit of iris at the pupillary margin; (2) when the iris has no fixed support behind it in the lens (in dislocations or absence of the lens). For in the latter case when the arms of the forceps are pressed against the iris, in order to grasp it, the iris gives way, whereas it can be picked up with a hook. [See also remarks on extra-ocular iridotomy, page 982.]

854. Accidents —The *mishaps* which may occur in the course of an iridectomy are: 1. Injury of the iris or lens with the lance, either through the clumsiness of the operator or through the restlessness of the patient. Injury done to the lens capsule entails a traumatic cataract, which not only produces a new obstacle to vision but also endangers the eye by giving rise to inflammation or increase of tension. 2. Iridodialysis. By this the excision of the iris is rendered difficult, great bleeding is set up, and often, too, a double pupil is produced (see page 440). 3. The last-named result may also occur from the fact that the sphincter pupillæ at the site of the iridectomy is left behind, so that it separates the pupil from the coloboma like a bridge. This accident may occur because the iris is excised before it has been drawn far enough out of the wound. We shall not have to complain of this disagreeable occurrence if we observe the two following rules: The first is, not to grasp the iris with the forceps until we have pushed the instrument forward as far as the pupillary margin so as to get this latter between its branches. The second rule is, to cut off the iris only when it has been drawn out far enough for its black posterior surface to be visible (Fig. 406). If, never-



[Fig. 444.—Instruments for Iridectomy and Iridotomy.

a, straight lance knife. *b*, angular lance knife. Used in iridectomy (page 973), paracentesis (page 967), simple linear extraction of cataract (page 991). *c*, *d*, *e*, iris forceps for passing into the wound made by the lance knife and grasping the iris. *f*, Tyrrel's blunt iris-hook used when the lens is absent or dislocated. *g*, curved scissors for excising the iris. *h*, De Wecker's scissors (pince ciseaux). *i*, combined spatula and blunt-pointed probe. Other instruments required are a spring speculum or Desmarre's retractor, to keep the lids apart, and a fixation forceps to hold the eyeball (see Fig. 400).—D.]

theless, the sphincter should remain behind, we enter the anterior chamber again with a blunt hook and draw up the bridge of sphincter in order to cut it off. There may, however, be another reason why the sphincter remains in situ, namely that it is so firmly adherent to the lens capsule that it tears away from the iris rather than follows the latter as it is withdrawn. In this case we refrain from any further attempt to remove the sphincter, as otherwise we might easily injure the capsule of the lens. 4. When we operate in a case of total posterior synechia it often happens that the retinal pigment of the iris within the area of the coloboma remains upon the lens capsule, with which it is intimately united by exudation. In that case, immediately after the iridectomy is completed, we may suppose that we have made a fine black coloboma, and it is only upon lateral illumination that we become convinced that the coloboma is not black, but dark brown—i. e., is filled with pigment. The optical result of the operation is then nil. It happens not less frequently in total posterior synechia that it is absolutely impossible to bring the iris out of the wound for the purpose of cutting it off. The iris, on the one hand, is so rotten, and, on the other hand, is so firmly attached to the lens, that the forceps, instead of drawing the iris out, only tears small fragments out of it. Both in this case and in the one in which the pigment layer remains behind, there is nothing left to do but to remove the lens also, by an extraction, even when the lens is still transparent. 5. Prolapse of the vitreous is particularly apt to occur in iridectomy when the zonula is diseased, as, for example, in subluxation of the lens or in hydrophthalmus, and also when we operate on very small children, in whom the zonula is as yet very frail. [6. Hæmorrhage into the anterior chamber (see page 923).—D.]

855. Optical Iridectomy.—The *indications* for iridectomy are:

1. *The presence of optical obstructions.* These consist in opacities of the refractive media, occupying the area of the pupil. Among these belong: (*a*) Opacities of the cornea; (*b*) a membrane in the pupil (occlusio pupillæ); (*c*) opacities of the lens, such as lamellar cataract, nuclear cataract, or an anterior polar cataract of particularly large diameter. Furthermore in cases in which the pupil is considerably displaced toward the periphery as the result of an incarceration of the iris, iridectomy is done to bring the pupil behind the center of the cornea; and it is also done in subluxation of the lens when the attempt is made to shift the pupil to a place in front of the part that contains no lens.

For any good to be gained from the performance of an iridectomy for optical purposes, the following *conditions* must be present:

(*a*) The opacity must be so *dense* that it prevents the formation of distinct images upon the retina, and does not simply interfere with vision by giving rise to dazzling. In the latter event the dazzling would actually be increased by the iridectomy. A mistake that we frequently meet with is that of making an iridectomy when there are comparatively slight opacities of the cornea; by such an operation the sight is made worse instead of better. To avoid this mistake we first make an accurate determination of the visual acuity, then dilate the pupil with atropine, and once more test the sight. If the latter then proves to be considerably better than before the pupil was dilated, iridectomy is indicated; otherwise not (see also page 979).

(*b*) The opacity must be *stationary*. In the case of opacities of the cornea the inflammatory process must have completely run its course; in opacities of the lens it must be stationary forms of cataract that are in question. Otherwise we run the risk of having the very spot become opaque which we have selected for making the artificial pupil in.

(*c*) *The parts concerned with the perception of light*—the retina and optic nerve—must be capable of performing their functions. This fact is determined by testing the vision and the field of vision, especially with the candle flame (see pages 115 and 845). Upon the degree to which the perception of light is retained will depend the question whether an iridectomy for optical purposes is undertaken at all or not. These requirements regarding the perception of light hold good, moreover, not only for iridectomy, but also for all operations undertaken for the restoration of sight, and particularly for the operation of cataract.

The following conditions must be regarded as *contraindications* to iridectomy for optical purposes: 1. Deficiency or total absence of light perception. 2. Strabismus of long standing in the eye which is affected with the opacity. In this case, even if the operation was technically a perfect success, not much gain in sight would be got, owing to the amblyopia ex anopsia which exists in such eyes. The result of the operation is doubtful and it is often technically impossible to perform in —3. Flattening of the cornea. For, where applanatio corneæ has developed, it is always a sign that there has been in conjunction with the keratitis an irido-cyclitis, which has left thick membranous exudates behind the iris. Hence, even if we actually succeed in excising the iris, a free aperture is not produced, and we are confronted by a hull of exudation. 4. Incarceration of the entire pupillary margin in a corneal scar with consecutive forcing of the iris against the posterior surface of the cornea. When this condition has lasted for quite a long time it is impossible to excise the iris, because the latter in consequence of atrophy is too friable, and because it is too firmly agglutinated to the cornea (page 325).

The *success* of an optical iridectomy as far as the amount of vision is concerned, very often falls short of the expectations which both physician and patient have entertained in regard to it. This is especially the case with iridectomy in scars of the cornea. In this case there are various reasons for the vision's being often so defective, even when the operation itself has been a complete success. The chief one is that normally a considerable degree of astigmatism is present in the peripheral part of the cornea which has been used for the iridectomy. This astigmatism is increased partly by the effect of the adjoining scar, partly by the operation itself. To this is added the astigmatic refraction of those rays which in the peripheral portion of the coloboma pass through the margin of the lens. This astigmatism, which is for the most part irregular, has a greater effect than usual, inasmuch as the new pupil is large and is almost or quite immovable, and hence cannot lessen the size of the diffusion circles (see page 850). Moreover, the cornea over the coloboma is often less transparent than was supposed before the iridectomy, for slight opacities are scarcely visible when a light-colored iris

is behind them, while they at once become obvious when after iridectomy a black coloboma forms the background. Still greater is the disappointment in store for the operator when after a successful iridectomy he finds the coloboma white instead of black, because the lens has become opaque.

The optical result of an iridectomy made on account of a corneal scar may often be improved if we make the scar opaque by tattooing it (see page 972).

It is obvious that the degree of sight that is regained depends also upon the condition of the percipient parts—a condition which should be ascertained before the operation by testing the perception of light. Special stress must be laid on the tests of the [vision and] visual field (see pages 115 and 845).

How must a man adjust his eye in order to see with an *eccentrically situated pupil?* Let us assume that the eye is affected with a central scar of the cornea (*n*, Fig. 445), so that it can see only with the assistance of a coloboma which has been made upward. Must this eye, in order to fix an object, *o*, be turned downward so that the coloboma may lie opposite the object? By no means. The refraction of the rays in such an eye takes place in precisely the same way as in a sound one. The only difference is, that the portions of the beam emanating from *o* which enter the eye are not those situated in the center, but those situated above and corresponding to the coloboma. These latter throw the image upon the fovea, *f*, provided the object lies in the line of vision. An object, o_1, which is situated opposite the coloboma would form its image at *b*, below the fovea, and would therefore not be seen by central vision. Hence an eye with an eccentrically placed pupil performs fixation in the same way as does a normal eye. It is not superfluous to lay particular stress upon this point, inasmuch as many erroneous ideas prevail with respect to it. In a very learned treatise on retinitis pigmentosa, one can read how in this disease an iridectomy does no good if central opacities of the lens are present, because in that case the images of objects would fall upon the peripheral portions of the retina which are insensitive! As a matter of fact, this would be so only if the objects themselves were situated in the periphery of the visual field

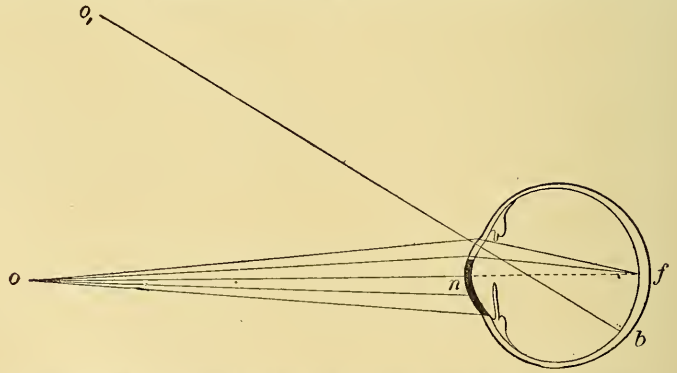


Fig. 445.—Path of Rays in the Case of an Eccentrically Situated Pupil.

The considerations just adduced also furnish an answer to the question whether a man would see double who has colobomata in both eyes which extend in different directions—e. g., upward in the right eye and inward in the left. In this case there will be binocular single vision, since the object of fixation forms its image at the same spot in both eyes—namely, the fovea—no matter where the coloboma is situated.

856. Site of Coloboma in Optical Iridectomy.—A coloboma which is made for optical purposes must be so fashioned as to cause as little disturbance from dazzling as possible. This result is secured when the coloboma

is narrow and does not reach to the margin of the cornea (*O*, Fig. 446). An excision extending up to the root of the iris would expose the margin of the lens and also the interspace between it and the ciliary processes, and thus admit a great quantity of irregularly refracted rays into the eye. In order to make the coloboma narrow and not too peripheral, the incision must be short, and lie at, or even inside of, the limbus. Those cases constitute an exception to this rule, in which only the most exterior marginal portion of the cornea has remained transparent, so that the iridectomy must, for obvious reasons, be quite peripheral.

That spot is chosen as the *site* of the coloboma at which the media are the most transparent. Wherever possible, we avoid making the coloboma above, as in that case it would be partly covered by the lid. If the media are everywhere equally transparent (as when there is a cicatrix situated exactly in the center of the cornea, a pupillary membrane, or a perinuclear cataract), the iridectomy is performed downward and inward (Fig. 446), because in most eyes the visual axis cuts the cornea a little to the inner side of the apex (page 766).

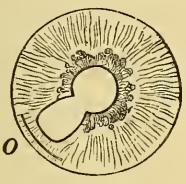


Fig. 446.

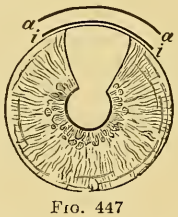


Fig. 447

Fig. 446.—Optical Iridectomy. Magnified 2 × 1.
Fig. 447.—Iridectomy in Increase of Tension. Magnified 2 × 1.—*a a*, external; *i i*, internal wound. See description under Fig. 402.

[Where to place the iridectomy can be determined by the *pear-shaped stop*. This is a disk resembling in appearance Fig. 446, in that it has in its center a circular aperture 4 mm. in diameter, with an off-set notch 4 mm. long and 2.5 mm. wide. The pupil is dilated with homatropine, and the disk is set as close to the eye as possible and with its central hole concentric with the pupil. The disk is then turned until the patient sees best, when the position of the off-set notch will indicate the proper site for the iridectomy.—D.]

857. Other Indications for Iridectomy.—2. *Increase of tension.* Iridectomy is hence indicated in primary glaucoma, and also in secondary glaucoma resulting from ectasiæ of the cornea or sclera, from seclusio pupillæ, from irido-chorioiditis, etc. In hæmorrhagic glaucoma iridectomy is often a failure. In general, the success of the operation is better the earlier it is performed. Nevertheless, an operation is sometimes done in cases of increased tension even when the perception of light has been already abolished, in which case there can be no idea of restoring sight. Then it is simply a case of relieving pain or of avoiding further degeneration (and especially further ectasis) of the eyeball.

When iridectomy is made for increase of tension, the section must be situated pretty far back in the sclera and must be long. The coloboma need not be very broad, but ought to reach to the ciliary margin of the iris (Fig. 447). If there are not at the same time any optical conditions, to which regard must be paid in making the iridectomy, the latter must be directed upward so that the coloboma may be partially covered by the upper lid, and thus the confusion due to dazzling may be lessened.

[As a preliminary to iridectomy in glaucoma it is important to reduce the tension as much as possible by miotics, cathartics, and sedatives. In this way it may be possible to avert too sudden a reduction of tension with consequent post-operative hæmorrhage. In cases where there is considerable danger of the latter, it is advisable to precede the iridectomy with a posterior sclerotomy (see page 1005).—D.]

The effectiveness of iridectomy in glaucoma is regarded as consisting in the fact that the scleral scar, acting as a substitute for the impermeable ligamentum pectinatum, enables the aqueous to filter out (filtration scar, page 512). Increasing condensation of the iridectomy scar is, therefore, thought to be the cause of the subsequent recurrences of increase of tension. If this view is correct, the cystoid scars, in which the œdema of the conjunctiva over the scar leaves no doubt as to the oozing of the liquid through it, must be regarded as being the most advantageous. In order to make a cystoid scar designedly Lagrange [and Elliot] have devised a special way of making the section so as to cut out a portion of the sclera and form a thin cicatrix. In other words, the operation is an iridectomy combined with a sclerectomy. For description see under Sclerectomy, page 984.—D.]

3. *Ectatic cicatrices of the cornea* (partial staphylomata), in order to cause their flattening This is the more likely to succeed, the more recent and the thinner-walled the staphyloma is—that is, the closer akin it is to a prolapse of the iris In thick-walled and unyielding staphylomata it is best to combine iridectomy with excision [and conjunctival transplantation] (see page 971) The iridectomy should be performed in such a way that the incision lies in the sclera and a broad coloboma is produced, reaching to the margin of the iris We try to find for our iridectomy that spot which represents the greatest improvement that can be made in the vision, the pupil being shifted to a point behind the most transparent part of the cornea. Furthermore, by means of the iridectomy, an increase of tension is prevented from developing, or, if it has already developed, it is done away with. Moreover, in cases of staphylomata with thin walls, a flattening of the ectasis is obtained by the iridectomy, if the eye is kept beneath a pressure bandage for a long time after the operation.

4. *Recurrent iritis*, in which case the iridectomy is designed to prevent the recurrences—an object, however, which is not always attained. The operation is to be done during an interval in which there is no inflammation.

5. *Fistula of the cornea.* Iridectomy here serves the purpose of securing

the formation of a firm cicatrix. We must wait to perform the operation until at least some trace of the anterior chamber has been restored, as otherwise the operation is impracticable on technical grounds.

6. *Foreign bodies* imbedded in the iris, which sometimes can be removed only by excising the portions of the iris in which they occur. The like is true of cysts and small tumors of the iris.

7. *As a preliminary to the operation for cataract,* iridectomy is done mainly when we are dealing with complicated cataracts (e. g., those complicated with posterior synechiæ, increase of tension, etc.).

If iridectomy is made as a preliminary to a cataract extraction the excision of the iris must be made upward, so that the coloboma may also be utilized for the extraction of the cataract, which as a general thing is made upward.

V. Iridotomy

858. Iridotomy consists in simply dividing the iris without excising a piece of it, and in this respect differs from iridectomy. It serves the purpose of making a new aperture in the iris when the pupil is closed and of thus producing a new pupil. As the incision in the iris would also affect the lens which lies behind it and would thus produce traumatic cataract, this operation is adapted only to those cases in which no lens is present. In most cases the operation has to do with eyes which have been operated upon for cataract but which have lost their sight again through a subsequent irido-cyclitis. In these cases the iris is united with the exudation membrane and with the secondary cataract to form a firm diaphragm, which separates the cavity of the chambers and the cavity of the vitreous. To restore sight the diaphragm must be perforated. This can be accomplished by a simple incision, if this is so directed as to divide the diaphragm along a line perpendicular to that of greatest tension; then the incision gapes from retraction of the edges of the wound and leaves a slit-like pupil (cat's-eye pupil).

The operation may be performed with—

(*a*) The *Graefe knife.*—This is plunged through cornea and diaphragm, and the latter is divided in a direction perpendicular to that of greatest tension. This method is applicable only when the diaphragm is not too thick. Were this the case, the diaphragm would offer great resistance to the knife, and in the endeavor to divide it the ciliary body would be pulled upon, and this might start a new attack of irido-cyclitis.

(*b*) The *scissors forceps* (pinces ciseaux) by De Wecker's method. With the lance knife an incision is made along the corneal margin, and through this the scissors forceps is introduced closed into the chamber. Here the instrument is opened, and its posterior sharp blade is plunged through the diaphragm while the anterior blade remains in the anterior chamber; then,

by closing the scissors forceps, the diaphragm is divided perpendicularly to the direction of greatest tension. This is a severer operation than the former, and is also usually associated with loss of vitreous. On the other hand, it does not cause any dragging, as in it the diaphragm is divided just as a sheet of paper is cut in two by scissors.

[(*c*) *Ziegler's knife-needle* (Fig. 450 *f*).—This is entered at the upper sclero-corneal junction, with the blade turned on the flat. The point is carried down toward the bottom of the anterior chamber and then swung 3 mm. to the left. Next the edge of the knife is turned downward, the point is plunged into the iris, and the blade gradually drawn back so as to make an oblique cut in the iris from below upward. The blade is then swung over to the other side of the chamber and a similar cut made from below upward meeting the first cut at its apex. A triangular tongue of iris is thus marked out, which falls down, leaving a triangular pupil.—D.]

Iridotomy sometimes fails owing to the too great firmness of the diaphragm, which may actually be ossified; but even an excellent immediate result may be nullified because the old irido-cyclitis is lighted up again by the operation, and the pupil made by it is closed again by renewed exudation. Hence we put off the performance of iridotomy as long as possible until all inflammatory symptoms have disappeared, unless we are compelled to operate speedily by special circumstances, such as protrusion of the iris, increase of tension, or beginning atrophy of the eyeball.

In order to be able to perform an iridotomy without danger to the lens, in case this is present, we may do an *extra-ocular* [or *precorneal*] *iridotomy*. We make a puncture in the limbus with the lance knife, as for an iridectomy, draw out the iris [preferably with a blunt hook], incise it in a radial direction (from the pupillary to the ciliary border), and then return it to the anterior chamber. In this way a V-shaped gap is made in the iris, and accordingly we use this method of operating in place of an optical iridectomy, when we wish to get a very narrow coloboma.

Another case in which it is possible to perform iridectomy when the lens is present, without injuring the latter, is found in hump-like protrusion of the iris such as results from seclusio pupillæ; for here a considerable interspace—namely, the enlarged posterior chamber—separates the iris from the lens. Iridotomy in this case may be done by *transfixion* of the iris (Fig. 174). A Graefe knife is entered about 1 mm. to the inner side of the temporal margin of the cornea, passed through the anterior chamber, and made to emerge at a spot near the inner margin of the cornea and symmetrically situated with respect to the point of entry. The points of entry and exit lie in the horizontal meridian of the cornea, and the knife is held so that its blade is parallel to the base of the cornea. Since the iris is driven forward, the knife as it is being carried through the anterior chamber pierces the most protruding portion of the iris both temporally and nasally and makes holes in it. These holes remain permanently open and restore the communication between the anterior and posterior chambers; the iris returns to its former position, and the intra-ocular pressure becomes normal. In cases of hump-shaped protrusion of the iris, this operation may consequently be made instead of an iridectomy; and in cases of this sort it may be done as a preliminary operation to an iridectomy, so that the latter can be done later under more favorable conditions.

VI. Iridotasis

859. [This old operation has been recently revived by Borthen and used with apparent success in glaucoma.

Atropine is instilled half an hour before the operation. A transverse cut, 6-8 mm. long is made through the conjunctiva at a point 10 mm. above the cornea. The conjunctiva is undermined down to the corneal margin and the flap reflected. A keratome is entered in the sclera just above the cornea making a wound 4 to 5 mm. broad. (It is essential that this should not be too broad, or the iris will not stay in place.) The iris forceps is introduced and the iris is grasped by its free edge and pulled into the wound with its under surface up. The conjunctival flap is then replaced so as to cover in the scleral wound containing the iris (Harrower).—D.]

VII. Anterior Sclerotomy and Sclerectomy

860. Anterior Sclerotomy.—*Paracentesis of the sclera* (sclerotomy) done so as to enter the anterior chamber is an operation performed for glaucoma.

Sclerotomia anterior by De Wecker's method (Fig. 448) is made as follows: The Graefe knife is entered 1 mm. outside of the temporal margin of the cornea, and brought out at an equal distance to the inside of the nasal margin. The points of entrance and emergence are therefore symmetrically situated, and are selected as though the intention was to form a flap 2 mm. high out of the upper part of the cornea. And, in fact, after the counter-puncture has been made, the incision is carried upward by sawing cuts just as if this flap was to be separated, but the knife is withdrawn before the section is completed. Thus, at the upper margin of the cornea there remains a bridge of sclera, which connects the flap with the parts below and prevents the gaping of the wound. Hence by this operation two sections at once are made in the scleral margin separated by a narrow bridge (s and s_1). Sclerotomy may be made downward as well as upward. [In Herbert's small-flap sclerotomy an opening is made into the anterior chamber through a ⌐¬-shaped incision in the sclera close to the cornea.—D.]

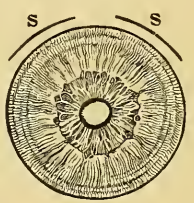


Fig. 448.—Sclerotomy by De Wecker's Method.

Sclerotomy, on account of the peripheral position of the wound, is very likely to cause prolapse of the iris. We should therefore try to produce a marked miosis by eserine before the operation; the spasmodically contracted sphincter then keeps the iris in the anterior chamber. If in spite of this the iris becomes wedged in the wound, and cannot be satisfactorily replaced it must be drawn out and cut off. The danger of a prolapse of the iris is not present if there is already a coloboma of the iris and the sclerotomy incision is placed near the site of the coloboma—the sclerotomy being made because of the recurrence of a glaucoma for which an iridectomy has already been performed.

Sclerotomy is performed in glaucoma, but its results are not as certain, and, more particularly, not as lasting as those of iridectomy. Sclerotomy, accordingly, is performed only in exceptional cases [and even in these is now largely replaced by trephining (page 984)—D.]

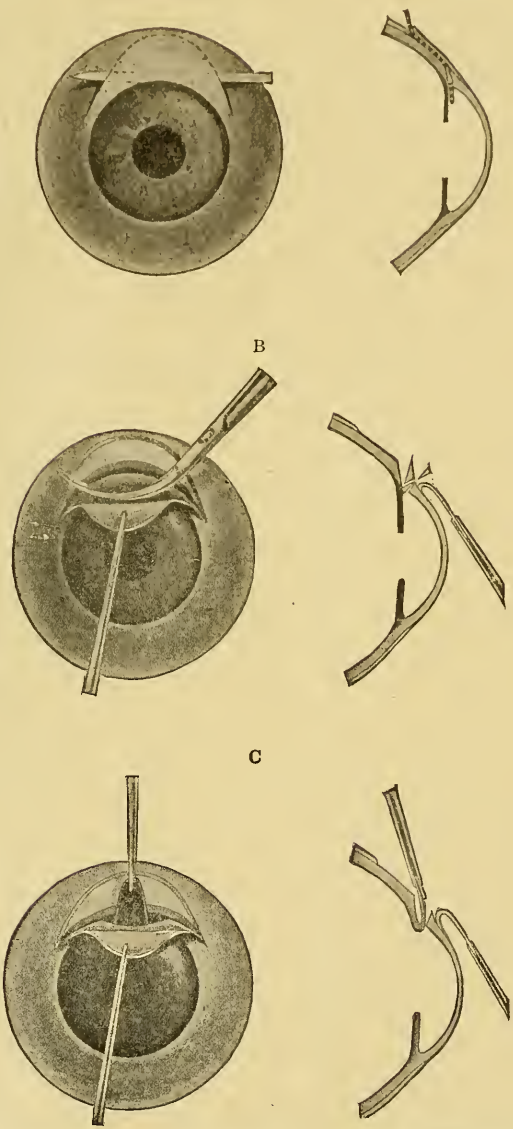
861. Anterior Sclerectomy.—Sclerectomy denotes the excision of a bit of thickness of the sclera.

(*a*) *Lagrange's operation.*—In this the incision is made with a Graefe

knife [in such a way as to divide the sclera in the irido-corneal angle. Puncture and counter-puncture are placed 1 mm. from the limbus.] The operator, as he cuts, holds the knife flat, so as to pass very obliquely through the sclera, and come out beneath the conjunctiva several millimetres back of the limbus. He then divides the conjunctiva so as to form a flap. After the conjunctival flap has been laid back upon the cornea, the tongue which the knife has fashioned from the sclera becomes visible. By grasping this tongue with the forceps and dividing it with the scissors, a defect is made in the sclera which reaches to its deepest layers. [In other words, the sclera is deeply beveled. After this sclerectomy has been done, iridectomy is performed in the usual way, and the conjunctival flap is brought up and laid over the defect in the sclera.—D.] The direct results of this method of operation are in my experience good. Whether it can permanently forestall recurrences of glaucoma, more prolonged observations will have to show. [These so far have confirmed this good opinion.—D.]

862. *Sclerostomy.*—[In some forms of sclerectomy a permanent hole is made in the sclera. The chief varieties of this sclerostomy (Elliot) are]:

(*b*) *Elliot's sclerectomy* or *trephining* of the sclera. This is performed in the following way. The conjunctiva is divided with the scissors 5 mm. above the cornea and in a direction concentric with the latter, and the two ends of the incision are prolonged downward as far as the margin of the cornea, so that a conjunctival flap is formed, the base of which lies at the upper limbus. This flap is dissected off as far as the limbus, and the dissection is carried even somewhat beyond the latter by undermining the limbus and the superficial layers of the cornea with the keratome, so that the real junction of the cornea and sclera is exposed. To cut the sclera the trephine devised by Elliot is used. This is a steel tube, whose inferior sharp edge has a diameter of 1.5 -2 mm. This is set on the eye in such a way that the opening of the tube lies half on the cornea, half on the sclera. By revolving the trephine between the fingers we make it gradually enter the sclera. When it penetrates the latter we appreciate the fact by the cessation of resistance. After the instrument has been withdrawn the little disk of sclera that has been excised falls out, or, if it is still adherent at one spot, it is snipped off with the scissors and with it the iris which protrudes through the trephine opening. We thus get a narrow coloboma often not reaching to the pupillary margin. Then the conjunctival flap is put back in its former place where it soon becomes adherent without being attached by sutures. In its technique Elliot's trephining is easier than simple iridectomy and Lagrange's operation and has the advantage that in doing it we are not as a rule compelled to enter the anterior chamber with an instrument. Its fistulizing effect is perhaps more permanent than that of an ordinary iridectomy, but it shares with the operation of Lagrange the disadvantage that it gives rise much oftener than iridectomy to late infection [see page



[Fig. 449.—Lagrange's Operation—after De Schweinitz.
A.—Division of Sclera and Conjunctiva. B.—Resection of the Sclera.
C.—Making the Iridectomy.]

512]. For this reason in all ordinary cases of glaucoma I have returned to the old operation of iridectomy.

[Opinions are divided as to the frequency of *late infection* after this operation. That it is a real danger there seems no doubt, the result when it occurs being sometimes panophthalmitis, sometimes irido-cyclitis with destruction of sight, and sometimes a mild iritis with inflammation about the aperture, going on to recovery (Butler). Hence, in America, too, there has been a tendency to return to iridectomy.

Trephining finds its most extensive *application* in glaucoma, especially in the chronic congestive and the non-congestive forms, and particularly in cases in which the visual field is contracted nearly to the fixation point, since in these iridectomy sometimes causes further contraction with consequent loss of central vision (page 511), while with trephining this is less likely to occur, but has also been used with success either alone or combined with other measures (cauterization, excision of cornea, paracentesis of sclera) in keratoconus, staphyloma of the cornea, and detachment of the retina (see pages 595, 971, and 972).—D.]

[(*c*) *Holth's punch operation.*—In this a conjunctival flap is cut and reflected as in the Elliot operation, and the cornea is split as in the latter but only so far as to enable the opening in the sclera to be made up to the corneal edge. A special bent keratome or broad needle is introduced at a point 1.5 mm. above the cornea and carried into the anterior chamber. Into this cut a delicate punch is introduced which cuts out a D-shaped segment of the sclera. Over the hole thus left the conjunctival flap is replaced and sutured. By some (Butler, Koller) this operation is preferred to trephining, because with it no pressure is exerted on the glaucomatous eye. Otherwise it would seem to have the same advantages and disadvantages as the Elliot operation.—D.]

[(*d*) *Seton Drainage.*—This operation, originally performed by Zorab and revived by Wood, is designed mainly for employment in cases of absolute glaucoma. It consists in passing a silk thread into the anterior chamber through a scleral or sclero-corneal opening and imbedding the outer end of the thread under the conjunctiva. Prince inserts in a trephine opening a ring of very fine gold wire to act as a permanent drain.—D.]

863. Operations for Opening Chamber Angle.—[Artificial drainage of the anterior chamber, undertaken in order to relieve glaucoma, may be effected not only exteriorly, as in sclerectomy, but also directly into Schlemm's canal or into the perichorioidal space. Of the methods of effecting this,] two may be mentioned. One is the operation of *cutting into the angle of the anterior chamber* from the anterior chamber itself (by De Vincentiis' method). By this means the path for the escape of the aqueous into Schlemm's canal is opened again. The operation is made with a special, small, sharp-pointed knife, plunged in at the corneal margin and carried through the anterior chamber so as to scarify the opposite portion of the sinus of the chamber. This operation is technically feasible only when the anterior chamber is sufficiently deep. Hence, it does not come in for consideration in most cases of inflammatory glaucoma. In appropriate cases, with deep enough anterior chamber, I have sometimes tried it, when an iridectomy has been without re ult, and have then several times seen a good effect from it. In Heine's *cyclodialysis* the operator, by a short incision situated 5 mm. from the corneal margin, divides the sclera, proceeding carefully from without inward down to but without injuring the chorioid; then carries a spatula forward between chorioid and sclera until its tip appears in the anterior chamber. Thus the ciliary body is detached from the sclera and a communication effected between the anterior chamber and the perichoricidal space, by which now it is expected that the escape of aqueous will take place, if the way through the ligamentum pectinatum is blocked.

CHAPTER V

OPERATIONS ON LENS AND VITREOUS CHAMBER

I. Discissio Cataractæ

(*a*) *Discission of Soft Cataracts*

864. Technique.—Discission[1] of soft cataracts has for its object the opening of the anterior capsule of the lens in order to effect the resorption of the latter. Discission is performed with a sickle-shaped needle [or with the knife-needle (see Fig. 450)], which is passed in through the cornea (keratonyxis)[2]. The site of the puncture is the center of the lower and outer quadrant of the cornea, at which spot the needle is passed through the cornea and perpendicular to the latter, and is then pushed forward in the anterior chamber as far as the anterior capsule of the lens. [In order to minimize the risk of post-operative infection, some prefer to enter the conjunctiva beyond the sclero-corneal margin and then through the periphery of the anterior chamber (Byers).—D.] The lens-capsule is then laid open by one or more incisions in the area of the pupil (which has previously been dilated with atropine). The needle must be handled very lightly, no pressure being made with it, but simply sweeping movements; moreover, the incisions should not penetrate deep into the lens. The needle is then withdrawn from the eyeball, this being done quickly so that the aqueous may not escape.

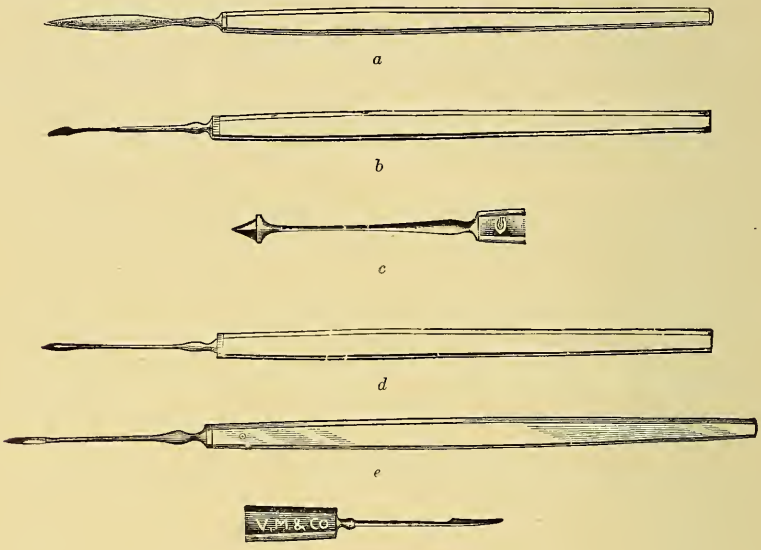
After the operation the aqueous enters the lens through the wound in the capsule, and the lens swells up and is gradually absorbed in the manner described at length under the head of traumatic cataract (see page 548). In fact, discission is nothing but an imitation of the kind of injury of the capsule that accident so frequently produces.

Discission is adapted for all soft cataracts—i. e., for those which are capable of complete resorption because they have as yet no hard nucleus. This is the case in children and in adolescents. Discission may also be made in those cataracts that still contain transparent portions of lens substance, since these become opaque under the influence of the aqueous. The form of cataract most frequently requiring this treatment is perinuclear cataract. Lastly, discission may also be employed for removing perfectly transparent lenses, when it is a question of doing away with a high degree of myopia by operation.

In the cases cited discission may be the only operation done; i. e., after doing it we simply wait for a gradual resorption of the lens to take place.

[1] From *discindere*, to split (i. e., the lens capsule). [2] From κέρας, horn, and νύττειν, to prick.

This usually requires some months, and a repetition of the discission is often necessary. But, to shorten the duration of treatment, we may also employ discission simply as a preliminary operation to make the lens fit for extraction. For this purpose we make the discission quite free, so as to get a rapid opacification and swelling of the lens. As soon as this has advanced to a certain point—which is the case some days or weeks after the discission—the softened and disintegrated lens is removed by a linear extraction.



[Fig. 450.—Instruments for Paracentesis and Discission.

a, broad needle for paracentesis. *b*, Bowman's stop needle. *c*, Desmarre's broad paracentesis needle. *d*, Beer's cataract needle. *e*, Knapp's knife-needle. *f*, Ziegler's knife-needle. Other instruments required are a speculum and fixing forceps (see Fig. 400) and for some membranous cataracts De Wecker's scissors (Fig. 444, *h*),—D.]

The main advantage of discission consists in the freedom from danger of the operation itself and in the simplicity of the after-treatment. Since the small puncture in the cornea closes again directly, the patient is not compelled to keep to his bed after the operation and the bandage may be dispensed with after one day. If the course is favorable, no further treatment is required than to keep the pupil dilated with atropine until the resorption of the lens is complete. Discission, therefore, is the only cataract operation which can be employed with very small children who do not keep quiet after the operation.

865. Accidents.—During the after-treatment various accidents may occur necessitating interference on the part of the physician. These are sometimes produced by the fact that the process of swelling takes place with too great violence; sometimes, on the contrary, by the fact that the swelling and absorption of the lens are brought to a standstill.

The *violent swelling* of the lens may be caused by too extensive a splitting of the capsule, owing to which the lens is exposed to the action of the aqueous over an excessively large area. In other cases, again, there exists in the lens a peculiar tendency to swell, which makes itself apparent even with small incisions of the capsule. The results of a rapid swelling of the lens may be either increase of tension or irido-cyclitis. The former manifests itself by the dull appearance of the cornea, by an increase of tension perceptible to palpation, and often by the occurrence of pain; and these symptoms, if they were allowed to persist, would lead to amaurosis due to excavation of the optic nerve. The iritis is caused either by the mechanical injury (pressure) or the chemical irritation which the swelling masses of lens substance produce in the iris. Both increase of tension and iritis are particularly to be apprehended in elderly persons, because these stand swelling of the lens worse than others do. To avoid these accidents the pupil must be kept well dilated with atropine, so that the swelling masses of lens substance may come into contact with the iris as little as possible. Excessive swelling is most effectively combated by iced compresses, which, moreover, have an antiphlogistic action. The best remedy against the consequences of excessive swelling of the lens is the removal of the swelling masses by extraction. If we had intended at the start to have an extraction succeed the discission, a considerable swelling of the lens is rather what we should desire. But if we wish to get along with a discission alone, we must make the discission itself quite limited so as not to be compelled by excessive swelling of the lens to perform an extraction afterward.

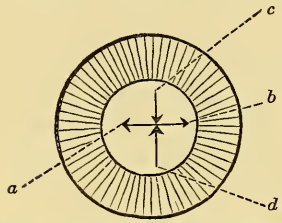
In contradistinction to the cases just mentioned, there are others in which from the outset the processes of swelling and resorption of the lens *take place to an insufficient degree*. In this event we are often dealing with the kind of lenses which are most likely to be found in aged persons, and which can swell but little. In other cases everything goes well at first, but after a part of the lens has been absorbed the swelling and resorption come to a standstill. The cause of this commonly lies in a union of the capsular wound, which takes place to such an extent that the aqueous no longer comes into contact with the lens fibers. In either case the indication is to repeat the discission, in doing which one may proceed more boldly than in the first operation and make an extensive opening in the capsule.

866. Contraindications.—Discission is contraindicated—1. In elderly persons whose lenses already have a nucleus, and whose eyes, moreover, do not bear well the swelling of the lids. 2. In subluxation of the lens, a condition which is recognized by the tremulousness of the latter. In this case discission is impracticable on technical grounds, since the lens being insufficiently fixed in its place would recede before the discission needle. 3. When there is considerable thickening of the capsule of the lens, as in this case the discission needle would cause luxation of the lens before it could tear

through the capsule. 4. In the presence of posterior synechiæ, which render the dilatation of the pupil by atropine impossible. In such a case an iridectomy would have to precede the discission.

(*b*) *Discission of Membranous Cataracts* (*Dilaceration*)

867. The discission of membranous cataracts is not made with the view of effecting their resorption, since shrunken cataracts no longer contain much or any matter capable of being absorbed. On the contrary, their object is to make a free opening in the cataractous membrane by tearing it apart, and for this reason it had better be called dilaceratio cataractæ. [The name is not applicable to the operation with the knife-needle, or Graefe knife, which is a cutting not a tearing (see page 991).—D.] The operation may be performed either through the cornea or through the sclera.



[Fig. 451.—Incision with the Knife-needle in Soft Primary and in Secondary Cataract. (After Knapp in Norris and Oliver.)

The knife-needle is entered 3 mm. from the margin of the cornea in the horizontal meridian. The needle is advanced to about 2 mm. beyond the anterior pole of the lens, thrust through the lens capsule and (if the lens is present) through the superficial layers of the lens, and drawn back so as to make a horizontal incision, *ab*, 4 to 5 mm. long. Then the point of the needle is raised toward the cornea, and pushed upward in front of the capsule to the point *c* from 2 to 2.5 mm. above the horizontal incision. Here the capsule is transfixed and is divided by a downward sweep as far as the horizontal incision. The same manœuvre is done on the lower half of the capsule from *d*, below upward, so that the three cuts make a crucial opening with arms 4 or 5 mm. long. When the operation is done as a preliminary to extraction, the incision may be longer and deeper, and in membranous cataracts will be varied in size and situation to suit the exigencies of the case.—D.]

In the operation *through the cornea* (keratonyxis) the puncture is made in the center of the outer and lower quadrant of the cornea, as in the discission of a soft cataract or in the limbus in its outer and lower part. The needle is then pushed forward and plunged through the cataract, and then the attempt is made by means of sweeping movements to tear the cataract in all directions, so that as large a gap as possible may be formed in it.

In the *operation through the sclera* (scleronyxis[3]) the needle [or better a Graefe knife or the knife-needle (see page 991)] is plunged in perpendicularly through the sclera, 6 mm. behind the external margin of the cornea, and somewhat below the horizontal meridian, and is then pushed forward so that its point passes through the cataractous membrane into the anterior chamber close to the external margin of the pupil. Then the attempt is made to tear the cataract to the greatest possible extent by means of sweeping movements in which the point of the needle travels from before backward. The difference between discission through the cornea and that through the sclera lies in the fact that we are able, by the latter method, to bring a much greater force to bear upon the cataract with the needle and even depress it into the vitreous.

Discission is adapted for all membranous cataracts, provided they are

[3] [From *sclera* and νύττειν, to prick.]

not too thick, and that there are no extensive adhesions of the cataract to the iris. Discission is frequently done as a secondary operation after the extraction of cataract, to remove a secondary cataract.

Scleronyxis is adapted only to those cases in which there are but few, if any, portions of the lens left which are able to swell up. In soft, non-shrunken cataracts, which it is designed to subject to the processes of swelling and resorption, one should not make discission through the sclera, for in that case, in order to split the anterior capsule, we would have to pass the needle through the entire lens and break the latter all to pieces—a procedure which, apart from the fact that we might easily luxate the whole lens in doing it, would give rise to excessive and violent swelling.

Dilaceration of a membranous cataract is an operation productive of but little disturbance as long as there are no adhesions between the cataract and the iris. In the latter case there is danger of undue traction being made upon the iris, with consequent irido-cyclitis. Simple discission should be made only when the cataractous membrane is thin enough to be torn apart without being pulled upon. In the case of rather thicker membranes the operation can be done according to the method proposed by Bowman. In this two needles are passed through the cornea at the same time, one near the inner, the other close to the outer corneal margin. Then the points of the needles are plunged into the center of the membrane and drawn apart by sweeping movements. Thus the membrane is torn in such a way that the part pulled upon lies between the two needle-points—i. e., in the center of the cataract—while the iris remains free from traction of any kind. Another and much better method of avoiding making traction in the operation [particularly when the membrane is tough] consists not in tearing it with a needle, but in dividing it with a sharp Graefe knife [or Knapp's knife-needle or DeWecker's scissors] introduced through the cornea [see Fig. 451]. Sometimes there is an indication for carrying the section on into the iris, so that the capsulotomy is combined with an iridotomy.

II. Extractio Cataractæ

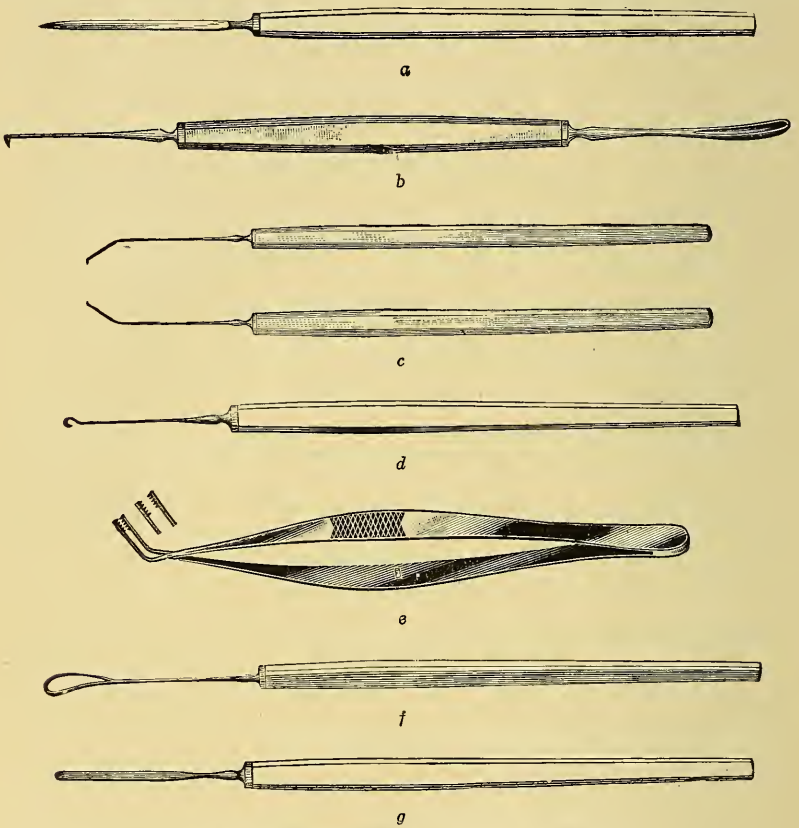
868. The object of cataract extraction is to remove the lens from the eye immediately, and as completely as possible. It consists essentially of three steps: 1. The making of a section whose dimensions vary in accordance with the size and consistence of the cataract. This section may lie in the cornea or in the sclera. 2. Opening of the anterior capsule in order to allow the lens to escape from it. 3. Expulsion (delivery) of the lens by pressure exerted upon the eye. In many cases a fourth step is added to the operation—namely, the excision of a portion of the iris. This iridectomy is regularly performed directly after the completion of the section. The most usual methods of extraction are:

(*a*) *Linear Extraction*

869. Linear extraction is so-called because the section is at least approximately linear (see page 917). Linear extraction, like discission, is performed both in soft and in membranous cataracts, and consequently is done in two different ways, which are modifications of each other :

1. In operating upon a *soft cataract* the lance knife is introduced at the limbus in the lower margin of the cornea, during which manœuvre the blade must be held parallel to the corneal margin. [H. Knapp, followed

by others, made the incision in clear cornea 2 mm. from the periphery of the latter.—D.] The lance is then pushed forward until the wound in the lower margin of the cornea has a length of 4 to 7 mm. (*S S*^{1}, Fig. 453.) [In order that the incision should be sufficiently wide a very broad keratome should be used (Wilder).—D.] Then the lens capsule must be very



[Fig. 452.—Instruments for Cataract Extraction.

a, Graefe's cataract knife (linear knife). Used also in paracentesis, iridectomy, iridotomy, sclerotomy and discission. (Pages 968, 974, 981, 983, 990, 991, and 1006.) *b*, cystitome and Daviel's spoon. (Pages 968 and 994.) *c*, Knapp's cystitome (right and left), used in cataract extraction without iridectomy. *d*, Tyrrel's sharp hook for tearing the capsule. The blunt hook (Fig. 444, *f*) is also occasionally used. *e*, capsule forceps. The Kalt capsule forceps used in extraction in the capsule (page 997) has cup-shaped and flattened extremities without teeth. *f*, wire loop. *g*, spatula to reduce the iris and stroke it out smooth. Other instruments are a speculum and fixing forceps (see Fig. 400) and if the cataract is done with iridectomy an iridectomy forceps and scissors (see Fig. 444).—D.]

thoroughly torn up in the area of the pupil, which has been previously dilated by means of atropine. For opening the capsule we may use either a discission needle, a sharp hook (Fig. 452, *d*) or a capsule forceps (Fig. 452, *e*). After this the lens masses are discharged by making pressure on the eyeball with the finger through the upper lid and at the same time depressing the peripheral lip of the wound with a Daviel's spoon. By this means the

contents of the eyeball are subjected to quite a great pressure, and the wound is made to gape open. This manœuvre is repeated until all parts of the lens have been removed from the eye. [The lens masses may also be removed by irrigation (Wilder).]

If linear extraction is done to remove a lens that has been swollen up by a preliminary discission, the opening of the capsule is omitted, since the capsule has already been torn open by the discission.

Fig. 453.—Linear Extraction. Magnified 2×1. Pupil dilated with atropine.

2. When a *membranous cataract* is to be operated upon, the section is made in the same way. Then a sharp [or blunt] hook or a forceps is introduced through the section, and with these instruments the cataractous membrane is grasped and drawn out through the wound.

The advantages of linear extraction consist in the fact that the section is short and passes through the cornea obliquely, for which reason it closes readily, does not necessitate an iridectomy, and does not require any very strict after-treatment. But owing to this very fact that the section is so short, this method is adapted only to membranous or to soft cataracts—i. e., to those that have no hard nucleus, since the latter could be removed through such a wound only with difficulty, or could not be removed at all. [As pointed out by Beard, Wilder, and others, linear extraction may be used in cataracts—especially lamellar, traumatic, shrunken and membranous cataracts—occurring in people under 40, and may sometimes be performed up to the age of 45.—D.]

(*b*) *Flap Extraction*

870. Technique.—This operation produces in the margin of the cornea a curved section of an extent requisite for the removal of large, hard cataracts. The indication for its performance is furnished by all cataracts which have a hard nucleus [of any size] and are hence not adapted either for discission or for linear extraction. The operation consists of four steps:

First step: Performance of the section. This is done with the Graefe linear knife, which is introduced in the limbus at *S* (Fig. 454), in such a way that the cutting edge looks upward. Then the knife is passed through the anterior chamber to the point of counter-puncture, S^1. The counter-puncture should lie exactly opposite the site of the puncture, the two being, at such a level that the upper third or fourth of the cornea is separated by the section so as to form a flap. [H. Knapp, whose experience and judgment rightly carry the greatest weight, advised that the puncture be made about 1 mm. above the temporal end, and the counter-puncture 1 mm. above the nasal end of the horizontal meridian. (See also page 995.)—D.] [After the transfixion has been made, the section is completed by sawing

cuts, so that through its whole extent it divides the sclera just behind the limbus. As soon as the knife has cut through the sclera, it lies behind the conjunctiva, by cutting which a conjunctival flap is formed about 2 mm. broad. In doing this it is advisable to turn the knife rapidly upward so as to divide the conjunctiva quickly. Otherwise, since the conjunctiva is extensible and hence not easy to divide, the knife would strip it up from the sclera a long way back, and the conjunctival flap would become too broad.

The section is made upward, so that in case iridectomy is performed the coloboma, too, may be situated above and be covered by the upper lid. The section performed as above described is long enough for the largest-sized cataracts. If we have a cataract to operate upon whose nucleus is probably small, we may make a section of correspondingly smaller size.

Second step: Iridectomy. After the conjunctival flap has been turned down upon the cornea, so that the wound may be exposed to view, the latter is entered with the iris forceps, the iris is grasped close to the pupillary margin, drawn out, and cut off with a single sweep of the scissors.

It is best to make the excision of the iris as slender as possible (Fig. 454). With this object in view, we draw the iris from the wound only far enough for its pupillary margin to become visible, and then, holding the scissors forceps perpendicular to the direction of the wound, snip off simply the apex of the tag of iris. A small coloboma averts prolapse of the iris as certainly as does a large one (see pages 923 and 996), and causes less confusion from dazzling.

Third step: Opening of the capsule. This is performed with the capsule forceps, the delicate teeth of which are directed backward (Fig. 452, *e*). The forceps, closed, is introduced and passed on in the anterior chamber until the center of the pupil has been reached. Here the forceps is allowed to open, and by the exertion of light pressure the capsule is grasped over as large an extent as possible, and is drawn out of the wound.

The opening of the capsule was made by Von Graefe with a cystitome—i. e., with a triangular cutting lancet (Fig. 452, *b* and *c*), and by others was made with the discission needle or with a sharp hook (Fig. 452, *d*). An important improvement has been the introduction of the capsule forceps. With this the anterior capsule is not only split, but also has a piece taken out of it. Thus, the capsular wound is prevented from closing quickly, and by its closure interfering with the resorption of the fragments of the lens that remain. Since the employment of the capsule forceps, secondary cataract has become much less frequent, although at present unripe cataracts are operated upon much more often than formerly.

Fourth step: Expulsion of the lens. The Daviel's spoon is placed parallel to the wound against the lowermost part of the cornea, and light pressure is exerted with it upon the latter upward and backward. Instead of the spoon, we may use the finger, pressing with this through the lower lid upon the region of the lower margin of the cornea. The pressure must be stopped the instant the greatest diameter of the lens has passed through the wound.

After the operation is finished, the "*toilet*" of the eye is next in order. The portions of the cataract which are still left in the eye, and also any extravasated blood, are removed by stroking with the lower lid,[4] the iris is replaced from the wound into the anterior chamber by the introduction of the spatula, until the pillars of the coloboma have the proper position (see page 921), then the conjunctival flap is stroked out smooth, and the eye is bandaged.

Many operators follow the extraction with *irrigation of the anterior chamber* with weak antiseptic solutions, partly in order to wash out the fragments of lens which remain behind, and partly to disinfect the interior of the eye. I have employed irrigation pretty often, but have sometimes seen permanent opacity of the cornea or iritis result from it, so that I have given it up again. [Irrigation has still many advocates. If made with normal (or, better, half-strength normal) salt solution or boric acid it has little tendency to produce opacity of the cornea or iritis. Special instruments have been devised for it (syringe of Mac Keown, Lippincott's irrigator), but a simple rubber bulb with slender, flattened glass tip answers all requirements.—D.]

Some operators, after the operation is concluded, sew on conjunctival flaps; others [Kalt, Williams] suture together the lips of the corneal wound. But if we operate with a conjunctival flap, the latter lies so well on its bed and becomes adherent to the latter so quickly that suturing is superfluous. I apply a suture only in case the wound gapes a good deal after there has been loss of vitreous or in case the corneal edges show a tendency to turn over. [Among those who cover in the wound with preformed conjunctival flaps are Stanculeanu, Czermak, and van Lint. The objections to such flaps, as summarized by Major Smith, are increased time and traumatism in making the operation, obscuration of the field by blood and loose conjunctiva, difficulty in making the incision of the proper size, and increased difficulty in extracting the lens and replacing the iris.—D.]

871. Varieties of Section.—With regard to the way *of making the section*, different operators differ, some making it in the transparent cornea (Fig. 455), in which case no conjunctival flap is formed, while others carry the section through the marginal portions of the conjunctiva covered by the limbus, or even through the adjoining sclera (Fig. 454), so that after making the division they bring the knife up under the conjunctiva, and form a flap out of it. This conjunctival flap has the advantage of becoming very quickly agglutinated to the subjacent parts, and so closes the wound externally even when the edges of the incision in the cornea or sclera have not yet united. It thus protects the wound from subsequent infection.

872. Simple Extraction.—Of the four steps of the operation the second may be omitted, and the operation done *without iridectomy*. [The operation is then called simple extraction, as distinguished from the combined extraction, or operation with iridectomy.—D.] In the latter case the iris must be carefully replaced after the operation is finished, and then eserine[5] must be instilled in order to prevent by the contraction of the pupil any subsequent prolapse of the iris (Fig. 455). A position midway between the oper-

[4] [Or with a spatula —D.] [5] [This is omitted by many operators.—D.]

ation with and without iridectomy is the modification proposed by Pflüger and Hess. This consists in exsecting, after the delivery of the lens has been effected, a very small bit of that part of the periphery of the iris which lies directly beneath the wound. The small hole thus made is concealed by the limbus and by the uper lid, so that the cosmetic and optical effect of the operations is like that of an operation without iridectomy, while yet the danger of a subsequent prolapse of the iris is rendered very much less (see page 923). [This buttonhole iridectomy was devised by Chandler, who calls the

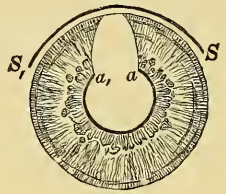


Fig. 454.

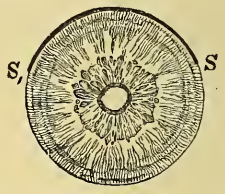


Fig. 455.

Fig. 454.—Flap Extraction with Iridectomy. Magnified 2×1. *S* S_1, scleral section, concentric with the limbus. a_1 *a*, edges of the sphincter.

Fig. 455.—Flap Extraction without Iridectomy. Magnified 2×1. *S* S_1, corneal section, which everywhere lies in the limbus. The pupil is greatly contracted by means of eserine. As a consequence of this marked miosis, the pupil is not perfectly circular, but somewhat irregular, and the fringe of pigment lining it has become broader.

operation when so made "modified simple extraction." Chandler points out that the retained cortical lens matter can be expelled through the hole thus made.—D.]

The omission of iridectomy has the advantage of keeping the patient's pupil round and mobile, but it also entails many disadvantages, which limit the number of cases in which extraction without iridectomy is indicated. Thus: 1. The delivery of the lens is more difficult without iridectomy, because the lens has to be expelled through the narrow pupil, and to accomplish this pretty strong pressure is required. Hence, this method is not adapted to those cases in which a very easy delivery of the lens is desirable, as, for instance, in cases of tremulousness of the lens in which any kind of strong pressure would produce rupture of the zonula and hyaloid membrane, and consequently prolapse of the vitreous. 2. When the pupil is narrow the removal of cataractous remnants is difficult, for which reason the operation with iridectomy is preferable for unripe cataracts. 3. Extraction without iridectomy is not adapted to cases in which there is a complicated cataract connected with the iris by synechiæ. 4. In spite of the use of eserine, prolapse of the iris may take place in the days following the operation. In this case we are obliged to make a secondary excision of the prolapsed iris. Accordingly, extraction without iridectomy is not adapted to cases which show a great tendency toward prolapse of the iris, nor to those in which we can not count upon the patient's remaining quiet after the operation. It may also happen that an operator may, after taking all these facts into consideration, have decided upon an extraction without iridectomy, and yet in the course of the operation may find himself compelled to excise the iris. This is the case, for instance, when the pupillary portion of the iris is so unyielding (as it often is in old people) that it does not allow the cataract to pass through the pupil, and the latter has to be widened by an iridectomy before the passage can take place. In other cases the delivery of the lens goes on well, but the iris shows a tendency, in spite of careful reposition, to fall again into the wound. In this event it is better to cut it off at once than to run the risk of a subsequent prolapse of the iris. We may therefore say: Flap extraction without iridectomy gives under favorable circumstances

the most perfect result, but is not adapted to all cases, and in many cases it can not be done at all; moreover, owing to the danger of a subsequent prolapse of the iris, it does not attain to the almost absolute certainty of success that belongs to flap extraction with iridectomy.

873. Extraction in the Capsule.—The ideal cataract operation would be to remove the lens in the closed capsule, so that no residue of the cataract would remain, no tags of capsule could be incarcerated in the wound and no after-cataract could form by thickening and folding of the capsule that had been left behind. In delivering the lens in its capsule it is desirable to combine the section with an iridectomy, in order that the exit of the lens may be easy. The exit is effected by strong pressure on the lower part of the cornea. But this, again, has the disadvantage that it often produces escape of vitreous and thus renders the operation more serious. The ideal results obtaining in successful cases of this method of operating, which was proposed by Smith, are therefore purchased at the expense of quite a large number of losses.

[Extraction of the lens in its capsule was proposed by Pagenstecher for cataracts with thickened capsule. More recently it has been advocated as a routine method of dealing with cataracts of all kinds by Major *Smith* of India, who has operated on an enormous number of cases with great success. In the operation practised by him, as described by Vail, the eye is first thoroughly douched with 1:2000 bichloride solution. Puncture and counter-puncture are made in the limbus and about in the horizontal meridian of the cornea, the blade of the knife being turned forward so as to cut the cornea more at a right angle and make a perfectly smooth incision which lies wholly in the cornea, but still is large. Iridectomy is then done, special care being taken not to injure the lens capsule. In the expression the assistant holds the lids far from the eye with fingers and with a blunt hook, the patient looks up, and the operator with another blunt hook presses on the cornea till the lens breaks loose and tilts forward, and with the same hook, engages the lens as it comes out. A spoon is placed behind the lens only if vitreous presents, and then not to lift the lens out but to keep the vitreous back. Many doubt whether the final results of the operation are as good as those of extraction with capsulotomy. Outside of India it has been adopted by few operators.—D.]

[*Extraction after Subluxation with Capsule Forceps.*—To avoid the dangers of undue pressure and consequent loss of vitreous, that obtain in the Smith operation, A. Knapp subluxates the lens by traction instead of by pressure. He makes an incision just short of half the corneal circumference and with a conjunctival flap. After making an iridectomy he grasps the lens capsule below its center with a Kalt capsule forceps, which takes firm hold of the capsule without tearing it (see description Fig. 45, *e*). With this he rocks the lens until the lower portion of the capsule ruptures and the lens subluxates. Then withdrawing the forceps, he makes pressure straight backward on the lower part of the cornea until the lens turns over and presents. The part of the capsule that still remains adherent to the lens is separated by a lateral stroking motion. A similar operation has been proposed by Stanculeanu. Török grasps the capsule with the Kalt forceps and by making lateral and circular movements with this loosens the zonular attachments; then expels the lens by traction with the forceps combined with intermittent pressure on the sclera with the Daviel spoon.—D.]

874. Other Modifications of the Operation.—(*a*) We give the name of *preliminary iridectomy* to one that is made several weeks before a cataract operation. Many operators perform such an iridectomy even in uncomplicated cataracts because they believe

that thus they make the cataract operation itself less severe and hence less dangerous. But preliminary iridectomy is done particularly in unripe cataracts in order to ripen them (*maturation*). This operation which was devised by Förster consists in rubbing the cornea in a circular direction with a blunt instrument (Daviel's spoon or a squint hook) after the excision of the iris. Since the cornea is so thin that it is dimpled by the rubbing, the latter in the pupillary area acts also upon the lens whose capsular epithelium in this area is in part lacerated and detached. Hence the capsule is made permeable for the aqueous, by means of which the lens fibers are opacified. The massage of the lens results in the latter's becoming completely opaque within a few weeks or even a few days. The extraction of the lens should follow this preparatory iridectomy not sooner than four weeks.

Like many others I have pretty much given up preliminary iridectomy as superfluous, except in those cases in which there are complicated cataracts. In such cases a preliminary iridectomy must above all be made when the cataract is associated with increase of tension because otherwise we should run the risk of having a destructive intra-ocular hæmorrhage (see page 924).

Ripening by Förster's method I also consider as an operation that we can dispense with. We can without waiting extract even unripe cataracts with success if we open the capsule quite extensively with the capsule forceps.

(*b*) [The *preliminary capsulotomy* of Homer Smith is done especially in immature cataract. The capsule is opened with a scalpel-shaped knife-needle four to six hours before the extraction. The aqueous thus gains access to the lens substance, causing the soft portions of the latter to swell and become loosened from the capsule, so that when the extraction is made they come out easily.—D.]

(*c*) *Wenzel's Extraction.*—When owing to severe irido-cyclitis there is an adhesion between the surface of the iris and the lens, excision of the iris in the regular way is impossible. We then cut through the iris and open the capsule at the time of making the section in the cornea, by carrying the knife not only through the cornea, but also through the iris and lens capsule, which can easily be done since the anterior chamber in such cases is generally shallow (Wenzel).

875. Accidents Occurring in the Operation for Cataract.—The extraction may be made difficult or fail altogether, owing to accidents of various nature. Many of these are the fault of the operator. If the section proves to be too short or if the capsule is insufficiently opened, the delivery of the lens is difficult or impossible. In this case the section must be enlarged,[6] or the capsule must be again ruptured, and this time more thoroughly. If the operator exerts too strong a pressure with his instruments upon the eyeball as a whole, or upon the iris or lens, the zonula ruptures and the vitreous gushes out. The greater the operator's skill grows with practice, the less frequently do these unlucky accidents happen to him. But there are other accidents which are caused by the abnormal condition of the eye that is operated upon, and in that case it generally does not lie in the power of the operator to prevent them. The most frequent of these accidents is *prolapse of the vitreous.* This takes place when the zonula ruptures. Such rupture not infrequently happens because the patient screws his lids tightly together and thus presses upon the eyeball. It also occurs when the zonula was defective before the

[6] [This is done with slender but strong and sharp blunt-pointed scissors (Stevens's tenotomy scissors.—D.]

operation, and hence especially in hypermature and in complicated cataract. The significance of prolapse of the vitreous for the subsequent course of the operation differs according as it takes place before or after the delivery of the lens. In the former case the lens cannot be evacuated in the usual manner by pressure exerted upon the eye; for then the larger part of the vitreous would escape before the lens itself came away. Hence, the lens must be drawn out of the eye with instruments—i. e., extracted in the true sense of the word. For this purpose the proper instruments are Weber's loop (Fig. 452, *f*) or Reisinger's [or Agnew's] double hook, which are introduced behind the lens and lift it out by force of traction.

Prolapse of the vitreous is much less to be dreaded when it takes place after delivery of the lens. The most serious harm that prolapse then does is that it hinders the accurate replacement of the iris, and also that the prolapsed vitreous lies between the lips of the wound and prevents their accurate coaptation. The vitreous may also give rise to suppuration of the wound, since it is very apt to become infected.

[If the vitreous escapes in any quantity, there will be more or less collapse of the eyeball. This may be remedied by injecting warm sterile salt solution into the anterior chamber (see page 923).—D.]

[For hæmorrhage after cataract extractions, see page 923.—D.]

A rare but unpleasant accident is that in which the lens, before being delivered, becomes *luxated*, and disappears into the vitreous from which ordinarily it can not be extracted [but see page 1005.]

876. Result of the Cataract Extraction.—An eye whose lens has been removed is *aphakic*. It presents the following appearance when the operation and the healing of the wound have pursued a normal course: The cicatrix left by the operation, if situated in the cornea, appears as a narrow gray line; if the section has been made in the limbus or in the sclera, the resulting cicatrix becomes later on almost unrecognizable. The anterior chamber is abnormally deep, the iris is tremulous and, when the operation has been done with iridectomy, presents a coloboma above. The pupil is of a pure black, but on lateral illumination presents to view a membrane of silky luster, which not infrequently is thrown into folds. This is the lens capsule which was left in the eye when the lens was removed. It is, however, only the posterior capsule of the lens (*h*, Fig. 456) that is intact throughout. The anterior capsule ($v\ v_1$) where it occupies the area of the pupil, is lacerated and in part deficient; the remains of it are applied directly to the posterior capsule. Since both capsules are transparent, the pupil appears round and black. Behind the iris the anterior capsule, where it was protected from the instrument used for making the opening, is preserved intact, and in conjunction with the posterior capsule incloses remains of the lens, which correspond to what was once the equator of the latter (Fig. 456, *k*). As the anterior and posterior capsules become agglutinated together in the pupillary area, these remains of the lens are

shut off from the aqueous, and can not therefore be absorbed; in fact, they usually increase in amount, owing to *proliferation* of the cells of the capsule. They then form an annular swelling lying behind the iris (Soemmering's crystalline swelling). The lumen of the ring, which corresponds to the pupil, is closed by a thin, transparent membrane consisting of the two apposed layers of the capsule. As the opaque and swollen mass behind the iris is completely concealed, it in no way interferes with vision. If the operation has been made with an iridectomy, the mass is wanting in the course of the coloboma, because the anterior capsule was opened there likewise.

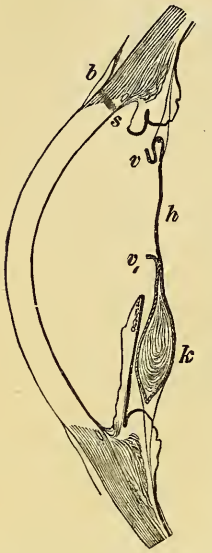


FIG. 456.—CROSS SECTION THROUGH THE ANTERIOR SEGMENT OF AN EYE, UPON WHICH AN EXTRACTION HAS BEEN PERFORMED BY MEANS OF A FLAP SECTION. Magnified 4 x 1.

The section, *s*, which was made upward lies by its inner portion in the cornea, by its outer portion in the sclera; the latter is covered by the conjunctival flap, *b*. At a point corresponding to the section the iris is wanting, except for a short stump. The anterior capsule presents a large aperture, the edges of which (v v_1) are curled over, while the posterior capsule, *h*, although slightly wrinkled, is unruptured. In the lower part of the eye behind the iris the remains of the lens, which are inclosed in the folds of the capsule, form Soemmering's crystalline swelling, *k*, which is wanting in the parts above that correspond to the coloboma.

Without glasses the *sight* of persons who have been operated upon for cataract is just sufficient to allow them to go about alone or to do very coarse work. Distinct vision is possible only with the aid of convex glasses, since by the removal of the lens the refractive power of the eye has become too small, and hence there is a high degree of hypermetropia. If the eye was emmetropic before the operation, the hypermetropia afterward amounts on an average to from 10 to 12 D. It is otherwise in cases in which an error of refraction already existed beforehand. If a hypermetropia was present previously, it enhances that which is acquired by the operation. If, on the other hand, the eye was myopic before the operation, the subsequent hypermetropia is less; extremely myopic eyes may actually become emmetropic after a cataract operation, or even remain a little myopic.[7] The aphakic eye, moreover, is destitute of accommodation. The eye is incapable of altering its refractive state. Hence, it follows that by a single glass the latter is corrected for a single distance only. Accordingly, an eye that has been operated upon for cataract needs at least two glasses—one for distance, and the other for near. [Owing to the alteration in the corneal curvature produced by the contraction of the operation scar, usually a considerable amount (from 1 to 4 D) of inverse astigmatism is produced by the cataract operation. This usually diminishes during the

[7] [If the eye was myopic before the extraction, the amount of residual hypermetropia depends upon whether the myopia was all axial or was also partly due to increased curvature of the cornea. In the former case, if H denotes the residual hypermetropia in dioptries and M the pre-existing myopia then, generally speaking, $H = 11 - \frac{M}{2}$. If M is over 22 D, then H becomes negative, i. e., represents too many dioptries of residual myopia.—D.]

first few months, so that the ultimate correction by glasses may be materially different from the immediate correction.—D.]

For *erythropsia* after cataract extraction see page 22.

877. After-Cataract.—It often happens, even in cases in which the operation has been well performed, that the result of the operation is impaired by the *retention of portions of the cataract*. This happens particularly when the operation is done on immature cataracts, but by no means fails to occur also in those that are mature and hypermature. If the anterior capsule is thoroughly opened, the portions of the lens left behind (if they were not already opaque previously) grow opaque, swell up, and become absorbed. In this case, therefore, a pure black pupil is ultimately obtained. But if the layers of the capsule become agglutinated early and shut off the remains of lens substance from the aqueous, these remains are not absorbed but persist as a white membranous opacity. This is called after-cataract (*cataracta secundaria*). If this is present in only one part of the pupil while another part of it is quite clear, the sight may be perfect. But if the whole pupil is filled by the secondary cataract, the sight is diminished in proportion to the density of the opacity. It may also happen that the after-cataract does not develop until later on; the epithelium of the anterior capsule which has been left behind proliferating and inducing a secondary thickening and opacity in the latter. Similarly the capsule, even without becoming opaque, may induce diminution in sight, if in the course of time it becomes more and more wrinkled and thus causes irregular refraction of the rays of light.

After-cataract, when it interferes with sight, requires a secondary operation—namely, discission, or linear extraction. The secondary operation should not be performed until the eye ceases to show any trace of irritation, and in no case sooner than two weeks after the cataract extraction.

The result of a cataract operation may furthermore be impaired by *inflammation* (see page 928). If suppuration of the wound sets in, the eye is almost always lost. If irido-cyclitis develops, the secondary cataract is united by the exudate which is formed, to the iris and even to the ciliary processes (cataracta secundaria accreta). It depends upon the condition of the light perception whether the sight in such a case can be restored or not by a secondary operation (iridectomy or iridotomy).

878. History of Cataract Operations.—In the preceding pages it has been shown that there are various ways open to us for removing cataract. We may either subject it to resorption by means of discission, we may tear a hole in it by dilaceration, or we may remove it altogether from the eye. But not even by this list are all the methods of restoring the sight of an eye blind with cataract exhausted. We might also, instead of removing the opaque lens, push it away from its place behind the pupil so that the latter becomes free again. This artificial luxation is not only practicable, but as a

matter of fact it has been practised for a thousand years; it is the oldest method of operating for cataract. This method, called *depression of cataract*[8] (depressio cataractæ), was made in the following way: A needle was passed into the sclera on the outer side of the margin of the cornea and about 4 mm. behind it, and it was pushed forward until at length it lay against the upper border of the lens. Then the point of the needle was lowered by a sweeping movement, and the lens was thus depressed into the vitreous. The moment this was done the pupil became black and the patient regained his sight. This was the only method of operating upon cataract practised in ancient times and throughout the Middle Ages. As time went on it was modified in various ways. The last and most important modification consisted in turning the lens over instead of depressing it. The needle in this case was passed by the margin of the pupil and into the anterior chamber, and with it pressure was made upon the upper part of the anterior surface of the lens. The latter was thus turned over in such a way that its anterior surface looked upward, its posterior surface downward. This procedure was called *reclinatio cataractæ*.

The operation above mentioned, or "cataract pricking," was, as a rule, practised by special physicians. In the Middle Ages these went from one annual fair to another, and there operated upon those who were blind with cataract. When the operation had been successfully performed and the honorarium had been paid, the "cataract pricker" traveled to another place. He did not see his patient again after the operation, and it was a good thing for him that he did not, for the later consequences of the operation were as melancholy as the immediate result was brilliant. For the eyes very often were destroyed either by inflammation or by increase of tension. The inflammation probably was caused as a rule by infection with the cataract needle and not infrequently was transmitted to the other eye in the form of a sympathetic ophthalmia. At present inflammation might generally be avoided by operating aseptically, but we have no means of combating the other deleterious consequences of depression or reclination, and particularly the increase of tension that so frequently occurs when the lens is luxated. Hence the repeated attempts that have been made to take up reclination in recent times have always been abandoned again. [The operation is still much practised by native physicians in India.—D.]

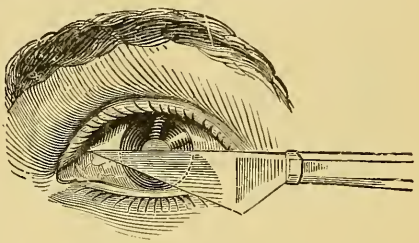
It sometimes happened after depression or reclination that the lens failed to remain in its place in the vitreous, particularly when the latter was liquefied. In such cases either immediately after operation or later, in some cases not till years afterward, it rises and places itself in its old position behind the pupil; it may even pass through the pupil into the anterior chamber. Such cases of luxation of the lens into the anterior chamber gave the first occasion for the performance of *extraction* of cataract. This method of operating, if we are to believe some authors, was perhaps practised now and then even in ancient times, but at any rate it had in the Middle Ages fallen completely into oblivion. The first information that we have in regard to it we get from the seventeenth century, when there are several instances in which the lens was removed from the anterior chamber into which it had got after the operation of depression. The Frenchman *Daviel* had already done this in several cases, when in the year 1745 he first dared to undertake this operation upon a cataract which was situated in its normal position. In so doing Daviel inaugurated a new era in the history of cataract operations, since from that time the extraction of cataract began more and more to take the place of depression.

The original method of Daviel was naturally very much in need of improvement. Of the many modifications which it underwent in the course of time the last and best was that of *Beer*. The latter made the section with a knife invented by himself [Fig.

[8] [Also couching or displacement of cataract.—D.]

457], which broadens from point to handle so as to have a wedge shape. With Beer's cataract knife it is possible to complete the section by simply pushing the knife forward after it has been entered, and owing to this fact the section acquires a high degree of regularity. The section ran somewhat inside of the limbus, and separated exactly the lower half of the cornea from the sclera. Then, after opening the capsule, the lens was delivered, but no part of the iris was excised.

Beer's procedure was soon generally adopted, and was for a long time the prevailing method. In successful cases it gave ideal results. The pupil was black, round, and perfectly movable, and it was only upon close examination of the eye that it could be discovered that an operation for cataract had been performed at all. Unfortunately, it always happened that a considerable number of eyes were lost after this operation, especially by suppuration of the cornea. As at that time it was not known that this was caused by infection of the wound, the method of operating, and particularly the way in which the section was made, were regarded as accountable for it. Hence, other better procedures were sought after, and this time Von Graefe was the one to take the most important step forward and create a revolution in the methods of performing extraction, by the invention of his method.



[Fig. 457.
Beer's operation for cataract by inferior flap with a Beer's knife. Beer's knife is also used for the ablation of staphylomata of the cornea (page 969).—D.]

Von Graefe considered that the cause of the suppuration of the cornea in Beer's method lay in the shape of the section which was made with a flap. This gives rise to great gaping of the incision, in consequence of which the lips of the wounds are not properly applied to one another, and this fact was supposed to furnish the cause of the suppuration. Von Graefe accordingly believed that the linear incisions were preferable, as he had become convinced of the promptness with which they healed in the case of simple linear extraction, an operation which had already been practised by him. Accordingly, he, as well as others, attempted to apply the *linear section* which was made with the lance knife, and which originally was employed only for soft or shrunken cataracts, to large cataracts with a hard nucleus. With the object these experimenters tried to make the linear incision as large as possible by placing it in the upper part of the cornea, and by combining it with iridectomy. Others tried to diminish the size of the lens first by crushing it so as to be able to extract it through the section. But these attempts were all unsuccessful. The section always remained too small for the cataract, which in its passage contused the lips of the wound, so that inflammation frequently ensued. *Jacobson,* who sought the remedy in another way, obtained better results. He placed the section in the sclera. He gave up the linear character of the section, and made a flap incision, skirting the lower margin of the cornea, but situated still in the sclera. With this he combined iridectomy. This method gave better results, and particularly a less frequent suppuration of the wound. The cause of this was regarded as consisting in the fact that the sclera, being a vascular tissue, is less disposed to suppuration than the non-vascular and hence more poorly nourished cornea.

Von Graefe now attempted to combine in a new method both advantages—namely, the linear character of the section, which ensures a good coaptation of the edges of the wound, and the position in the sclera, which affords protection against suppuration of the wound. It soon became apparent to him that a linear section, which should

be of the necessary length and situated in the sclera, could not be performed with the lance-shaped knife. The lance knife must be pushed forward parallel with the plane of the iris, and therefore, as soon as it is desired to make a wound of any length at all, produces a section which is nearly concentric with the margin of the cornea, and hence has the character of a flap. Von Graefe accordingly devised the narrow or linear knife, which soon proved to be one of the most useful instruments in ophthalmology. With this knife he performed the section in such a way that at its center it was in contact with the summit of the cornea, but at its ends was removed a considerable distance from the corneal margin. The point of entry is determined by means of a tangent ($t\,t_1$, Fig. 458), which is conceived to be drawn through the external margin of the cornea; the puncture (s) is situated in this and at that point of it where it is at a distance of 1 to 1½ mm. from the margin of the cornea. The point of emergence, s_1, lies directly opposite the point of entry. While the section is being performed, the edge of the knife, which at first was directed straight upward, is turned a little forward, so that the center of the section gets to lie right behind the limbus. The new way of making the section had the conjunctival flap and iridectomy as its necessary concomitants. The iridectomy had to be performed as a regular thing, since otherwise the iris, owing to the peripheral situation of the wound, would certainly have become incarcerated in it. (In the old methods of extraction iridectomy was done only when there was some necessity for it.) As cataract operations combined with iridectomy were called "modified" operations (so named in contradistinction to those that are "simple," i. e., performed without iridectomy), Von Graefe called his new method "*modified linear extraction.*" Later on, one made a virtue of necessity, and laid special stress upon the advantages of the iridectomy that was combined with the extraction. It prevented, they said, the incarceration of the iris, made it possible to open the capsule more thoroughly, facilitated the removal of the remains of the cataract, and afforded a protection from subsequent inflammation of the iris. Hence, people soon got to regard the excision of the iris as one of the additional advantages of the new method.

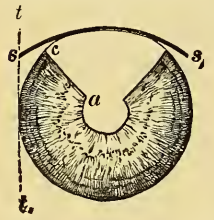


FIG. 458.—MODIFIED LINEAR EXTRACTION BY VON GRAEFE'S METHOD. Magnified 2×1.

The section, $s\,s_1$, lies in the sclera; the iris shows a large coloboma with very divergent limbs, $a\,c$.

The results of Von Graefe's linear section were, in fact, much better than those which the earlier methods had given. Suppuration of the wound, in particular, had become less frequent. But yet the method had its dark side, too. Its performance required more operative skill, and the delivery of the lens was made more difficult, owing to the slight tendency to gape that the wound possessed. Other disadvantages arose from the peripheral situation of the section, which brought the latter, especially at its extremities, close to the zonula and the ciliary body. Prolapse of the vitreous was frequently met with, and also inclusion of the limbs of the coloboma in the wound. While suppuration of the wound proved to be less frequent, iritis and irido-cyclitis became proportionately more common, and in consequence sympathetic disease of the other eye was observed more frequently than before. These facts induced operators to depart more and more from this section, which was felt to be too peripheral in its situation, and in particular led them to place the extremities of the latter nearer the cornea. If Von Graefe's original section, as Von Graefe himself gave it, was itself not a pure linear incision, this is still more the case with the section as it was subsequently performed. It had become a curved section, forming an arc of small altitude. Under this somewhat modified form, scleral extraction soon became the method that was generally employed.

Since with the introduction of the antiseptic method the danger of suppuration

of the wound had been reduced to a minimum, operators no longer hesitated to make the section in the limbus or in the transparent cornea itself—as for example, is done in the corneal flap extraction above described. Other improvements that have been made have had regard to the *excision of the iris*. Operators had already learned by careful reposition of the iris to avoid the dangers arising from its incarceration. When this is done there is no need of making a large coloboma such as Von Graefe had described—indeed it is better to make the iridectomy as slender as possible.

When at length surgeons again adopted the corneal section, they took the last step and operated entirely *without iridectomy*, as Daviel and Beer had done in former times.

879. [Removal of a Dislocated Lens.—Dislocation of the lens into the anterior chamber is uncommon; dislocation into the vitreous is more frequent. Dislocation may occur in the course of a cataract extraction or as the result of accidental injuries; or, it may be spontaneous and in that case is sometimes congenital.

A lens dislocated into the anterior chamber should always be removed. The lens if soft, can be needled and the fragments be removed by linear extraction. If the lens has a large hard nucleus, a sufficiently large incision should be made, through which the lens can be lifted out with a hook and spatula or wire loop. In order to prevent the lens slipping back through the pupil, the latter should be contracted with eserine.

A lens dislocated into the vitreous causes trouble sooner or later. When it does so it should be removed. In the case of a lens dislocated after cataract extraction it is sometimes possible to expel it by Knapp's method of making systematic pressure with the lower lid against the lower part of the sclera right toward the center of the eyeball (De Schweinitz). Otherwise, especially in the cases of spontaneous dislocation, in which the vitreous is usually fluid, the lens may either be lifted out with a delicate wire loop or flat (Smith) spatula, or be drawn out with a bident or double hook (Agnew) or with a not too curved sharp Tyrrell's hook (Fig. 452, *d*). The hook, with which the lens can often be slid along the anterior wall of the vitreous chamber with but little disturbance of the vitreous, serves better than the loop, unless the lens has sunk to the bottom of the chamber, where the hook might readily become entangled in the tunics of the eye (Beard). In suitable cases the hook and spatula may be used simultaneously, traction being made with the one, while the lens is supported with the other. Before the attempt is made to remove the lens, the pupil should be dilated with atropine and a wide iridectomy made. Then in accordance with the recommendation of Paine, the depth of the eye should be fully illuminated with an electric light, placed a little above and to one side of the eye, and with a condensing loupe, so that the dislocated lens is visible in the vitreous—the operator standing so as to look down to the very bottom of the latter. In this way it has been possible to remove lenses in cases apparently desperate.

A subluxated lens, such as is found after reclination as practiced in India, often floats up behind the pupil after a regular cataract incision is made, and may then be removed by slipping a spatula behind it and then sliding it up along this by pressure made on the cornea (Major Smith).—D.]

III. Posterior Sclerotomy

880. In posterior sclerotomy (*sclerotomia posterior*) an opening is made in the posterior division of the sclera [so as to enter the vitreous chamber.]

The section should be meridional—i. e., run from behind forward, since this is the direction of most of the scleral fibers, and hence such sections gape the least, and besides with the incision in this direction the fewest chorioidal vessels are encountered, since these too have mainly a meridional course (Fig. 154). The position of the

section must be chosen, so that neither an ocular muscle nor the ciliary body is injured. For the latter reason the section should not extend farther forward than at most to a point 6 mm. from the corneal margin. The indications for sclerotomia posterior are:

1. *Detachment of the retina.*—A broad Graefe knife is thrust into that spot of the sclera which corresponds to the most prominent part of the detachment. As soon as the knife has penetrated the sclera and chorioid and into the subretinal space, it is turned a little, so that the wound is thus made to gape. We then observe the conjunctiva lifted up by the subretinal fluid which escapes from the wound, so as to form a yellowish vesicle. As soon as the fluid ceases to escape, the knife is withdrawn again.

2. *Glaucoma,* when the anterior chamber is obliterated, and hence iridectomy has become technically impossible (cases of glaucoma malignum and glaucoma absolutum). The operation is performed in the same way as in detachment of the retina, except that instead of subretinal fluid some vitreous is withdrawn. Owing to the greater consistence of the vitreous it is usually necessary to make the section somewhat longer. After sclerotomy the anterior chamber is usually restored, so that an iridectomy can be performed later on.

3. A meridional section of pretty great extent is made when *extraction of a foreign body* or of a cysticercus from the vitreous, is in question. [In this case it is well to add a small cross-cut at the end of the meridional incision, so as to give the latter a T-shape (Weeks). See § 881.—D.]

[4. To *divide a membranous cataract* by scleronyxis (see page 990).—D.]

[In doing a posterior sclerotomy it is advantageous to cover in the incision with a conjunctival flap. A quadrangular flap is made and laid back, and then the sclerotomy is made in the portion of sclera thus exposed. The conjunctiva being replaced covers in the puncture.—D.]

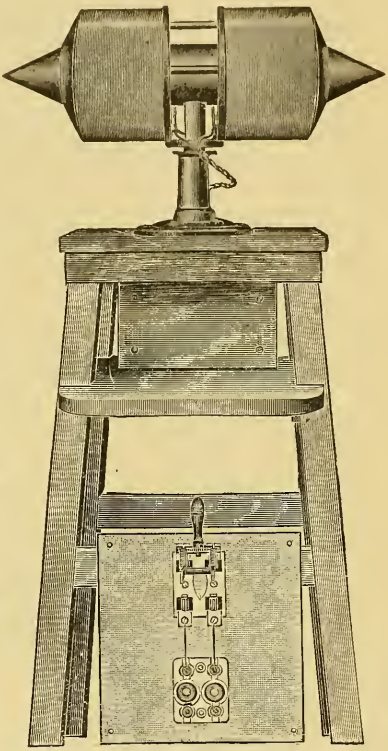
IV. Removal of Foreign Bodies from the Eye

881. [The removal of foreign bodies (including entozoa) presupposes a careful preliminary examination to determine the precise location of the foreign body. In some cases this can be done with the ophthalmoscope. Metallic foreign bodies can be located by radioscopy, and the presence of a magnetic metal can be detected by the magnet (see page 347).—D.]

If the wound is still gaping and is large enough, we can pass a well-disinfected instrument in through it, and endeavor to grasp the foreign body. If the wound is not adapted for this procedure, either because it has already closed up or because it presents unfavorable conditions on account of its size or position, it is better to make a new wound, located in the cornea or the sclera, according to the situation of the foreign body. In placing it in the sclera, the region of the ciliary body must be avoided; the section must lie behind the latter, and is best made in a meridional direction (i. e., running from before backward), since such wounds gape the least. Through the wound we enter with the instruments in search of the foreign body; but the operation of grasping and extracting it is often attended with great difficulty, and very frequently miscarries. The best prospect of success is afforded by foreign bodies in the anterior chamber, since we can be guided by sight in taking them out; also by fragments of iron, for whose extraction we employ an electro-magnet.

The attempt to remove the foreign body which has penetrated into the eye is often beset with great difficulties, and very frequently is unsuccessful. No fixed rules can be laid down for the procedures to be employed for this purpose, as almost every individual case has its peculiarities and calls for an operation devised specially for itself.

The extraction of a chip of iron from the vitreous by means of a *bar magnet* introduced through an incision on the sclera was first performed by MacKeown (1874). Hirschberg constructed the first available *electro-magnet*, which is the form now commonly employed. About a rod of soft iron is wound a spiral coil of copper wire, not too thin, the two ends of which are connected with a powerful galvanic element. The ends of the iron rod, which project somewhat beyond the spiral, are a little bent and end in a blunt point, adapted for introduction into the interior of the eye. For this latter purpose we enter either through the wound itself, in case this is large enough and is still open, or we make an incision in a suitable situation in the cornea or sclera, according to the location of the foreign body. Now very powerful electro-magnets are used (Haab, and others). [Mellinger's inner pole magnet is an electromagnet in the form of a ring big enough to contain the patient's head. An iron rod brought into the center of the ring becomes magnetic and can be used to withdraw the foreign body.] Giant magnets are not introduced into the eye, but are merely applied to it exteriorly, as they are able to attract even small fragments of iron when at some distance from them. They have the following advantages over the small magnets: (1) It is not necessary to make a wound in the sclera; (2) the situation of the fragment need not be known precisely; (3) even very small fragments can be removed in this way.



[Fig. 459.—Haab's Giant Magnet.—D.]
(After Norris and Oliver.)

It is not often that the point of entry of the iron chip is a proper place to apply the magnet to, in order to extract the fragment from the eye. Generally, it is better to place the tip of the magnet against the center of the cornea, so as to bring the fragment through the pupil and into the anterior chamber, from which it may then be removed by an incision made at the margin of the cornea (anterior operation). [This is the operation of election when the foreign body is in the lens or iris. When it is in the vitreous, many prefer to withdraw it either by the giant magnet or the hand magnet through a T-shaped incision in the sclera as close as possible to the site of the foreign body as previously determined by radioscopy. If the situation of the foreign body is determined accurately, so that the minimum of traumatism is produced in removing it, and if conjunctival flaps are used, this posterior method appears to give good results. In certain cases when the foreign body does not yield to the magnet, Jackson connects the latter with a pair of sharp scissors, which, being thus rendered magnetic, is introduced into the wound and is used both to tunnel a way to the foreign body and to withdraw the latter when detached.—D.]

CHAPTER VI

OPERATIONS UPON OCULAR MUSCLES AND ORBIT

I. Squint Operations

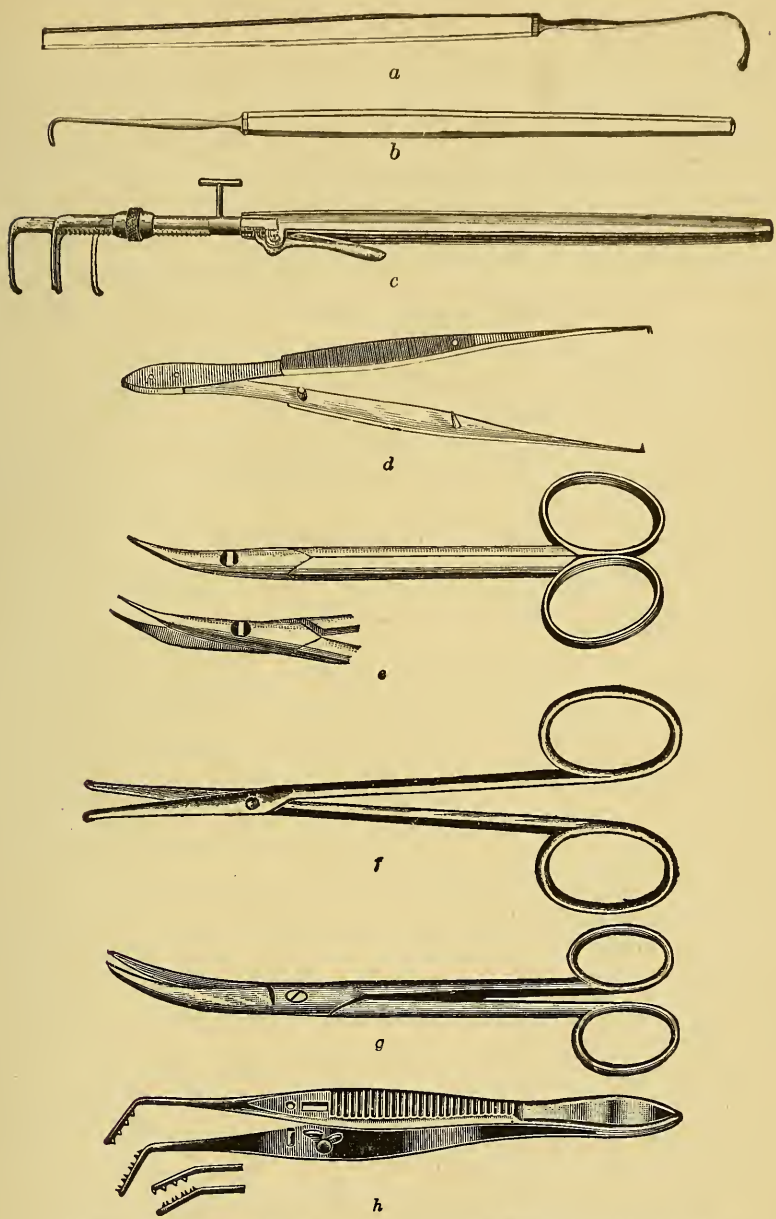
(a) Setting Back of an Ocular Muscle (Tenotomy).

882. Technique.—Tenotomy is performed upon the internal or external rectus; less often, upon the other ocular muscles.

Tenotomy of the internal rectus by Arlt's method is performed as follows: The conjunctiva on the nasal side of the cornea is lifted up with a fixation forceps so as to form a horizontal fold in which a vertical cut, situated about 4 mm. from the margin of the cornea, is made with a single snip of the scissors. The incision is then enlarged upward and downward, and the conjunctiva to the nasal side of it is undermined. Starting from the wound, the fixation forceps is passed inward till it reaches the tendon, which is grasped, drawn somewhat away from the eyeball, and divided close to its insertion in the sclera. For this purpose a small pair of curved scissors is used, the blades of which should have blunt points, so as not to stick into the sclera. [It is more usual to follow Von Graefe's method of operating. In this an incision about 10 mm. long is made through the conjunctiva at right angles to the tendon and right over the insertion of the latter. A buttonhole is made in Tenon's capsule above or below the margin of the tendon and close to its insertion, and through this hole a squint hook is inserted beneath the tendon and raised so as to lift the latter away from the sclera. The tendon is then divided close to the hook with the scissors.—D.]

After dividing the tendon the next thing to do is to see whether there are not some strands of tendon still remaining at its upper or lower border. A squint hook (Fig. 460, *a*) is accordingly passed in beneath the tendon and is carried upward and downward so as to explore all parts, the intention being to catch up upon the hook any fibers that may chance to be intact and then to divide them.

After the division of the tendon has been completed, a test must be made of *effect* of the operation, which should be neither excessive nor insufficient. We first (1) make the eye that has been operated upon turn toward the side of the divided muscle. If the tendon has been cut through completely, there must be a considerable diminution of the motility inward. If the eye can be turned inward as well as it could before the operation, this proves that some strands of the tendon have remained undivided. As in this case the result of the operation would be nil, these fibers must be sought out with the hook and divided. On the other hand, the diminu-



[Fig. 460.—Instruments for Tenotomy, Advancement, and Enucleation.

a, squint hook for picking up the tendon and putting it upon the stretch. *b*, Stevens's s tenotomy hook, used especially in performing partial tenotomies. *c*, Bruns's modification of Clark's hook for tucking-advancement. *d*, strabismus forceps for taking up conjunctiva and tendon in the button-hole operation. *e*, Stevens's tenotomy scissors, with cutting edges near the end. *f*, straight probe-pointed scissors. *g*, heavy scissors for dividing the optic nerve in enucleation. *h*, Prince's forceps for holding the tendon securely in advancement. Similar in principle is Reese's forceps used in resection. Other instruments used are a spring speculum or Desmarre's retractor, for holding the lids open, and a fixation forceps (Fig. 400). A delicate needle holder and curved needles are also required for stitching the conjunctiva, and, in the case of advancement, for suturing the tendon.—D.]

tion in motility may be too great, in case we have not only divided the tendon, but have also loosened up too much its connections with Tenon's capsule. In this event the effect of the operation must be decreased, the tendon which has slipped back too far being reattached further forward with stitches. (2) We make the patient fix his gaze upon the finger held in front of him, and then keep bringing the latter nearer and nearer to his eyes. After a properly performed tenotomy of the internus, a convergence to at most 12 cm. should be still possible. If the eye that has been operated upon halts in its movement of convergence before it reaches this point, this argues an excessive effect of the operation. The working capacity of the internus is then so greatly weakened that we should have to fear the subsequent development of a divergent strabismus. In this case also the effect of the operation has to be reduced again. [3. A more accurate way—and for heterophorias and operations on the vertical muscles the only satisfactory way—of testing the effect of an operation consists in prismatic measurements of the deviation for distance and near by the screen, parallax, and, if binocular vision is present, by the Maddox rod and phorometer, or red glass (see pages 767–771). The deviation is determined by these means before the operation and repeatedly during the operation, which is carried on by cautious division of the lateral attachments of the tendons until a satisfactory result is secured. By these tests we can measure to within 1△ or less of the actual deviation.—D.]

The extent to which the strabismic deflection is corrected by the operation is a matter of but secondary importance. In fact, when the deflection is great, the correction can not possibly be secured by a single tenotomy.

When, by testing in the manner given, the effect of the operation is discovered to be satisfactory, the operation is finished by uniting the conjunctival wound with a suture.

The operation can be rendered nearly painless if, besides instilling cocaine and adrenaline before the operation, some cocaine solution is injected beneath the conjunctiva at the spot where the tendon is divided. [This, however, sometimes puffs up the tissues enough to obscure the proper relations of the parts.—D.]

Tenotomy of the external rectus [and also of the superior and inferior recti] is performed in an analogous fashion. We must simply bear in mind that the insertion of these muscles lies farther from the cornea than does that of the internus [see page 714].

[In tenotomy of the *external rectus*, we usually wish to secure a very thoroughgoing effect, and unless this is accomplished and a moderate primary over-correction obtained, the after results are apt to be disappointing. It is usually well to get as the immediate result of the operation a slight limitation in outward movement, but this should not exceed 1 or 2 mm. In tenotomy of the *superior* and *inferior recti* great care must be taken

not to produce even a slight over-correction and in no event to make very extensive division of the lateral bands attaching the tendon to the sclera. Otherwise a complete paralysis of the muscle may be produced which is pretty hard to remedy. This paralysis may occur long after the operation. Tenotomy of the inferior rectus should in general be avoided, as the results of operation on this muscle are particularly uncertain and paralysis of it causes greater discomfort to the patient than that of any other muscle, since it gives rise to confusing diplopia in looking down, i. e., in the habitual direction of the gaze.—D.]

[It is possible to tenotomize the *superior oblique* by dividing the reflected tendon through a cutaneous incision in the brow. Tenotomy of the *inferior oblique* is done by dividing the tendon of origin of the muscle through a cutaneous incision 15-20 mm. long, made at the lower inner angle of the eye just below the lachrymal sac. The incision is carried down to the bone, and the orbital septum is divided just where it joins the lower and inner margin of the orbit. A squint hook is swept inward along the floor of the orbit with its point directed upward and inward until it engages the tendon. The latter, which is more or less buried in fat and fascia, is completely divided with the scissors. The effect is a complete paralysis of the muscle. —D.]

Tenotomy of an ocular muscle was first tried by Strohmeyer upon the cadaver and some years later (1839) was performed by Dieffenbach upon the living subject. Dieffenbach did not divide the tendon, but the belly of the muscle. Hence, it not infrequently happened that the posterior half of the muscle retracted so far that it could never again become attached to the eyeball. The divided muscle was then completely paralyzed, and when the operation had been performed upon a convergent strabismus, the latter was transformed into a marked divergent squint. Owing to such bad results, the operation gradually fell into such disrepute that surgeons were on the point of giving it up again. Then Böhm proposed a new and improved method, namely the division of the tendon as we practice it now. Von Graefe added to this the method of regulating the operation with precision, showing how its result could be increased or diminished.

[Some use Critchett's *subconjunctival* method. In this the tendon is not exposed by a long conjunctival incision, but a buttonhole is made in the conjunctiva and capsule above or below the tendon and just beyond its insertion. (Thus in a tenotomy of the internal rectus it would be made at a point 6 or 7 mm. to the inside of the cornea and on a level with the lower corneal margin.) Through this hole a hook is inserted beneath the tendon, and on this the tendon is divided by scissors passed beneath the conjunctiva. No sutures are necessary. The translator prefers the open method described in the text.—D.]

[In *Snellen's* subconjunctival tenotomy a buttonhole is made through conjunctiva, capsule, and tendon in the middle of the latter and just beyond its insertion. Starting from this hole the tendon is divided upward and downward by the scissors carried underneath the conjunctiva. This method was used by Stevens in his partial tenotomies (see page 1013).—D.]

[When we wish to secure a large effect, especially in operating on the external rectus, we may divide the tendon 2 or 3 mm. beyond the insertion. As it is possible in

this way to cause a complete paralysis of the muscle, it is well to insert a *stitch* in the tendon before dividing it, so that if an undue effect is produced it can be remedied at once by drawing the tendon up again until only a moderate impairment of the rotating power of the muscle is left. This is a good precaution in tenotomy of other muscles especially the superior and inferior recti.—D.]

[When a tenotomy is done merely to *relieve a temporary strain* on the opposing muscle (as in advancement of the latter when we wish the stitches to be subjected to no extra pull), it should involve simply the insertion without going into the lateral attachments or producing any real impairment of rotation in the muscle tenotomized. —D.]

883. Method of Action.—Tenotomy acts by displacing the insertion of the divided muscle to a spot situated farther back. The divided tendon glides back upon the sclera and forms a new attachment to the latter. Owing to the fact that the insertion of the muscle now lies farther back, the effect of the latter upon the eyeball is permanently impaired.

How does tenotomy of a muscle *weaken* the latter? Let us assume that a tenotomy has been performed upon the right internus for right convergent strabismus. The right external rectus, which was stretched and elongated as a result of the convergent squint, now tends to return to its normal length. Hence, after the division of the tendon of the internus, the externus draws the eye outward, and thus diminishes the strabismic deviation. In proportion as the eye thus rolls outward, the tendon of the internus glides backward over the sclera. This retraction is increased by the elastic contraction which every divided muscle shows: hence, the tendon of the internus lies farther back upon the sclera, and becomes attached there anew. The insertion of the muscle is thus approximated to its point of origin at the optic foramen, and the muscle is shortened. Before being divided, the muscle in its relaxed state had a certain length, from which by its contractile force it was able to shorten down to a certain minimum. After the division this minimum remains the same, but the length of the muscle in the state of relaxation is less; hence, the difference between the state of rest and of maximum contraction is diminished. But this difference corresponds to the power of adduction of the eye, which, accordingly, is permanently reduced after the tenotomy. We can readily convince ourselves that, after an operation, the eye cannot be brought inward as far as it could before; indeed, it is from this fact that we determine whether the operation has been successful.

It is hence clear that the correction of the faulty position due to the squint is purchased at the expense of the adduction. In fact, the loss in the motility inward is always greater than the gain in the position of the eye. The former loss is of no moment in cases in which the adduction is greatly increased. Here, even if this is reduced by the operation to a point somewhat below its mean value, this reduction would still fail to become noticeable except when the eyes were in the extreme lateral position. But the case is different when the attempt is made to correct a marked strabismic deflection by a very thoroughgoing tenotomy of the internus, with which there would necessarily be associated a considerable diminution of the power of adduction. We would then, it is true, have the eyes in a correct position while the gaze is directed straight forward; but as soon as the patient wished to look toward the side where the tenotomy had been done (e. g., in the case of right convergent strabismus, to the left) the eye that has been operated on would not be able to follow the movement properly. In this direction of the gaze divergent strabismus would appear just as in the case of a paralysis of the internus. We should not therefore try to correct a marked strabismic deviation by making the tenotomy more extensive, but should increase the effect by

a simultaneous advancement of the external rectus. It is still better to distribute the operation between the two eyes. In this case we ought not to operate on the second eye at the same time with the first but should operate some weeks later, when we can form some sort of idea as to the permanent effect of the operation on the squinting eye.

In tenotomy the setting back of the tendon is mainly due to the fact that after the latter is divided its antagonist draws the eye toward its own side. The *effect* of a tenotomy, therefore, depends essentially upon the condition of the antagonist of the contracted muscle. The more powerful this is, the greater will be its ability to bring the eye into correct position after the division of the contracted muscle. But for the force of the muscles we have a sure gauge in the amount of the lateral excursion (abduction and adduction). [If the muscular power is normal, the outer rim of the cornea should reach the external canthus in abduction, and the inner rim of the cornea the caruncle in adduction. We should determine the abduction and adduction in this way and also the absolute or relative near point of convergence (pages 775, 776)] before every squint operation; since by these means we can predict approximately the effect of the tenotomy.

884. Restriction of Effect.—If the effect of a tenotomy should turn out to be too great, there are the following means at our command to *diminish it*:—

1. When the suture is applied that is designed to close the wound in the conjunctiva, a wide and deep grasp is taken upon the conjunctiva, so that the needle is passed through Tenon's capsule as well. Then, when the knot is drawn tight, the tendon is drawn forward a little along the conjunctiva.

2. If it is apparent that the divided muscle has suffered too great impairment of its action, its end must be grasped and attached again farther forward by means of threads. This event occurs when the connection of the muscles with Tenon's capsule has been loosened to too great an extent, or when the operation has been done for a very slight strabismic deviation. In cases of the latter kind the attempt has been made to modify the tenotomy, so that its action shall be very slight, this being effected by leaving some fibers of the tendon undivided. (Partial tenotomy—see below.)

885. [Partial Tenotomy and Tendino-extension.—Differing in method of action from a total tenotomy are the various forms of partial tenotomy. In these the intention is to divide most of the fibers of the tendon in such a way that the tendon without losing its primitive attachment shall elongate somewhat and thus become more slack.

Von Graefe in 1861 did partial tenotomy by making a central buttonhole in the tendon, carried nearly to the edge of the latter; and also by making a cross-cut in the tendon involving one edge and nearly the whole width. Stevens, adopting Von Graefe's first procedure, and operating subconjunctivally after Snellen's method (page 1011), made very extensive use of partial tenotomy in heterophoria. Other methods consist in making two cross-cuts in the tendon, one on each edge, opposite each other, and each involving one-third the width of the tendon (Ziegler); a pair of such cross-cuts (Abadie); a pair of such cross-cuts combined with a central buttonhole (Verhoeff); two cross-cuts, one on each edge, but not opposite (Todd).[1] Ziegler now combines a pair of cross-cuts according to his original method with a paring down of the central bridge of tendon, continued until the desired effect is attained.—D.]

[According to the translator's experience, the central buttonhole operation produces no material nor permanent effect unless the tenotomy is made practically total, i. e., severs the whole insertion. The graduation of the effect in this case is gotten by

[1] [These methods are not given in chronological order. As Ziegler points out, Abadie followed Von Graefe, and Stevens, Abadie—these being succeeded in turn by Ziegler, Verhoeff and Todd.—D.]

cautious division of the lateral attachments until the desired result as determined by repeated testing is obtained. With the other methods of partial tenotomy he has had no experience.—D.]

[Partial tenotomy is done in low degrees of squint and in heterophoria of moderate amount. It seems to the translator that it should be restricted to tenotomies of the superior and inferior recti (pages 1022, 1023). In esophoria and esotropia of low degree, advancement (resection, or tendon tucking) of the external rectus should be done instead of a partial tenotomy of the internal rectus; and when tenotomy of the external rectus is indicated at all, it had better be complete (see pages 1010, 1011, and 1022). —D.]

886. Results.—The immediate result of tenotomy is greater usually than it is found to be afterward. The more solid the union which the divided tendon forms again with the sclera, the stronger is the action which it can exert, and thus the effect of the operation diminishes in the succeeding four or six weeks.

As regards the *final result,* cases behave differently. The most frequent outcome is that the effect of the operation increases somewhat in the first few days, then diminishing again, and ultimately becoming less than it was directly after the operation. Sometimes the diminution proceeds so far that the effect of the operation almost completely disappears and the operation has to be repeated. This is particularly apt to be the case in divergent strabismus, but it also occurs in convergent strabismus when it has been operated on simply by advancement. In an operation by tenotomy the reverse frequently occurs—i. e., the effect slowly but steadily increases until finally divergent strabismus supervenes. [This may take place even years afterward.] Such a divergent squint has a specially ugly look, much more so indeed than a convergent squint which has recurred after an advancement. Unfortunately, it is impossible, either before or directly after the operation, to foresee with certainty which of these occurrences is going to ensue, so that we cannot at once take measures to combat them.

[Over-effect after tenotomy may be due to mangling of the tendon which makes it curl up and adhere to the conjunctiva instead of the sclera or which produces other untoward results. It is also apt to occur when a muscle is tenotomized for the second time. Re-operation on the same muscle should be avoided when possible, as the results are always uncertain.—D.]

Among the unpleasant results that sometimes accompany tenotomy is *sinking in of the caruncle,* which looks as if it had been drawn far back. This occurs only after tenotomy of the internus, and is due to the fact that the muscle as it retracts draws the conjunctiva of the inner half of the eyeball after it. This can be prevented if the conjunctival wound is closed with a suture and the conjunctiva thus kept in its proper place.

Impairment of the cosmetic result may also be produced by *exophthalmus.* This arises from the fact that after division of one of the recti the eye is not retracted into the orbit with as much force as formerly. For the same reason a slight degree of exophthalmus is observed not infrequently in paralyses of the recti. The exophthalmus cannot be removed but when it has a disfiguring effect it can be concealed, for in exophthalmus of such a slight degree as is here the case the conspicuous thing is not so much the protrusion of the eyeball as the increase in size of the palpebral fissure which is caused by the protrusion, and this latter defect can be corrected by shortening the palpebral fissure at the outer angle of the eye (tarsorrhaphy). [In many cases what occurs is not an exophthalmus but simply a *dilatation of the palpebral fissure* due to retraction of the lid. For as the divided tendon recedes it pulls the lid back, too, by means of

the slip of fascia attaching it to the latter. This retraction is particularly marked after tenotomy of the superior and inferior recti. Per contra, after advancement of these muscles the palpebral fissure is *contracted* (see page 656).—D.]

During the after treatment of a squint operation there is formed not infrequently a *nodule of granulation*, growing out of the sclera at the site of the conjunctival wound. Subsequently this becomes constricted at its base and ultimately falls off. It may also be snipped off readily with the scissors.

Serious accidents, such as suppuration of the wound, exudation into Tenon's space with protrusion of the eyeball, and actual panophthalmitis, can occur only when the wound has been infected during the operation. At the present time these accidents are among the greatest of rarities. If we should perform the operation with a sharp pair of scissors, and at the same time are dealing with an unruly patient, it may happen to us to perforate the sclera. If the operation has been performed aseptically, this accident will, as a rule, pass over without producing any bad results. In general, the squint operation, if carefully performed, may be said to be perfectly free from danger; and it is one of the operations for which patients (especially those of the female sex) are most grateful.

(*b*) *Advancement of an Ocular Muscle*

887. Technique.—[The term advancement may be applied in a general sense to any operation designed to enhance the action of an ocular muscle. A great many advancement operations have been proposed. They aim to effect their object either by attaching the tendon further forward, so as to increase the arc of contact of the muscle with the eyeball and also put the muscle itself more on the stretch (advancement proper); or by shortening the tendon and so putting the muscle on the stretch. Shortening the tendon may be effected either by cutting a piece out of it (resection) or by making a permanent fold in the tendon (tucking or folding operations). In some of the operations two of these methods are combined.—D.]

[In all methods of operation the tendon should be fully exposed by a transverse incision not less than 12 mm. in length, and should be completely freed from any attachments to the sclera or other part, that might prevent its assuming the position required. Especially should the attachments to Tenon's capsule be divided by incisions carried well back along either margin of the tendon.—D.]

[(1) *Advancement Proper.*—In this the tendon is divided at its insertion, brought forward into a pocket made between the conjunctiva and sclera, and sewed to the latter close to the cornea. In doing this it is important not only that the conjunctiva should be dissected freely to form the pocket, but that all adventitious tissue in the latter that might interfere with a firm attachment of the tendon should be removed (Worth). Of the many methods employed we may mention the following.—D.]

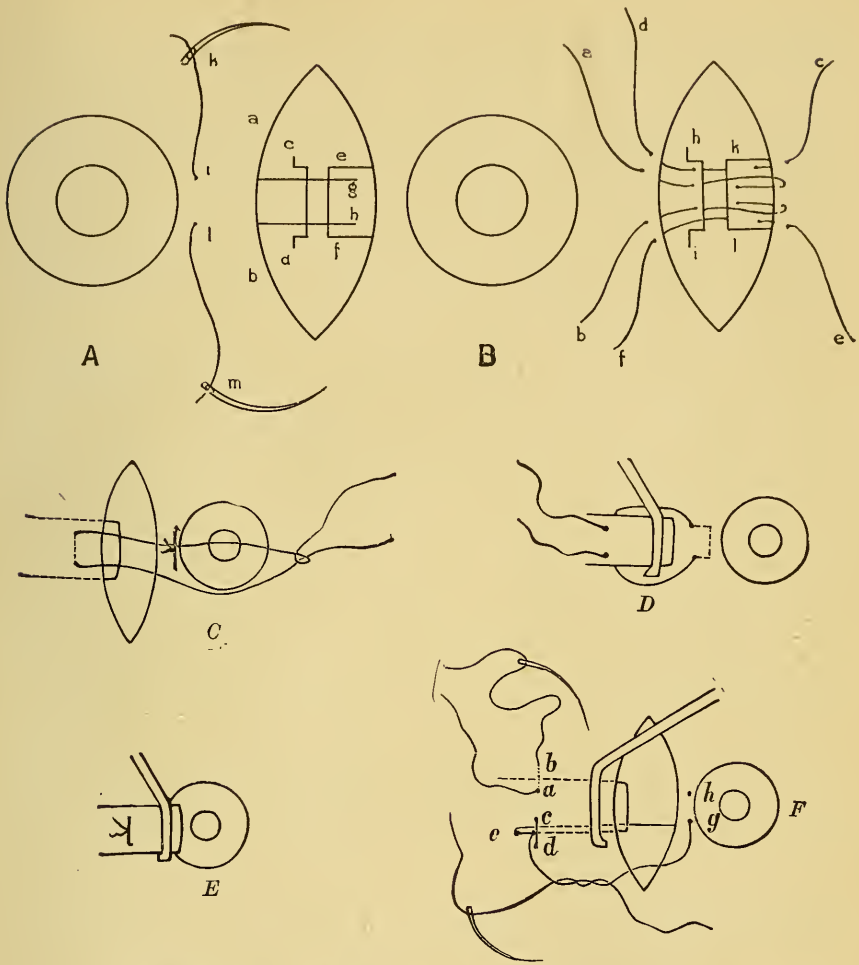
(*a*) Suppose that the case is one of strabismus convergens. In this case the external rectus would be advanced in the following way: The conjunctiva over the tendon of the external rectus is divided horizontally, and is dissected up both over the tendon and also as far forward as the margin of the cornea. The tendon, being thus exposed, is picked up on a squint hook

which is passed beneath it, and two double-armed threads are passed through the muscle as far as possible behind its insertion. One of the threads is passed beneath the upper, the other beneath the lower border of the tendon, and the latter is transfixed from behind forwards at about the middle of its breadth. The muscle is now divided right in front of the sutures, and the most anterior portion of the muscle, which still adheres at the muscular insertion, is cut off. The other ends of the threads are now attached to a point near the nasal margin of the cornea, the needles being carried vertically, i. e., in a direction tangent to the corneal margin and close to the latter, through the superficial layers of the sclera. Then the upper and lower threads are tied separately. The tighter the threads are drawn, the farther is the muscle carried forward—even, in fact, up to the corneal margin. In this way the effect of the operation can be regulated.

[(*b*) A method which the translator has found to give excellent results is the *single-stitch operation* of Oliver (Fig. 461 *A*).

After exposing the tendon, a buttonhole is made in Tenon's capsule just above the tendon. A squint hook is passed through the hold and under the tendon, hugging the insertion. As the point of the hook emerges from beneath the lower side of the tendon, a buttonhole is cut over it in the capsule, so that the point comes through. A Prince's forceps (Fig. 460, *h*) is then substituted for the hook and is locked. The tendon is now divided right in front of the forceps, i. e., at the very insertion. Then the tendon is lifted away from the sclera and its lateral and inferior attachments are thoroughly divided, so that it can play freely backward and forward. Both needles of a double-armed thread are passed through the tendon well back and from within outward (at *g*, *h*) so as to include a good bit of its breadth in a loop, the bight of which is on the under surface of the tendon; then are carried under the conjunctiva and through the episclera, emerging close to the cornea some millimetres apart (at *i* and *l*). Then the small portion in the grasp of the forceps is excised, (or, if a greater effect is desired, a larger portion of the tendon is resected) and the two ends of the looped thread are gradually drawn tight so as to slide the tendon forward under the conjunctiva. The two ends are then tied together with a half hitch, and the result tested. If this is insufficient the knot is tightened, and when the result is satisfactory is tied securely.—D.]

[(*c*) *Worth's operation.*—A curved incision with its convexity toward the cornea is made through conjunctiva and Tenon's capsule (Fig. 461 *F*). The external lip of the wound, including conjunctiva and capsule, is pushed back so as to expose the insertion of the tendon. A Prince's forceps is introduced with one jaw beneath the tendon, the other outside the outer lip of the wound so as to include in one grasp the tendon near its insertion and the cut edge of the capsule and conjunctiva. The tendon being thus securely held, is divided with its attachments close to its in-



[Fig. 461.—Advancement Operations. In part after Haab.]

A. Single stitch advancement.—*ab*, inner lip of the wound in the conjunctiva; *cd*, tendon stump; *ef*, detached tendon; *g*, *h*, points of emergence of the two branches of the loop, the bight of which is on the under surface of *ef*; *i*, *l*, points of emergence of threads after passing over *ef* and *cd* and under *ab*; *h*, *m*, free extremities of threads, afterwards tied. See for description, page 1016.

B. Reese's resection.—*a*, *b*, two arms of the central stitch. These form a loop, the bight of which is on the under surface of *kl*, the detached tendon. *cd*, *ef*, lateral stitches; *hi*, tendon stump. See text.

C. Prince's operation.—A thread, designed to act as a pulley, is passed through the conjunctiva and episclera at the corneal margin and in a direction tangent to the latter. The tendon is then grasped and separated in the manner described on page 1015, and the two needles of a double-armed thread are passed from within o tward through tendon, capsule, and conjunctiva, thus forming a loop on the under side of the tendon. One of the free ends is laid across the pulley stitch, which, as shown in the figure, is then tied over it. Traction is made until the tendon is in the proper position, and the free ends are then tied, one being under, the other over the pulley stitch.

D, E. Verhoeff's operation.—The tendon is grasped with a Prince's forceps and detached from its connections as described on page 1015. A double-armed stitch is introduced into the sclera (previously exposed) at a d stance of 1 mm. from the cornea and is passed for some 6 or 8 mm. through the superficial layers of the sclera in a direction at right angles to the axis of the tendon (vertical dotted line in Fig. *D*). The needle is reintroduced at the point of exit and carried a short distance in the sclera horizontally outward toward the tendon. The same is done with the other needle. Both needles are then passed through the tendon (Fig. *D*), the tendon is brought forward with the forceps (Fig. *E*) to the proper point, and the stitches are tied.

F. Worth's operation.—D.]

sertion. A single-armed suture is passed in through conjunctiva, capsule, and tendon, at *a* and out through capsule and conjunctiva at *b*. A similar suture is passed in at *c* and brought out at *d*. The ends at *c* and *d* are then crossed so as to make a half hitch, and the end with the needle is entered again at *e*, carried under the tendon to the cornea, and brought out at *g*. The same is done with the upper stitch which is brought out at *h*. Before the stitches are passed forward to the cornea the outer lip of the wound, including the strip of conjunctiva, capsule, and tendon in the grasp of the forceps, is excised. The end at *g* is tied to the long end left at *c* and the end at *h* to the end at *a*. Conjunctiva, capsule, and tendon are thus brought forward together.—D.]

Advancement was first performed by Guérin and soon after by Von Graefe. The latter proposed the procedure known as the *thread operation*. This is distinguished from the method first described only in the following particular: The contracted muscle is not divided directly at its insertion, but somewhat behind it, so that a short portion of the tendon remains upon the sclera. Through this portion a thread is passed by means of which the eyeball can be drawn as far as desired toward the opposite side. After the operation has been finished, the ends of the thread are fastened in the vicinity of the eye by means of adhesive plaster, so as to keep the eyeball in the position desired. In this way both the effect of the operation is increased and the tension upon the muscle that has been advanced is lessened. [A similar operation is advocated by Gruening for reinforcing the effect of bilateral tenotomy of the external recti in divergent squint. A stitch passed through each tendon stump is tied over the nose and drawn tight so as to place both eyes for twenty-four hours in a position of extreme convergence.—D.]

[*Prince's* method is shown in Fig. 461 *C*, *Verhoeff's* in Fig. 461 *D* and *E*.—D.]

(2) [*Resection.*—Of the resection operations the best is that devised by *Reese* (Fig. 461 *B*). In this after the tendon has been exposed and separated from the conjunctiva and from the fascial attachments on either margin, the Reese muscle forceps (which resembles a Prince's forceps) is passed with one jaw over and one jaw under the tendon, 3 mm. from the insertion of the latter, and is clamped. The tendon is divided 1 mm. from its insertion so as to leave a stump, *hi*. Both needles of a heavy double-armed suture, *ab*, are introduced through the tendon from beneath, so as to form a loop, 2 mm. broad on its under surface, and are brought out through the conjunctiva at the outer lip of the wound. To reinforce this central suture, single-armed lateral sutures, *cd* and *ef*, are introduced, one above, the other below. These are entered in the conjunctiva at the outer lip of the wound a little above and below the points of emergence of the central suture and are then passed down through the upper and lower margins of the tendon, respectively. First the central suture, then the lateral sutures are passed beneath the tendon stump, then made to transfix the latter, then brought out through the conjunctiva on the inner lip of the wound (see Fig. 461 *B*). All three sutures are tied (*a* to *b*, *c* to *d*, *e* to *f*.—D.]

[The tendon stitches after an advancement or resection should be left in at least a week, and it is expedient to bandage both eyes for five or six days. Worth leaves his stitch in ten days and keeps both eyes bandaged all this time. Reese leaves his central stitch in 10 to 14 days.—D.]

888. (3) [*Tendon folding.*—In one variety of this operation the tendon is shortened by making a permanent tuck in it with some suitable instrument (Savage, Valk, Todd). A combination of tucking and advancement, devised by *Bruns*, gives excellent results.

In this the modified Clark hook (Fig. 460, *c*) is used. This hook has a middle prong which can be set below the level of the side prongs or raised above the latter to any desired extent. The tendon is exposed in the usual way, and the hook with its middle prong depressed is inserted in such a way that the middle prong is beneath, the side prongs above the tendon. The instrument is swept along the tendon to free it from any adhesions to the sclera. Then, keeping the hook 6 or 7 mm. out from the insertion of the tendon, the operator by turning a screw in the handle of the instrument gradually raises the middle prong, carrying a fold of tendon with it. When this tuck is raised to the desired height, the center of its base is transfixed by a needle in the middle of a long slender thread. The needle being drawn well through is cut off, leaving the tendon thus transfixed by two threads, each of which is then tied down over the corresponding edge of the tuck. This keeps the latter from flattening out when the hook is removed. Through an eye in the center prong of the hook a thread is carried, so that when the hook is withdrawn, the thread remains behind in the bight of the tuck. This thread, being armed at each end with needles, is passed forward into the episcleral tissue near the cornea and tied, thus laying the tuck flat and advancing it at the same time. By the amount of this advancement the effect of the operation can be regulated.—D.]

[In *O'Connor's* operation the tendon is split by blunt dissection into a central and two outer strands. The central strand is separated from the insertion and laid back. Then a catgut loop is passed round each outer strand and tied, the result being that the strand is pursed up and shortened in much the same way that a saddle girth is cinched.—D.]

889. Mode of Action.—By advancement the insertion of the muscle is brought nearer the cornea, and thus there is given to it more power over the eye. The farther back the threads are passed through the muscle, and the farther forward the anterior end of the latter is attached, the greater is the effect of the operation. Later on, however, the effect diminishes, for which reason we gauge the operation so as to get a primary over-correction.

[In resection and tendon-tucking the shortening of the tendon renders the muscle taut and thus increases its effectiveness.—D.]

[Worth finds that in the operation as he performs it no primary over-correction is required—the primary effect in this case being also the permanent effect. This seems also to be the case with the Bruns tucking-advancement.—D.]

[Advancement not only reinforces the action of the muscle operated on, but, if extensive, also hampers the action of the opponent—sometimes to a marked degree. This is of particular importance in advancement of the superior rectus, which may so restrict the action of the inferior rectus as to produce considerable and annoying diplopia in the lower part of the field of fixation.—D.]

890. Indications for Squint Operations.—The correction of a faulty position due to squint and the displacement of the range of lateral movement of the eye in favor of abduction or adduction may be obtained either by tenotomy of the shortened, or by advancement of the elongated muscle. In tenotomy we weaken the muscle operated on, and the effect is like that of a paresis of the muscle; the range of lateral movement as a whole is diminished. By advancement we heighten the functional capacity of the muscle concerned and enlarge the total range of movement. Tenotomy involves the danger that, in consequence of the weakening of the muscle, the eye may gradually go too far toward the opposite side, so that a strabismus convergens may be transformed into a strabismus divergens—a thing which may occur years after the operation. In advancement this danger does not exist. Advancement, therefore, is preferred to tenotomy. That nevertheless tenotomy is more often done in practice is due to the fact that advancement is a more complicated operation requiring experience and good assistance as well as a rigorous after-treatment. Yet, thanks particularly to the efforts of Landolt, advancement is gaining more and more adherents all the time. [Advancement, however, also has its dark side. The results are more difficult to estimate than those of a tenotomy and the final result often differs materially and by an uncertain amount from that immediately obtained. In particular, an effect which is good at first and which remains so for some time may months afterward diminish so that the operation proves insufficient. Again, if the stitches tear out or the adhesions give way, the tendon may slip far back, so that the original deviation is made worse than ever. Finally, advancement may cause trouble by hampering the action of the opponent to the muscle advanced. See § 889.—D.]

Of the several varieties of imbalance—

1. *Comitant strabismus* affords the most important and frequent indication for the squint operation. In *strabismus convergens* the operation is indicated in all those cases in which thoroughgoing and protracted orthoptic and refractive treatment shows that they cannot be cured in a non-operative way. With regard to the operation we must bear in mind that the squint really affects both eyes, since the internal rectus is contractured in both (see page 789). Hence it is only in the less-marked cases (below 25° = 5 mm. of strabismic deviation) that it suffices to operate on the squinting eye alone. When the deviation is greater, the other eye also must be operated on. Accordingly, we must estimate the amount of strabismic deviation accurately before the operation. Furthermore we ought to measure the lateral excursion of the eye, i. e., the adduction and the abduction, and the convergence near-point, absolute or relative, since upon these measurements the selection of our method will depend. Simple tenotomy of the internal rectus is allowable only when a marked increase in the adduction coexists with nearly normal abduction. Otherwise ad-

vancement of the external rectus in one or both eyes is indicated. When the deviation and with it the increase in the adduction are particularly marked, the advancement of the external rectus may be combined with tenotomy of the internal rectus.

There is no harm done, if, in order to prevent a divergence of the eyes' developing some considerable time after a successful operation, there is left a slight amount of inward squint imperceptible to the laity. This disappears under the use of the proper convex glasses which are required by the hypermetropia, which is usually present. [If there is any tendency to divergence we may have to under-correct the hypermetropia. By so doing we call upon the patient to use some extra accommodation and with it an extra converging effort which will oppose the tendency of the eyes to diverge. See remarks on page 781.—D.] If the eye which formerly squinted is not too amblyopic, we also institute exercises in binocular vision by means of the stereoscope [converging and diverging prisms, and the amblyoscope], partly in order to prevent a return of the squint, partly to abolish the slight residual convergence. [These are serviceable even when the amblyopia is considerable, and the amblyopia itself can often be helped by systematic exercise of the vision (see page 793).—D.]

In *strabismus divergens* we have to deal, not with a contracture of the external rectus, but with a weakening of the internal rectus. The latter must be strengthened, and hence an advancement of the internal rectus is always indicated. As a rule, this must be combined with a tenotomy of the external rectus, if the effect is to be sufficient. Moreover, the indication in this operation—in contradistinction to that for strabismus convergens—is to secure with it a decided over-correction, since the effect of the operation often diminishes pretty markedly in the course of time.

That form of strabismus divergens which has developed out of a strabismus convergens after too free a tenotomy is associated with considerable weakness of the severed internal rectus and hence always requires a thoroughgoing advancement of the latter with simultaneous tenotomy of the external rectus. [Divergent squint is not always by any means due primarily to weakening of the internal rectus. It often starts as a divergence-excess (see page 785) and then, the squint may often be best relieved by a simple tenotomy of one or preferably both external recti. The cases suitable for this operation are those in which the power of inward rotation of the squinting eye is still good, the convergence near-point approximately normal, the deviation decidedly more marked for distance than for near, and the diverging power, measured by prisms, very large. This seems to be true even of some cases of divergence after tenotomy, although, as the author says, these cases practically always demand advancement.—D.]

[Before correcting a lateral squint, whether convergent or divergent, it is important, especially in children, to ascertain whether there is any considerable vertical deviation present. Such vertical deviation is often due to congenital anomalies, and is the cause of the lateral squint (see page 788).

The latter then is not readily remedied unless the vertical deviation that is the cause of it is first abolished; and in some cases the removal of the vertical deviation suffices by itself to relieve the lateral squint.—D.]

2. *Heterophoria.* In *latent divergence* [*exophoria*] an operation is done (*a*) When it causes the symptoms of a muscular asthenopia; (*b*) when it threatens to pass into strabismus. Operation, however, should not be determined on until all non-operative measures opposing the latent divergence have proved fruitless. Furthermore the latent divergence must be so considerable that it will not by any chance be transformed into its opposite by the operation: for we should then have to deplore the development of a strabismus convergens with distressing diplopia as the result of the operation. Formerly, in cases of latent divergence, tenotomy of the external rectus was very often done with the hope of thus checking the progress of the myopia which generally forms the basis of the latent divergence. To-day it is rare to operate for latent divergence, and, since what is to be effected is a strengthening of the convergence, advancement of one or both internal recti is preferred to tenotomy of the external recti. [Tenotomy would be indicated if the exophoria is due mainly to divergence-excess.

In *esophoria*, due mainly to a convergence-excess, which, as occasionally happens, does not yield to correction of the refraction or other non-operative means, a guarded tenotomy of one or both internal recti may be done, care being taken that the ability of the eyes to converge shall not be materially impaired. Tenotomy, however, is very rarely indicated in this condition. Esophoria which is due to weakness of the external recti or which is well-marked for distance as well as for near, should be treated by advancement or tendon-tucking of the externi.

In *hyperphoria*, if comitant, the best operation is tenotomy or tendon-tucking of the superior rectus, care being taken not to produce an over-effect. If non-comitant, it is treated according to the rules laid down in the next paragraph.—D.]

3. *Paralytic strabismus.* In this the squint operation is indicated only when we are dealing with an old paralysis, the spontaneous cure of which is no longer to be counted upon. The operation is most successful when the muscle, although enfeebled, is still capable of performing its functions, and the strabismic deflection is caused mainly by the contracture of the antagonist. It is only in the slightest cases that we can attain our object by making a tenotomy of the contracted muscle; as a rule, we must combine this with advancement of the paralyzed muscle. If the paralysis is complete, so that the paralyzed muscle is no longer able to exert any action whatever upon the eyeball, it is only from a very extensive advancement that we can expect to get any effect, and even then the only effect we can gain is that the greatly deviated eye is brought back approximately to the primary

position, while the free motility of the eye can in no way be restored. [This does not hold for traumatic paralysis in which the muscle itself or its tendon is injured.—D.]

In paralysis of the superior oblique advancement of the tendon of this muscle is for technical reasons impracticable. Hence, following Mauthner's [A. Graefe's] proposal, we tenotomize the inferior rectus of the opposite side, because section of this muscle causes the same defect of motility in the other eye, that is present in the affected eye as a result of the paralysis, and consequently the troublesome diplopia is abolished.

[Suppose, for example, that the right superior oblique is paralyzed. This muscle normally moves the right eye down, abducts it (carries it to the right), and rotates its vertical meridian inward (to the left); and if it is paralyzed, the movements of the right eye are restricted in all these senses. Diplopia therefore results, which we can prevent if we can restrict the movement of the left eye in the same sense and to the same extent. This we can do by tenotomy of the left inferior rectus, which moves the left eye down, adducts it (moves it to the right), and rotates its vertical meridian outward (to the left). Moreover, as this parallelism of action of the superior oblique of one eye and the inferior rectus of the other holds good for all directions of the gaze, the operation affords complete compensation for the paralysis. In ome cases of paralysis of the superior oblique, there is a decided over-action of the inferior oblique in the same eye, as shown by the decided upshoot of the eye in adduction, and then tenotomy of the inferior oblique is likely to produce marked improvement.—D.]

[A paralysis of the right inferior oblique would similarly be compensated for by a tenotomy of the left superior rectus. In paresis of the right superior rectus, if slight, we may advance the paretic muscle; if great, we may do a tenotomy of the contralateral left inferior oblique (page 1011). This latter operation is particularly indicated in congenital paralysis of the superior rectus, in which it gives excellent results, as it does also in some cases of traumatic paralysis of this muscle. In paralysis of the inferior rectus, advancement of the paretic muscle in general is indicated. In paresis of the externus, advancement of this muscle, combined with tenotomy of the internus in the other eye, is indicated, and will usually have to be supplemented by tenotomy of the internus in the same eye. So in paresis of an internus, its advancement combined with tenotomy of one or both externi is usually required.—D.]

Landolt even for paralysis of the superior oblique prefers advancement, this being done on the inferior rectus of the paralyzed eye. [Mauthner's procedure is much the better.—D.]

II. Enucleation of the Eyeball

891. Enucleation consists in shelling the eyeball out of Tenon's capsule, the conjunctiva and all the tissues adjoining the eyeball being left behind. Bonnet has the credit of having been the first to introduce this operation, which he did upon the basis of his studies upon Tenon's capsule (which hence is also called Bonnet's capsule). Before this, operators were in the habit of cutting the eyeball, together with the neighboring soft parts, out with a knife in a way not very different from that in which a butcher is accustomed to do it. This much more radical operation, which is called *extirpatio bulbi*, is at the present time performed only in those cases in which malignant neoplasms have grown out from the eyeball into the tissues of the orbit so that a simple enucleation of the

eyeball is no longer practicable. By *exenteration of the orbit* (exenteratio orbitæ) is meant a scooping out of the contents of the entire orbit so that nothing but the bony walls of the latter are left. This operation, too, is performed only for malignant new growths.

Enucleation, as done by Arlt, is performed as follows: The lids are separated by Desmarre's retractors [or a spring speculum]. For the operation itself a fixation forceps and a pair of straight scissors, which should have one point blunt and the other sharp, are employed. If the operation is on the left eye, the conjunctiva close to the temporal margin of the cornea is first picked up and incised. From this wound as a starting point the conjunctiva is divided all round the cornea, and then it is loosened from its connections still farther back. Then the external rectus is grasped with the forceps and divided behind the latter, so that a stump of tendon remains attached to the sclera. This serves to hold the eye with, during the subsequent course of the operation, which consists in the division of the rest of the ocular muscles and of the optic nerve. The blunt-pointed blade of the scissors is passed beneath the tendon of the superior rectus, and the latter is taken up upon the scissors and is divided close to the sclera by a single snip. The same is done with the inferior rectus. Then the scissors, closed, are passed from the outer side in behind the eyeball to feel for the optic nerve, which when the eyeball is drawn forward is put upon the stretch so as to form a hard cord. When the optic nerve is felt, the scissors are opened and the nerve is cut off as close as possible to the eyeball [except when the operation is done for the removal of malignant growths (see page 1026)]. As soon as this is done the eye can be pulled out of the orbit (luxated) in front of the lids. Then the remaining structures attached to the eyeball (the internal rectus and the two oblique muscles) are divided as close as can be to the eyeball. With this act the enucleation of the eyeball is complete. A wound cavity is now presented, which is bounded behind by Tenon's capsule, in front by the detached conjunctiva of the eyeball. Through the margin of the latter, which corresponds to the limbus of the conjunctiva, a thread is passed alternately in and out, so that a suture is formed like the string of a tobacco bag, and when this is drawn tight the conjunctiva is completely closed. [Many omit sutures.] Then by the use of a pressure bandage we take care that the conjunctiva shall be pressed against Tenon's capsule, so that it may become united with it.

In the right eye the operation is performed in the same way, except that the detachment of the conjunctiva is begun at the nasal side of the cornea, and the internal rectus is the first thing to be divided. This slight difference between the operation upon the right eye and that upon the left is explained by the fact that we try always to cut from right to left with the scissors, as this is the more handy way of doing.

Many operators use the squint hook for performing enucleation. The tendons that are to be detached are grasped with this, drawn out, and divided upon the hook. This method is easier, but also more elaborate and more tedious, than the method which Arlt devised of picking up and dividing the tendons with the scissors directly.

892. *Healing* takes place after enucleation without suppuration and by primary union. The cavity which remains after the removal of the eyeball is lined by Tenon's capsule, the raw, inner surface of which is presented to our view. Upon it can be recognized the divided ends of the ocular muscles, and at its most posterior part the cross section of the optic nerve surrounded by some orbital fat. This raw surface is covered by the bulbar conjunctiva, which after being detached from the eyeball hangs down so as to form the anterior wall of the wound cavity, and is then carried into the latter, so that its posterior, raw surface gets to lie against the anterior, raw surface of Tenon's capsule. The aperture which the conjunctiva has in its center corresponding to the cornea has been already closed by the tobacco-bag suture. Hence, there is no raw spot remaining uncovered.

Enucleation, if performed in an aseptic manner, is an operation perfectly devoid of danger. For purposes of anæsthesia we may employ either general narcosis or the injection of cocaine (see page 916). The hæmorrhage ordinarily is slight, so as to require no other measures for its arrest than the application of a pressure bandage upon the closed lids. In case the bleeding is more profuse, a tampon of iodoform gauze must be introduced into the orbit itself. Under normal conditions the operation wound heals within less than a week. Purulent inflammation (phlegmon) of the orbital issue occurs after enucleation only when the wound has been infected. When enucleation is performed upon an eye in which active panophthalmitis is present, purulent meningitis with a fatal issue sometimes sets in after the operation. Panophthalmitis, therefore, is a contraindication against enucleation (see page 472). [Many, however, enucleate provided the orbital tissues are uninvolved; others perform evisceration of the eyeball (see page 1027).—D.]

The *prothesis* (*artificial eye*) should not be inserted sooner than fourteen days at earliest after the operation. It consists of a shell of glass, which is made in imitation of the anterior division of the eyeball, and which is retained in place behind the lids. After an enucleation which has healed in the normal way there is found a cavity clothed with conjunctiva, which, behind the upper and lower lid, is converted into a deep furrow corresponding to the fornix conjunctivæ. It is into this furrow that the upper and lower rims of the artificial eye are inserted. The deeper the furrow is, the better will it keep the artificial eye in place. For this reason we take care in operating to preserve the bulbar conjunctiva as much as possible. In cases in which we are compelled to remove part

of the conjunctiva, the portion which remains may be drawn into the cavity by cicatrization, so that the fornix is made proportionately shallower. In this way it may become impossible for an artificial eye to be worn. [In such cases a new cul-de-sac may be fashioned, deep enough for the reception of the artificial eye (see page 941).—D.] The artificial eye moves conjointly with the other eye, although its excursions are smaller; for the ocular muscles, although detached from the eyeball, still retain their connection with Tenon's capsule. They move the latter in the same direction that the other eye is moving in, and with Tenon's capsule they move both the conjunctiva which lines it and the artificial eye which rests upon the conjunctiva.

The *artificial eye* should be taken from the orbit every night and be well cleaned. In time it loses its luster, and must then be replaced by a new one. It not infrequently happens that the conjunctiva is thrown into a state of catarrhal inflammation by the mechanical irritation which the artificial eye sets up. Then the wearing of the latter must be restricted to a few hours each day, or given up altogether for a while, and the conjunctival catarrh must receive appropriate treatment. But the opposite of this state of things also occurs—namely, that, through wearing an artificial eye, troubles that were formerly present are made to disappear. This is the case when, after an enucleation, the lids sink back and an entropion develops, in consequence of which the cilia, which are directed inward, irritate the conjunctiva. By the insertion of an artificial eye the lids receive support; the entropion disappears, and with it vanishes the condition of irritation in the conjunctiva.

The artificial eye may be worn not only in an empty orbit but also over the eyeball. The only prerequisite is that the eyeball shall be diminished in size, either as a whole through atrophy or phthisis, or at least in its anterior division through applanatio corneæ, or as a consequence of ablation of a staphyloma of the cornea. An artificial eye when resting upon the natural eyeball has a particularly deceptive and natural appearance, and also moves quite perfectly with the eye which lies beneath it. Unfortunately, the stump of an eye does not always bear an artificial eye over it. It may become irritated by the latter so as to get inflamed and painful. In such cases either the artificial eye must be laid aside or the too painful stump must be enucleated.

In an empty orbit the ordinary shell-like artificial eye sinks too deep and does not move very well. Hence for such cases eyes have been manufactured which are convex posteriorly (reform eyes of Snellen).

893. Indications.—The indications for enucleation are:

1. *Malignant tumors* in the eyeball, and also those on or behind it, provided they can not be removed radically by a less thoroughgoing operation with retention of the eyeball. In tumors which develop in the posterior section of the eyeball (gliomata of the optic nerve and sarcomata of the chorioid) there is a possibility that the new growth is in process of transmission backward along the optic nerve. In such cases, therefore, the optic nerve is not divided close to the eye, but as far back as possible. After the enucleation has been performed, the cross section of the piece of nerve attached to the eyeball is examined. If it should

prove to be attacked by the new growth, the portion of the optic nerve which has been left in the orbit must also be sought for and exsected.

2. *Injuries.* Enucleation is performed at once (primary enucleation), when such a considerable injury is present that the eye is lost beyond peradventure. This is the case in extensive laceration of the anterior portion of the eyeball, with escape of a part of the contents of the globe. By enucleation in such a case we spare the patient the panophthalmitis that otherwise awaits him, or the tedious and painful process of shrinking of the eye.

If the injury is of such a character that the preservation of the eye, at least so far as its form is concerned, is not altogether out of the question, we try first to save the eye by initiating that form of treatment which is indicated by the nature of the injury. If, nevertheless, inflammation develops, and the vision of the eye is absolutely abolished, the indication then is to perform enucleation (secondary enucleation) in order to prevent sympathetic inflammation of the other eye. Enucleation should also be performed upon those eyes which have been blinded by inflammation in consequence of an unsuccessful cataract operation.

3. *Irido-cyclitis, atrophy of the eyeball,* and *phthisis bulbi* furnish an indication for enucleation whenever sympathetic ophthalmia threatens to occur, or has already broken out. And even a condition of painfulness in the eye which can not be relieved in any other way demands the performance of enucleation, provided all hope for retaining or restoring a serviceable degree of vision has disappeared.

4. *Glaucoma absolutum,* when it is associated with continual pain, and when other less radical operations have either been already performed without success or are impracticable.

5. *Ectasia of the eyeball.* When the eyeball is very much increased in size either by large staphylomata of the cornea or sclera or by hydrophthalmus, it torments the patient by giving rise to continual attacks of irritation, by preventing the closure of the lids, and by producing disfigurement. Enucleation is then indicated provided the eyeball can not be diminished in size in any other way (e. g., by a staphyloma operation).

6. *Hæmorrhage* which comes from the eye that has been operated upon or that has been ruptured, and which can be arrested in no other way.

7. *Cosmetic considerations* sometimes furnish the indication for the removal of a blinded and very disfiguring eye, so as to allow an artificial eye to be worn in its stead.

894. Evisceration of Eye.—Inasmuch as the artificial eye acquires such a cosmetic advantage from being placed upon the shrunken eyeball, the attempt has been made to replace enucleation by an operation which does leave a stump in the orbit. This operation is *exenteratio bulbi* (evisceration of the eyeball). According to Alfred Graefe's method, it is performed in the following way: In the first place, the cornea together with an adjacent zone of the sclera is removed by first incising the sclera near the

limbus with a knife, and then detaching it by a circular cut with the scissors. Then the contents of the eyeball which has thus been opened are scooped cleanly out with a sharp spoon, so that the inner surface of the sclera lies exposed. Lastly, the opening is closed again by means of sutures passing through the conjunctiva and the cut edges of the sclera. In this way a stump is obtained which, however, shrivels up so much in the course of time, that it scarcely has any value as a support for the artificial eye. Hence, the attempt has been made to get a stump that shall remain large permanently, by introducing into the empty scleral capsule a chemically indifferent, sterilized foreign body and allowing it to remain there permanently [Mules's operation]. So, too, a foreign body can be inserted in the cavity between Tenon's capsule and the conjunctiva after enucleation. For this purpose glass, silver, or gilded spheres, or balls made of calcined bone, elder pith, or sponge are used. If the operation is done aseptically, these foreign bodies give no trouble while in situ, but it often happens that later—sometimes not till years later—they are expelled. Hence some after enucleation have inserted under the conjunctiva for permanent lodgement subcutaneous fat which they have taken from some other part of the patient's body. Later on, however, this inserted fat gradually shrinks and to such an extent that the maintenance of the cosmetic result aimed at is jeopardized.

Obviously exenteration can not replace enucleation, when it is a question of a malignant new-growth in the eyeball, and it is only in connection with the other indications for enucleation that it can be considered. But healing after an exenteration is not as smooth as after enucleation, and if the operation has not been perfectly aseptic, the stump that is left may suppurate. Moreover, the protection that exenteration affords against sympathetic inflammation is not as complete as it is with enucleation.

[Evisceration of the eyeball by some is preferred to enucleation when orbital cellulitis or panophthalmitis is present. In this case, Lister performs a *partial enucleation;* i. e., removes the cornea, scoops out the eye, and then removes all the sclera except a frill about the optic nerve. He believes that since the optic-nerve sheath is not opened, the danger of septic meningitis is averted, and that healing is quicker than when the whole sclera is removed.—D.]

895. Optico-Ciliary Neurotomy.—In the endeavor to be as conservative as possible, observers have also tried to replace enucleation by the division of the nerves going to the eye. This operation is optico-ciliary neurotomy (Boucheron, Schöler). First the conjunctiva over the internal rectus and then the muscle itself are divided. From the wound thus made the scissors are passed backward to the optic nerve, which is divided as far back as possible. It is then possible to rotate the eyeball so far outward that its posterior segment together with the stump of the optic nerve shall appear in the wound. The portion of the optic nerve still attached to the sclera is then removed close to the latter, so that, provided the nerve was divided well back the first time, a long piece of it is resected. Next, the posterior section of the eyeball as far forward as the equator is freed from all tissues attached to it, in doing which most of the ciliary nerves are divided. Then the eyeball is returned to its place in Tenon's capsule, and fixed there by uniting the divided ends of the internal rectus and the divided conjunctiva with sutures. After the operation is completed a pressure bandage is applied.

Neurotomy is suggested as a substitute for enucleation when we are dealing with eyes that should be removed on account of threatening sympathetic inflammation or on account of pain. Neurotomy, however, has not proved perfectly reliable. Sympathetic inflammation has repeatedly occurred after it, so that when there is a danger of such inflammation absolutely nothing else but enucleation is indicated. Pain, too, sometimes recurs after neurotomy. Added to this is the fact that this operation is more difficult to perform than enucleation and requires a considerably longer period

of healing. It is, therefore, indicated only when there is no danger of sympathetic inflammation, as for example in absolute glaucoma, and when the patient is unwilling to consent to an enucleation.

III. Removal of Orbital Tumors

896. In the removal of retrobulbar tumors the eyeball is in the way and prevents access to the back part of the orbit. If, nevertheless, we wish to perform extirpation of the tumor with preservation of the eye, there are two ways open to us. In cases in which marked exophthalmus has been present for quite a long time, the eye muscles and the optic nerve are very much stretched. Then it is usually easy, after dividing the conjunctiva on the temporal side and also the external rectus, to shove the eye toward the nose far enough for us to be able to get into the deeper parts between the eye and the temporal wall of the orbit. [To get the lids out of the way and to secure more room, the external commissure may be divided and the lids turned back.] When the eye can not be displaced sufficiently in this way we procure access to the back part of the orbit by making a *temporary resection of the outer wall of the orbit* (Wagner, Krönlein).

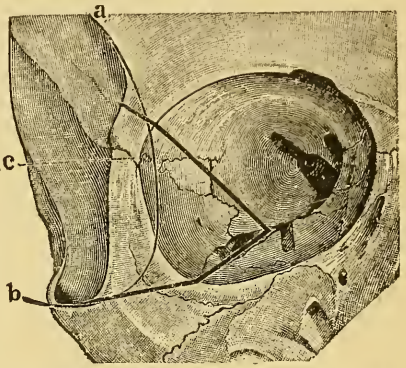


Fig. 462.—Temporary Resection of the Outer Wall of the Orbit. (After Haab.)

We first make an incision through the skin at the orbit's outer margin. This incision begins at the linea semicircularis of the frontal bone, passes in a slightly convex curve downward in front of the outer border of the orbit, and turns outward at the level of the upper border of the malar bone (Fig. 462, *ab*). Where the section runs over the outer margin of the orbit, it is carried through the periosteum down to the bone and then the periosteum is detached from the outer wall of the orbit backward and downward as far as the inferior orbital fissure. Then we divide the periosteum at the two places where the outer margin of the orbit is to be chiselled through; i. e., above the zygomatico-frontal suture (*c*), for one, and just above the place where the zygomatic arch is given off, for the other. Starting from these two points, we chisel through the orbit's outer wall, converging the two lines of incision (the two heavy black lines in Fig. 462) as we go backward so that they meet in the outer end of the inferior orbital fissure. The triangular portion thus defined in the orbital wall is then swung outward, and thus the back part of the orbit is exposed. [When the operation is completed the displaced segment of bone is swung back into position again.]

897. Exenteration of Orbit.—We ought not to push too far our efforts to preserve the eye. If after extensive extirpations the eyeball would have to be left denuded it would subsequently be destroyed by suppuration. After removal of the entire retrobulbar tissue the eye would sink back deep into the orbit, would be fixed there by scar tissue, and would be quite useless.

In such cases it is better to sacrifice the eye in the beginning, even if it should be normal, since by so doing the extirpation of the tumor can generally be done more quickly and with more precision. In such cases we remove the eye and the retrobulbar tissue in one mass—*exenteration of the orbit.* We begin by splitting the outer commissure of the lids out as far as the external margin of the orbit. By this procedure the lids are made freely movable, and can be turned back, the one up, the other down, so as to admit as free access as possible to the orbit. Then the soft parts behind the everted lids are divided with the scalpel down to the bony margin of the orbit. From this as a starting point the periosteum is detached from the bone all round down to the apex of the orbit. The entire contents of the orbit now form a wedge which lies free in the latter, and is attached only at the optic foramen by means of the optic nerve and the ophthalmic artery. This pedicle is now divided and the bleeding cut surfaces are cauterized with Paquelin's thermo-cautery or with the galvano-cautery, since ligation of the ophthalmic artery is impossible for technical reasons. Next, all shreds of tissue still attached to the bone are removed, so that the latter is completely denuded. Then the orbit, after suitable irrigation with a disinfecting fluid, is packed with tampons of iodoform gauze, and a light pressure bandage is applied over it.

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